WHITE HOUSE ENVIRONMENTAL JUSTICE ADVISORY COUNCIL (WHEJAC)

JANUARY 2022 MEETING SUMMARY

VIRTUAL PUBLIC MEETING
January 26–27, 2022

I, Richard Moore, Co-Chair of the White House Environmental Justice Advisory Council, certify that this is the final meeting summary for the public meeting held on November 17-18, 2021, and it accurately reflects the discussions and decisions of the meeting.

April 22, 2022

Richard Moore Date

I, Peggy Shepard, Co-Chair of the White House Environmental Justice Advisory Council, certify that this is the final meeting summary for the public meeting held on November 17-18, 2021, and it accurately reflects the discussions and decisions of the meeting.

Peggy Shepard

April 22, 2022

Peggy Shepard Date

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PREFACE

The White House Environmental Justice Advisory Council is established by Executive Order 14008, titled "Tackling the Climate Crisis at Home and Abroad" (issued on January 27, 2021). As such, this is a non-discretionary committee and operates under the provisions of the Federal Advisory Committee Act (FACA), 5 U.S.C. App. 2.

The WHEJAC will provide independent advice and recommendations to the Chair of the Council on Environmental Quality (CEQ) and to the White House Interagency Council on Environmental Justice (Interagency Council), on how to increase the Federal Government's efforts to address current and historic environmental injustice, including recommendations for updating Executive Order 12898. The WHEJAC will provide advice and recommendations about broad cross-cutting issues related, but not limited to, issues of environmental justice and pollution reduction, energy, climate change mitigation and resiliency, environmental health and racial inequity. The WHEJAC's efforts will include a broad range of strategic scientific, technological, regulatory, community engagement, and economic issues related to environmental justice.

The duties of the WHEJAC are to provide advice and recommendations to the Interagency Council and the Chair of CEQ on a whole-of-government approach to environmental justice, including but not limited to environmental justice in the following areas:

- Climate change mitigation, resilience, and disaster management.
- Toxics, pesticides, and pollution reduction in overburdened communities.
- Equitable conservation and public lands use.
- Tribal and Indigenous issues.
- Clean energy transition.
- Sustainable infrastructure, including clean water, transportation, and the built environment.
- National Environmental Policy Act (NEPA) enforcement and civil rights.
- Increasing the Federal Government's efforts to address current and historic environmental injustice.

EPA's Office of Environmental Justice (OEJ) maintains summary reports of all WHEJAC meetings, which are available on the WHEJAC website at: https://www.epa.gov/environmentaljustice/white-house-environmental-justice-advisory-council. Copies of materials distributed during WHEJAC meetings are also available to the public upon request. Comments or questions can be directed via e-mail to whejac@epa.gov

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Virtual Public Meeting
January 26-27, 2022

MEETING SUMMARY

The White House Environmental Justice Advisory Council (WHEJAC) convened via Zoom meeting on Wednesday, January 26, 2022, and Thursday, January 27, 2022. This synopsis covers WHEJAC members' deliberations during the two-day meeting. It also summarizes the issues raised during the public comment period.

1.0 WHEJAC MEETING

This section summarizes WHEJAC members' deliberations during the two-day meeting, including action items, requests, and recommendations.

1.1 Welcome, Introductions & Opening Remarks

Karen Martin, Designated Federal Officer (DFO), U.S. EPA, welcomed the attendees and gave a few announcements for the meeting. She noted that Spanish interpretation and closed captioning were available during this meeting.

Richard Moore commented on the importance of the work of the WHEJAC - to focus and to put an emphasis on environmental and economic justice and to place us at the highest level within the administration. He reminded everyone that it is important to understand that this is the first White House Council on Environmental Justice. This is, again, a very important historical moment in the life of many of our communities throughout the country. He thanked the Council members for their tremendous amount of work. He reminded everyone that sometimes charges are given by CEQ, by the White House, by the Interagency Council to give recommendations back to them on certain issues. The Advisory Council members along with the staff at CEQ moved very quickly on the charges that were given, and they'll continue to proceed with those recommendations and continue to work on the charges that have been put in front of them as a Council. At his suggestion, the Council members briefly introduced themselves and stated their affiliations.

DFO Martin informed the Council that the quorum was met.

Chair Moore introduced himself and turned the meeting over to Chairwoman Brenda Mallory for some opening remarks.

1.2 Welcome & Opening Remarks

1.2.1 Brenda Mallory, Chair – The Council on Environmental Quality (CEQ)

Brenda Mallory thanked everyone for the invitation to join the meeting and the hard work they put into the Council. She reminded everyone that it's the first anniversary of when President Biden signed Executive Order 14008. That executive order, which created this very body, sets up the most ambitious climate and environmental justice agenda ever pursued. She stated that she will talk about the big strides forward that have taken place over the past year. She also wanted to state more importantly and candidly about the work ahead to deliver the meaningful change that communities expect and deserve.

She recognized Dr. Cecilia Martinez who deserves enormous credit for the progress that was made over the past year and who served as the federal government's first-ever senior director for environmental justice here at the WHEJAC. From the president's campaign through the transition to this critical first year of the administration, Dr. Martinez gave all she had to the task of shaping and launching the president's environmental justice agenda. Millions of people across the country have benefited from Dr. Martinez's energy, wisdom, experience, kindness, and heart. Ms. Mallory stated that she is fiercely determined to build on the foundation that Dr. Martinez helped us lay over the past year.

There are some extraordinary champions for environmental justice and equity across this administration, from Secretary of the Interior Deb Haaland and EPA administrator Michael Regan to the talented young leaders in our administration who will be joining this meeting. Every one of them is sharply focused on keeping the president's promises on environmental justice. Make no mistake, they will continue to build the strength of our environmental justice team across the federal government including here at CEQ because we have a lot of work to do. It's hard to believe that a little over a year ago, CEQ did not have a single employee who focused on environmental justice. Today, there is a team of six, and more will be added soon.

She stated that one year ago, the president asked them to ensure that all communities have a voice in the decisions that affect them, including here at the White House. In 2021, they formed this very body, the White House Environmental Justice Advisory Council, composed of some of the nation's foremost environmental justice experts and leaders. One year ago, the president asked them to embed environmental justice into the DNA of how the federal agencies do their work.

In 2021, the CEQ established the White House Environmental Justice Interagency Council, which she is proud to lead, that helps coordinate our all-of-government approach to confronting environmental justice. From the Environmental Protection Agency to the Department of Agriculture to the General Services Administration, they have launched or strengthened environmental equity and justice offices, task forces, or strategies. One year ago, the president asked us to deliver the benefits of federal climate, environment, and clean energy investments to the communities that need them most. In 2021, they launched the Justice40 Initiative, where we are transforming hundreds of programs across the government, including programs in the Bipartisan Infrastructure Law, to guide 40 percent of their overall benefits to communities that are marginalized, underserved, and overburdened by pollution. This task is like steering a giant cargo ship in a new direction. Make no mistake, through the bureaucratic machinery of guidance documents and program reviews, they are turning that ship on a new course. Finally, and perhaps most importantly, one year ago, the president directed us to reduce the pollution burdens and climate change threats that communities are facing.

In 2021, they set out to deliver the clean air, clean water, and safe housing and communities that everyone deserves. The president himself helped deliver on this promise by getting the Bipartisan Infrastructure Law done and securing historic investments to clean up superfund sites and brownfields, replace lead pipes, deal with abandoned mines and oil wells, and lots more. As an administration, more broadly, we made big strides in the past year to reduce the burdens and confront the injustices that many communities are facing. To name a few, they are cracking down on PFAS and other toxic chemicals, banning chlorpyrifos, cutting vehicle pollution, reforming FEMAs disaster programs, lowering energy burdens, helping tribal nations bolster climate resilience, electrifying ports, confronting housing segregation and unequal housing opportunities, and stepping up environmental enforcement and inspections. In fact, just today, EPA Administrator Regan announced a series of concrete actions his agency is taking to help address the problems that he heard from communities during his Journey to Justice tour a couple of months ago. All of this work in 2021 is laying the groundwork for even bigger progress in the year ahead and for decades to come.

She agreed that there is a lot of work ahead to correct historic wrongs and decades of pollution and injustice in communities of color. In 2022, they are going to work harder than ever to deliver and to see more on-the-ground impacts and benefits of the president's Bipartisan Infrastructure Law. They will have a climate and economic justice screening tool to help ensure that the benefits of federal programs are reaching disadvantaged communities. They will publish the first annual Environmental Justice Scorecard to provide accountably on Justice40 and other key commitments. They will update the Environmental Justice Executive Order issued by President Clinton. We will restore basic community safeguards during federal decision-making processes by completing Phase 1 of the reforms of the National Environmental Policy Act regulations. They will make a lot more progress across the government to cut toxic pollution, bolster community resilience, and confront longstanding environmental burdens and injustices. They cannot do this work alone. They need WHEJAC and the public's help, recommendations, and partnership. She thanked the Council for all the service, time, and dedication. From the president on down, they will not stop working until they deliver on the commitments they made and the justice that our communities are owed.

She then introduced the next speakers: Admiral Rachel L. Levine and Dr. Arsenio Mataka.

1.3 U.S. Department of Health and Human Services Presentation & Discussion

1.3.1 Admiral Rachel L. Levine, Assistant Secretary for Health – U.S. Department of Health and Human Services; Arsenio Mataka, Senior Advisor for Health Equity and Climate – U.S. Department of Health and Human Services

Rachel Levine thanked everyone for the invitation and their hard work. The earth is clearly at a turning point, and history is going to look back at this moment and reflect on the actions and the inactions that are taking place now.

She stated that, as we've seen during COVID-19, we are all interconnected. We need to ensure that a healthier future includes eliminating health disparities and promoting health equity. The fact is that this pandemic has affected some communities far more than others. It has particularly impacted communities of color, and this underscores the profound disparities in health that have long plagued our nation for far too long. Our goal is to ensure that we address these inequities and prevent these inequities in the future. She emphasized that health equity is a key part of our mission at the United States Department of Health and Human Services. It is an absolute priority of our secretary, Secretary Becerra. That is why the secretary, and the Biden-Harris Administration are pursuing a comprehensive approach to advancing equity for all.

Now, that includes the COVID-19 Health Equity Task Force, of which she is very proud to have an ex officio seat. The COVID-19 Health Equity Task Force provided recommendations to the president in November on how to mitigate the health inequities caused or exacerbated by the COVID-19 pandemic and how to prevent these inequities in the future. The task force recommended the administration prioritize the following actions to address inequitable health outcomes that communities of color and other underserved populations have experienced during the COVID-19 pandemic. This includes investing in community-led solutions that address health equity, enforcing a data ecosystem that promotes equity-driven decision making, increasing accountably for health equity outcomes, investing in a representative health care workforce, increasing equitable access to quality health care for all, and leading and coordinating the implementation of the COVID-19 Health Equity Task Force's recommendations within an equity infrastructure within the White House. They are working on those right now. The recommendations have been made, and the administration is working on an implementation plan to ensure they learn from the lessons of today to create a more equitable and healthier future for us all.

She stated that she wants to prioritize the work that they are doing with our Health and Human Service Office of Minority Health and the secretary's Health Disparities Council, of which she co-chairs, which offer ongoing leadership in deepening our understanding of inequities and their drivers and making the connection across all of the different divisions within HHS to address them. One area that she is energized about is to engage in a new office that is within the Office of the Assistant Secretary for Health, and that is the new Office of Climate Change and Health Equity. They cannot for a moment take their eye off the COVID-19 response, but they must recognize the stress on our health system that climate change will pose in the future but also poses to them now and the challenges that they are seeing now from climate change, as well as other environmental justice issues. They need to address these and take comprehensive action now.

Health care crises don't wait their turn. They can descend in the same place on the same people at the same time. We have seen right now how extreme weather stoked by climate change has added to the health threats of COVID-19, for example, by shutting down testing of vaccination sites, by forcing people to balance sheltering and COVID-19 exposure in several different areas, and even threatening health care facilities that are already stressed by surges in COVID-19 cases. They are committed to this new office's mission to protect the health of those most burdened by the impacts of climate change knowing that many of those same people are also most burdened by health disparities in general and by COVID-19. This office was created in response to President Biden's executive order tackling the climate change crisis at home and abroad. It is part of the administration's ambitious plan to tackle the climate crisis. At HHS, they are so excited to be part of the change that brings a cleaner, healthier future to our health and for the health of our nation and the world.

The office has three main areas of work. The first area aims to build the resilience of communities to the health impacts of climate change, especially communities facing more than their fair share of the climate change burden. They're going to work very closely with each HHS division and with the regional offices to help tailor solutions that meet each region's unique needs. The second area focuses on harnessing the long-term resilience and recovery in infrastructure initiatives that we have to combine climate resilience with health equity. They go together. They know that environments where people are born, live, learn, work, play, and worship and age can either make people healthier and more long-lived or they can contribute to illness and health disparities and those disparities that make people more susceptible to the threats of climate change. They believe that the Office of Climate Change and Health Equity, which we affectionately call OCCHE, has an important role in assisting other agencies in optimizing the health benefits of their climate change-related investments.

The last area includes partnering with our nation's hospitals and health systems to help them reduce their greenhouse gas emissions and make them also more resilient to the impacts of climate change. The United States health care sector accounts for roughly 8.5 percent of the United States carbon emissions. Globally, the health sector accounts for about 4.5 percent of global carbon emissions. This effort is critically important towards meeting President Biden's goal for economy-wide greenhouse gas reductions by 2030. Now, as they look to reduce health disparities, whether they're related to COVID-19, related to climate change, related to environmental justice and beyond, they want to be as expansive as possible considering those who have been historically and currently denied opportunities and are disproportionately impacted by climate and environmental impacts, poor access to quality health care, and roadblocks to their wellbeing. That means being particularly mindful of how to be more inclusive in advancing equity for race and ethnic minorities, people in impoverished and rural communities, LGBTQI+ community members, and other historically underserved populations. The Department of Health and Human Services is committed to an evidence-based approach to understanding these barriers and advancing the opportunity for equity.

That is why they are committed to using every tool in our toolbox to expand the data that they have on race, ethnicity and primary language, sexual orientation and gender identity, geography, disability, and the social determinants of health. The social determinants of health are critical. As you know, those are the social issues that were not usually considered health issues but, in this context, clearly are. Economic opportunity and a living wage are health issues. Housing is a health issue. The environment clearly is a health issue. Education is a health issue. Transportation is a health issue. Nutrition is a health issue. All of those are health issues, so enhancing our data allows us to paint a better picture of the populations that we are serving and/or those that we are not serving well and to work to better innovate and focus our resources and policy changes to address these social determinants of health and health disparities. The National Committee on Vital and Health Statics at the CDC has been tasked with developing recommendations on the specific domains of these social determinants of health as well as sexual orientation and gender identity data that need to be collected for administrative surveying clinical data. The HH Office of the National Coordinator, the ONC, for Health IT is also leading efforts to make sure that they have the data to document and share information about the social determinants of health.

Of course, they need to expand our definition of health equity to included LGBTQI+ individuals, especially, for example, trans youth who are confronting so many challenges. They know that trans youth are vulnerable. They have a high risk of being bullied or harassed in schools that are in their community. In addition, they have to address issues of those that are most vulnerable, LGBTQI+ youth, LGBTQI+ seniors, LGBTQI+ immigrants, and particularly LGBTQI+ individuals of color. For example, trans individuals of color are at risk not only of discrimination or harassment but actual violence and murder. They are making strides, and they have started to revive and expand the HHS Health Disparities Council, which she co-chairs, to work on these LGBTQI+ policy changes across the agencies. We have a new LGBTQI+ coordinating council, which she also co-chairs. Recently, they've made some important policy changes to make the Affordable Care Act and other covered programs more equitable so that people can have access to care without being discriminated against because of sexual orientation and gender identity. I am also excited to serve as a member of the first White House Gender Policy Council, which was charged with leading the development of the first-ever national strategy on gender equity and equality. This was released in October 2021.

This national strategy sets forth an aspirational vision and a comprehensive agenda to advance gender equity and equality in domestic and foreign policy and demonstrates that families, communities, and nations around the world stand to benefit. This identifies ten interconnected priorities: economic security, gender-based violence, health, education, justice and immigration, human rights and equality under the law, security in humanitarian relief, climate change, science and technology, and democratic participation and leadership. We have a president who supports equality and works to ensure that everyone is represented, and that gives people a voice. It gives them a chance to affect change and to help people understand the diverse needs of our nation.

She acknowledged that she outlined a number of the steps that she is taking, her office is taking, and they're taking across HHS and the administration. They're looking at what else is under her purview and her portfolio to ensure health equity for all, and that includes our work on environmental justice. She is proud to say that her office is working on an environmental justice strategic plan that will inform our actions moving forward in this space. Already, some of these actions are in the works. This vital work on environmental justice is being led by Arsenio Mataka, our senior advisor for climate and health equity. She turned the meeting over to Arsenio to outline their upcoming environmental justice work.

Arsenio Mataka thanked everyone for the invitation to speak and for all their hard work. He gave his background story. He stated that a lot has changed at the state level from when he started in 2011, from just a few employees and attorneys who worked part time on EJ issues to a bureau that included EJ-only employees and attorneys. None of that would've been possible without advisory councils like this and communities that pushed and pushed for meaningful action. Frankly, that's why I'm really excited to be here at the Department of Health and Human Services. He stated that there is now an environmental justice unit that is focusing on some of these items.

The framework in the previous administration didn't leave us much of anything for environmental justice at HHS. His office immediately relaunched the HHS Environmental

Justice Working Group. That working group will facilitate strong collaboration across the department. That was the minimum basic they needed to get things going. As Admiral Levine mentioned, right now our EJ unit is looking to draft a new environmental justice strategy. Their environmental justice strategy is nearly a decade old at HHS. To pursue the administration's policy to reform environmental justice, there needs to be a new one. The strategy will focus on short-term, concrete actions alongside long-term planning. The other thing that the EJ unit is working on is -- the EJ unit is partnering with CDC to develop a cumulative environmental justice index for prioritizing environmental justice efforts in programs and tracking investments in disadvantaged communities throughout HHS. He stated that he has seen firsthand how you could use that data to really drive not just funds but policy enforcement. A lot of things can happen when you have a consistent approach to how an institution is looking at EJ and disadvantaged communities.

The other two items that the EJ unit is working on are they're helping coordinate environmental justice obligations flowing from certain executive orders, so 14008 with Justice40. They have some covered programs. They have a wonderful worker training program out of the National Institution of Environmental Health and Science. It's a covered program in Justice40. They also have the LIHEAP program. Recently, the secretary in LIHEAP announced another $100 million from the infrastructure bill to go out to folks to help them with their energy bills. The EJ unit is also working almost like technical assistants to our operating divisions to help with the Justice40 initiative and our environmental justice obligations. Then, finally, the EJ unit is also working with our Office of Civil Rights and partnering with them on an investigation that is at the intersection of environmental justice and civil rights.

We all know the history of these Title VI investigations. They long had been inadequate when it comes to delivering for communities. With that being said, though, he is excited to have this EJ unit partner up with our Office of Civil Rights because our EJ unit will be looking at these other types of services, these wrap-around services, that these communities need irrespective of whatever findings come out of a civil rights investigation. Even if there is no finding or a finding, the bottom line is those communities still need help. At HHS, they have a lot of authority. They have a lot of funding that may be able to help. The EJ unit is tasked with that piece of the puzzle. He turned the meeting over to Vice-Chair Flowers.

Vice-Chair Flowers reminded the Council of a few housekeeping measures for the question-and-answer part. She began with the question, what kind of support are being offered to people who are getting sick with COVID in factories? The state has not provided a way for them to get long-term care, especially for those with long COVID. They've found instances where people have gone to work and gotten sick. They've lost their job, but there's no means for them to get support.

Admiral Levine answered that, in terms of long COVID, they are working on that right now. The secretary has asked our Office of the Assistant Secretary for Health to bring together all of the different divisions within the Department of Health and Human Services to work with the White House on these important issues of long COVID. That includes research being done by the NIH. It includes more surveillance being done by the CDC. It includes the issues of insurance coverage for long COVID and its related conditions by CNS - about long COVID being taken care of in community health centers by HRSA. You can think of almost every division within

HHS. Actually, her office is bringing together all of those different aspects. This is under Admiral Iademarco, who is my deputy assistant secretary for science and medicine. They just had a discussion with the Office of Science and Technology at the White House about long COVID. They're directly working with the White House on this issue. Long COVID was mentioned significantly by the COVID-19 Health Equity Task Force because of the awareness of the health disparities that were articulated. They're going to bring together all of the different divisions within HHS. They're going to collaborate with the White House and other departments across the administration, and that includes the Department of Labor. That includes the DOD and VA all of these different departments to come up with a long-term strategy on long COVID and the people that it's impacting.

LaTricea Adams asked if she could speak a little bit more about the focus around maternal health, especially as it relates to black maternal health, which we know coincides and is adjacent to the climate crisis? Are there any programs that are a bit more in collaboration with the actual administration, being that V.P. Kamala Harris was one of the founding members of the Black Maternal Caucus? What programming exists to support and uplift black women who are literally dying while trying to give birth. The other part of that is what specificity with programing exists around addressing some of the childhood issues that are seen at the intersection of environmental injustice as it relates to childhood cancer and childhood asthma?

Admiral Levine answered that, in terms of maternal-child health and specifically maternal health, that is a very, very important issue. That's an issue that the vice president has worked on and feels so strongly about. Her office is involved in that as well as many different offices' divisions within HHS. It also is a strong health equity issue. Maternal health primarily impacts black women, African American women, and other women of color. We have to understand those disparities. They're working with the NIH, in terms of specific biological research. They're working with the CDC who has some specific programs to study the surveillance and the prevalence of that. In his previous position as the secretary of health of Pennsylvania, with CDC's help, they established a state-wide maternal mortality group to study maternal mortality across Pennsylvania. They're supporting that in many other states. They need to look at that in terms of our community health centers, and HRSA has a strong role as well as ARC, which looks at quality of care. There are so many different aspects of this that we are working on.

For some of the specific programs, his office can probably give even more specific details about what his office and the Office of Women's Health as well as the Office of Minority Health is doing, what HRSA's doing, what ARC is doing, what CDC is doing. They can give you some more specifics. He asked Mr. Mataka to speak on the other issue in terms of environmental issues related to children and, especially, to asthma.

Mr. Mataka stated that, as it relates to environmental health, NIEHS has several programs. He's familiar with one grant program out of California that was significant as it relates to asthma. It deals with childhood asthma, but it also deals with community research and not the community being researched but the community actually doing the research. That was in El Centro, California, where NIEHS had a program that funded research for that area. They had promotors who would go door to door and do interventions with the community. They do have some specific programs that are better than others on that type of approach. They have a presidential task force that's looking at lead as it relates to children. So, from a lead standpoint, they do have a lot of stuff going on with toxins and child centers and lead in child centers and other toxins. They'd be more than happy to give you a more detailed list of those programs. The NIEHS grant was truly transformative not just for that community. It actually reshaped the way that a lot of communities are dealing with low-cost centers and air pollution now. Back in 2011, that grant really propelled California to better develop its network of low-cost centers. It was because of a community research project that did it. He stated that he saw it because he was there, and it was amazing. They got the regulators to rethink how they are doing it.

A real quick example of that, they put up air monitors in the school. The parents were looking at the data and showing that they were getting spikes at 7:30 a.m. or p.m. The parents worked with the principal to move the drop-off point for the school buses. The school buses were idling. They moved the drop-off point 500 feet away because the drop-off point was right next to the playground where the kids were playing for the first 30 minutes before school started. Just that small change with the right data because of that research development brought the PM levels and exposure to those kids, at least for an hour every morning, way, way, way, way down. That's a little bright spot at NIEHS. The WHEJAC gave a recommendation for that program as a program that needs to be funded more prominently. He's seen the results of that. It's an amazing program. They will try to get back to the WHEJAC with a more comprehensive list of examples.

Ruth Santiago stated that she heard a reference to the LIHEAP program and the allocation of $100 million for that. She did not see Puerto Rico on the list. That's really ironic because they have very serious energy issues, and it's been estimated in the first or second-highest electric rate of any U.S. jurisdiction. Going beyond that, they're concerned with root causes and to what extent HHS gets involved. Air monitoring was mentioned. They have a huge problem with air monitors either not functioning or not being properly placed or just not enough. A lot of areas are unclassifiable because of that. She asked, to what extent does HHS get involved in that kind of thing and work towards, say, energy generation transformation, towards renewable, especially distributed renewables, so that these dirty power plants don't continue to overburden environmental justice communities?

Admiral Levine answered that there is a lot of collaboration across the administration on these issues. Some of these specific things that you're mentioning probably are more under the EPA. They have regular meetings about these types of issues across the administration with many of the different departments associated with environmental justice and with climate change. There are some specific roles for HHS and others for EPA and some other departments.

Mr. Mataka added that, on the LIHEAP program, he was just at that press conference. That's a surprise. He told Ms. Santiago that he would look into it. There's eligibility for Puerto Rico, so he's not sure where that information comes from. Concerning the air monitors, the last program that was mentioned was probably the most specific where he's seen HHS or operating divisions get directly involved in air monitoring in communities. Going back in time, he thought that was very progressive of NIEHS to do that because the state of California wasn't even included. They weren't even doing that stuff at the time. There is an appetite to do a lot more of that. There are some questions about appropriations and whether or not they have the funds to do it. That entity would be probably the best spot to do it because they've already done that. He mentioned that these are community-led air monitoring.

It used to be back in the days when the air districts would get their scientists around, and they would say, "You have to do it in these regions." No, these were community-led. Communities mapped them out saying "I'm concerned about these locations, and this is where we want them." Then the scientists came back and said, "Well, yeah. That could make some sense." But it's a community-driven approach. He observed the one at El Centro and others in California and San Jacinto. Now there is a proliferation of them. At HHS, the NIEHS under the NIH is probably the area that has the most experience in doing that. He would like to reach out and have a meeting with anybody to talk to the director about that, of how we do more of that.

Viola Waghiyi stated the background of her Alaskan community with their community-based project funded from NIEHS since 2000. She asked they have heard of sequestered persistent organic pollutants. These pollutants arrive in the Arctic through air and ocean currents and end up in the permafrost, glaciers, and ice that are now melting because of the warming of the planet. She encouraged the Department of Health and Human Services to meet with their tribes in rural Alaska to talk about the food crisis there. Has anybody ever looked at or heard of sequestered POPs? That's another route of exposure for my people on top of the military toxins, including PCBs, pesticides, heavy metal, solvents, from the two formerly used defense sites. She added that they need adequate health care related to the health disparities from the military contamination and assistance with the availability of our main foods, the marine mammals.

Admiral Levine stated that they would very much like to meet with her to discuss both of these environmental issues, the environmental justice issues that you're talking about sequestered hydrocarbons in the permafrost and also the impacts of climate change, which are very, very important. Her office would be very pleased to meet and discuss these issues.

Mr. Mataka stated that they should probably set something up and have a more detailed discussion.

Kyle Whyte asked, in the environmental justice and climate change work, are they taking into consideration the relationship between health and cultural activities and cultural practices?

Admiral Levine answered absolutely, they want to be culturally informed and culturally competent in all of the work that they do. That is critical to them being sensitive and successful in terms of our work.

Mr. Mataka stated that they are looking at TEK. They had some results in California, including TEK. They just pulled into his Office of Climate Change somebody from EPA that has some background on that, so they're looking at bringing that experience to our team on Office of Climate Change and Health Equity.

Angelo Logan thanked them for their presentation and their invitation to engage as a council as well as individual communities across the country. He asked, how do they ensure environmental justice and protect public health through the whole-of-government approach, specifically with departments like DOT and DOE, as billions of dollars are being spent on the construction of the infrastructure project, like whether they're highways or energy projects that have the potential to do extreme harm to local communities? How do they participate in that whole-of-government approach to ensure that they're protecting public health and that they're not taking one step forward and two steps backward when everyone knows projects like highway expansions are going to continue to perpetuate environmental racism? Specifically, how do they engage with the agencies like DOT and DOE to ensure that we're not building projects that are going to continue to cause more harm for the climate and the local community?

Admiral Levine stated that's a very important point. The Biden-Harris Administration is really committed to doing both, to economic growth but also do to do that in an environmentally conscious way so that they don't do more damage from an environmental justice point of view. They do have many meetings administration-wide as well as other meetings specifically with other departments to discuss those issues.

Mr. Mataka answered that there are some folks there who are taking a different approach now in leadership as well as within the ranks that are reaching out and asking those questions. It's going to be interesting to see how the cross agencies think about, for example, if they were going to fund an expansion of the 710 freeway, opportunities where they could head off some to those concerns at some of those interagency workgroup meetings. He thinks they've been very open to those discussions.

It's a matter of building up that capacity to engage at that level. He knows a lot of money's flying out the door right now, and those projects are being funded. There needs to be an interagency discussion about those impacts and trying to be more vocal. The secretary would be very, very interested in leaning into that conversation, especially under projects that may have a particularly high burden on an already burdened area. They're trying to figure that out right now. They're working through our interagency workgroups with DOT and DOE. They got folks there who understand these issues, and they're open for conversations on that. There's a lot more openness with his leadership.

Admiral Levine added that, at the highest levels, she's been at meetings with Secretary Granholm and Secretary Buttigieg where these specifics have been discussed, about how to enhance the infrastructure but how to do that in an environmentally sensitive way. They are thinking about it.

Mr. Logan stated that the invitation to engage is a two-way street. He looks forward to their leadership and brokering those types of meetings with folks from DOT and also for you all to engage with the Council here so that we can make inroads. Presentations are great, but action is better. Personally, he'll reach out to both of them to see how they might get those conversations going, specifically with DOT and DOE.

Miya Yoshitani followed up with, since they've talked about the impacts of COVID on communities and long COVID, the intersection between the communities who've been most impacted by the pandemic and by COVID and those same communities who are most impacted by poor air quality and air pollution and the relationship between that and the investments in the infrastructure bill, public dollars are going to increase pollution through investments in polluting industries in these same communities. The cumulative impact doesn't seem like there's any place where that actually lives centrally to ensure that, across the board, these different agencies and these public dollars are not creating additional harm in communities who have been most impacted. It feels like we are beyond the point of needing just open dialogue. It seems like there is a need for additional policy intervention.

Admiral Levine stated that, within the Department of Health and Human Services, the secretary's Health Disparities Council looks at all of the different health equity issues across the department to develop synergy. There are many different offices of health equity. They have an office called the Office of Minority Health. HRSA has an Office of Health Equity. CMS has an Office of Health Equity. They're looking to coordinate that across health and human services. Then, they do have many, many meetings at many different levels with other departments about these issues. There are several different administration-wide efforts run by the White House to coordinate actions and policy.

Mr. Mataka added that what you're describing is overall guidance to these infrastructures. Unfortunately, he would hand that over to CEQ and the White House to talk about how they're going about doing that. If you're looking to make overall guidance to all these investment projects to address cumulative impacts or other health impacts, whatever that may be, that would require overall, administrator-wide guidance. They have two items on the infrastructure bill. We have IHS for $3.5 billion for sanitation and LIHEAP. A lot of the programs are elsewhere. In an ideal world, you would have some sort of framework like that that could be applied throughout all the infrastructure dollars to flag health concerns, to flag cumulative impacts concerns, to flag all these things that we know. It could not improve, but maybe it exasperates the environmental pollution load in the community or the health disparity load in the community. He stated that that's a great question, but it's better suited for somebody else to answer

Nicky Sheats made three quick points. The first one is that he heard that in some way they were dealing with greenhouse gas emissions and trying to lower those. He suggested that also, from an EJ point of view, dealing with greenhouse gas coal polluted emissions, particularly something like airborne fine particulate matter, and lowering that is just as important, arguably more important from an EJ point of view, than the greenhouse gases. Go after the greenhouse gases, but he thinks the other things that are being emitted along with greenhouse gases should be gone after also. He asked Mr. Mataka if he said that 100 full-time people were working on EJ in California? **Mr. Mataka** answered, yes. That's the number he was given after all the legislation in the Disadvantaged Communities legislation. They're sprinkled throughout the administration.

Dr. Sheats suggested that maybe, for CEQ and other agencies on the federal level, that's a number they should shoot for. In proportion to the population they're serving, it seems that it should be even higher for agencies on the federal level.

Mr. Mataka commented that there's a caveat to that. The caveat is that, as legislation was getting signed, environmental justice positions were tacked on. It didn't all come in one swoop. For every little environmental justice bill passed, they would add full-time employees. That's how it was built up.

Dr. Sheats replied that that's a good goal. In proportion to their population, that's a good goal to hit at a time specified in the future. That's just a suggestion for other agencies in the U.S. government, all agencies in the U.S. government maybe. He commented that he hopes other agencies were listening about what they're doing with cumulative impacts. At some point, EPA needs to come up with a cumulative impacts policy that, under certain circumstances, forces applications for pollution permits to be denied based on cumulative impacts and environmental justice.

Mr. Mataka replied that they're looking at the cumulative impacts from environmental exposure, health factors, as well as social vulnerability. They're hopeful that that works for HHS to try to drive certain programs. When you think about it, we have HRSA in charge of the federally qualified health centers. There are just a bunch of programs. HHS is massive. A tool to help guide them on environmental justice will be critical. **Dr. Sheats** replied that they need to go beyond a tool and actually to policy. They need to have that policy that results in denying permits. If the federal government made regulations, it would be even more impactful.

Tom Cormons noted that they have two really important programs that are a part of the Justice40 pilot or that are Justice40 pilots: the Environmental Worker Training Program and the LIHEAP program. He asked in what ways do they hope that the mechanics of Justice40 can be a tool to transform both their agency and, even more importantly, its impact on the ground, recognizing that a tremendous amount, perhaps the great bulk of your portfolio as an agency, is something to which Justice40 is going to apply and does apply?

Mr. Mataka explained that it's almost like, if you're an EJ person and you go into HHS, you're like a kid in a candy shop because there are so many programs that touch everybody's lives. So many of those programs are really important, of course. He can't even imagine the level of impact that Justice40 will have on the institution. Regarding the LIHEAP and the Worker Training Program, the Worker Training Program serves as a program that could help Justice40 out. It's kind of reverse. It's like the environmental justice training program that deals with workforce training, which a lot of the Justice40 programs need. In his view, that's an asset to the Justice40 program. With LIHEAP, Justice40 will push LIHEAP to start thinking a lot more strongly. Dr. Howard, who leads that program, can speak to that herself. She heads it up. It already has challenged LIHEAP to rethink approaches as they get into high cost and energy bills and the need to transition communities, especially poor communities, to take part in a transition. Of course, to just transition, they want all segments of society to participate in that. LIHEAP, other than just paying bills, will have an important role in that just transition. It's something that they're going to have to work on.

Robert Bullard commented that that sounds great, but if there's no money, per se, or dollars-only benefits set aside or targeted, the NIEHS work training program is low-hanging fruit that's received inadequate funding. They've been able to do a lot with it. If that's designated as one of the pilots, that doesn't mean that more money will flow to that program or that initiative. It seems to me it would make a whole lot of sense that, if you have a program that's outperforming given a lot of money, then there should be targeted funding toward those efforts, so they get the biggest bang for the buck. That HHS climate program is very small, but climate change is a major health issue. There should be more money put into the climate program at HHS so that it can have an oversized impact when it comes to health impacts of climate change that would impact across the board where we talk about transportation, issues of housing, and issues in terms of other kinds of issues. In terms of the interagency piece and how, when you put $10 in transportation or $10 in housing or $10 in environment and $10 over here, you may have four organizations that have $10. But when you add it all up together, the multiplier effect might be a factor too.

Admiral Levine stated that they certainly agree that they need the funding to accomplish their mission. There is a significant amount of money in the Build Back Better Bill for the environment and climate. Of course, that's, at the moment, in Congress. We will await the efforts of the White House in terms of that. There is specific money in the 2022 budget and the 2023 budget for our Office of Climate Change and Health Equity. She certainly agrees that there needs to be more funding to accomplish our mission.

Vice-Chair Flowers stated that due to time, those people that didn't get a chance to ask their questions will ask their questions at the end. The Council will work with DFO to get those questions and also to provide the answer as well. She thanked Admiral Levine and Mr. Mataka for their time and turned the meeting over to Chair Moore.

1.4 Climate Resilience Draft Charge Presentation & Discussion

Chair Moore introduced the next speakers.

1.4.1 Mark Chambers, Senior Director for Building Emissions & Community Resilience – Council on Environmental Quality, Executive Office of the President; Krystal Laymon, Deputy Director for Climate Resilience – Council on Environmental Quality, Executive Office of the President

Mark Chambers stated that this is an amazing opportunity to be able to continue this pathway of engagement with the WHEJAC and to make sure that they're doing everything they can to open up new and existing lines of communication making sure that they're being as thoughtful as possible and as intentional about the types of conversations that they want to encourage and want to see going forward. They are going to talk about some of the work that's been happening throughout the government, in particular in terms of resilience and stats response, the overview of the interagency working groups that are now activated to tackle a lot of the resilience challenges and threats that they're facing, the resilience working group that WHEJAC's taking on to open up that conversation and make sure that we are all aligned with what's possible, and to get some charges to WHEJAC that they think are the best ways to start this conversation and to continue making sure that the right contributions and recommendations are being made for all this work because it affects so many different agencies and so many different programs that they want to make sure that we're being as consistent as possible and getting all of that feedback wherever possible.

Krystal Laymon thanked everyone for the invitation. She acknowledged and recognized the successful efforts because there is a historic investment in climate resilience. These historic investments will directly contribute to building resilience and reducing risks that everyone faces, especially during high seasons of drought and wildfire and hurricanes. It's no secret that they need to tackle these pieces, especially before the volatile season, and ensure that we're giving attention to supporting the resilience of not just wildfire, flood, but also pieces of the electricity grid that serve as a pinpoint for many people that need to recover after disasters or even during disasters too. In addition, they're really pleased that this will be an opportunity where individual tribes will engage to address their needs in regard to climate disasters and also climate resilience, really moving forward to being able to address many of these issues that people face today.

This is an investment in communities. The Infrastructure Law that was signed is a watershed moment for all of us and really a huge boom to communities. All the points listed are targeted investments to communities. Also, what's been heard in the past is "How do we pay for it?" Really, here it is now. They have this huge opportunity to be able to put the money where their mouth is and to show that they really care about these communities in a way that can move the needle positively to address these climate disaster impacts, as well as support communities that need to recover from these as well.

She highlighted and emphasized that the connectivity between climate resilience and resilience overall is very much intertwined. In regards to broadband expansion, it really does make a difference when you have access and resources to online applications and online resources that can actually help you in terms of applying for these opportunities and also being able to communicate needs and also being able to describe those needs in a way that can perhaps give you funding but then also can allow for better assistance forthcoming as the money is moved through the system. In addition, there was quite a bit of funding for the Department of Transportation. One, in particular, is on reconnecting communities. This is a big push because there have been so many communities that have been facing many challenges due to, really, transportation divides. Providing greater access will also help in terms of the climate resilience of many communities. This is before, during, and after a disaster. Resilience is one of those connective tissues that grants thriving and livable communities. All of this is connected, but they're excited to highlight and pinpoint the climate resilience purpose of this.

Here are some really good examples of information of communities that really showcase the on-the-ground efforts that are going to be made from these investments. They announced about 14 billion infrastructure dollars this year that will cover over 500 projects. No matter where you are, funding is there to achieve some real change and accomplish something on the ground for really a greater good. Also, these programs, from New Mexico to Alaska, are part of the president's Justice40 initiative. They really do want to keep that promise that they are paying attention on the ground, especially in terms of these before, during, and after disasters to make an impact in these communities that disproportionately are affected the most when a disaster does occur.

They also recognize the fact that the frequency of disasters is increasing, and we need to respond to them when they occur and also support communities when they recover. This is just a quick list of some of these opportunities that we've been able to pursue, in particular, FEMA announcing $5 billion to help communities increase their preparedness in advance of climate-related disasters. In addition, Congress directed emergency funding from the devastation communities felt and added funding for wildfire recovery, provided relief for crop loss, and also restoration of housing. There's also a pretty impactful whole-of-government effort to support Puerto Rico's recovery and renewal from Hurricane Maria as well as some of the past earthquake impacts.

Just this past year, they were able to put out a fact sheet listing some of these accomplishments, which includes a new White House working group on Puerto Rico, which is on the cabinet level, that was established last July, a joint launch of DOE and FEMA to conduct a comprehensive study to evaluate pathways to meeting Puerto Rico's 100 percent renewable energy targets, and then also additional relief from the Economic Development Administration, so EDA, to construct a disaster preparedness training center. Of course, overarching for a lot of these efforts has been the constant engagement of FEMA and HUD, which has been a pretty remarkable effort for the both of them to work together and provide relief for those that have been impacted. FEMA has really taken charge in being able to revamp their Individual Assistance Program that's heavily focused on survivors from disasters. Just this last year, they were able to support disaster survivors by expanding the types of documentation that homeowners and renters can use to prove their ownership of where they occupy. This has been pretty game-changing because the changes have resulted in about $120 million in financial assistance for mold remediation and have delivered to 100,000 survivors receiving home repair and rental assistance as a result of expanding ownership and occupancy documentation requirements, which has been disproportionate for low-, moderate-income communities.

She explained a snapshot of the type of activities that they have accomplished so far through directives that have spurred activity within the agencies and the White House, for example, ensuring appropriate programs are resilient to flood risks, which is called the Federal Flood Risk Management Standard, and also supporting federal agencies adjusting to the impacts of climate change, which is a big one because the portfolio at federal agencies is quite large. Of course, many of you recognize that 14008 has provided opportunities, for example, the National Climate Task Force that has taken upon itself at the cabinet level to really strive to make sure that agencies are promoting resilience tactics and looking to find opportunities to do so as well.

She highlighted some of the opportunities through the National Climate Task Force to align interagency coordination on various disaster impacts. They have five interagency working groups that were launched last year. The first one that was launched was in April of last year, focused on drought relief and resilience. This was a very important piece because historic drought really came to a head last year, in particular with the Klamath Basin as well as the Colorado River. This interagency effort led by the Department of Interior and USDA has been looking at opportunities to collaborate with the funding and then also being able to reach people on the ground to provide this type of relief effort.

In addition, this drought IWG, which we call an interagency working group, has been instrumental in working with other partners. We have an existing national drought resilience partnership that looks heavily with other agencies on how they can assist, for example, monitoring and other types of applications that may be helpful in terms of providing some sort of drought understanding and also drought relief. They also have a Coastal Resilience Interagency Working Group that was launched in June. This is a whole-of-government approach to look at not only what are some opportunities to help those who are on the coast, because quite a few of our community members live on the coast, but also what are some of the different solutions. Some of these nature-based solutions are oftentimes more amenable for those on the coast. Are there some trigger points there that they can encourage agencies to utilize that type of approach with grantees and stakeholders on the ground? In addition, this has also been a whole-of-government approach, working with existing partners, for example, the subcommittee on Ocean Science and Technology Workshop, where they held a great workshop and received information from agencies on where they could be looking at in terms of the opportunities on the ground, that is, a lot of the data pieces and also understanding some of those gaps that communities on the ground may need to have better decision making.

In addition, we have an Extreme Heat Working Group. This is great because it's led by HHS, EPA, and NOAA. This opportunity looks at providing the type of instructions for agencies as well as a trickle-down to their stakeholders that they work with in regard to abating extreme heat. Some of the more highlighted portions of this activity are the workplace heat standard and increasing enforcement. This is through an effort through OSHA, which is a great opportunity to be able to understand the type of regulations that may be put in place in order to protect people who are out in the heat during the hot summer seasons. In addition, this group also in the development of a Cool Communities Challenge to invest in heat resilience infrastructure. What's a huge opportunity in this regard is being able to work with communities in an interagency way to showcase what can we do to provide assistance? What are some innovative solutions in this space, especially abating the urban heat island effect and protecting people's health and also being able to understand how they can put that into play and also scale that? That's a great opportunity they're looking forward to.

In addition, they launched the Wildfire Relieving Interagency Working Group and have been excited to see some of the accomplishments from that group already, and this is per the infrastructure law that came out that was signed. We have established a Wildland Fire Mitigation and Management Commission. This commission will be quite instrumental in this policy realm that we have for wildfire. For example, this commission being stood up will be focused on providing recommendations on how they can better abate the perpetual challenge that they have with wildfires and also understand some of the opportunities that agencies can be able to take. This is also a whole-of-government approach. What's great is that USDA also recently put out a ten-year strategy on confronting wildfire and being able to work together to illuminate some of these challenges that communities that are near more vulnerable zones can take and also work with the federal agencies on.

Then, finally, we have the Flood Resilience Working Group. This is a pretty important working group because flooding is one of the higher risks and most damaging impacts on communities. One of the heavy focuses of this working group is the reestablishment and implementation of what's called the Federal Flood Risk Management Standard. That's looking to ensure that federal investments that go into communities, be it a building, an infrastructure project, do have a reduction of flood risks. Making that area and making that construction more resilient to flood and ensuring that, as they have expected having some sort of storm surge and also sea-level rise, there'll be a lessor impact of those areas. In addition, science is very important with that group. What's great is that they have also developed a subscience working group within that group that's really looking at the differences between coastal flooding and also the inland flooding and understanding how that makes a difference when it comes to these types of impacts.

She reiterated that they've been meeting at a regular cadence. This is an opportunity to provide recommendations on future work with these interagency working groups, not only just with that but also with other topics that are very important when it comes to building more community resilience.

Mr. Chambers acknowledged that there are a lot of different pieces. A lot is going on, and a lot of components are happening across the government. There's still a lot more to do and a lot more that they need help with. That's why this opportunity is so particularly necessary and so particularly exciting. It's an opportunity to not just have intervention points to address different issues but to have systemic interventions that allow for a lot of the work that everyone is dedicated to being more successful, more impactful, and more consistent. That is something that they're really dedicated to and really, again, excited for those of us that are working on these pieces all the time. The Climate Resilience Working Group is something that is going to be one of those really important ways to make sure that a lot of the recommendations are actually moving throughout the federal government in a way that can truly impact policy. Having the framework in place to be able to allow for WHEJAC to provide advice and recommendations is critical to the success of these pieces of policy that touch so many different parts of our lives. It's not just limited to environmental justice work, of course. In this way, they're talking a lot about climate change mitigation, resilience, and disaster management, and how they all intertwine. It's incredibly important to be successful at this, and so it's incredibly important that they try as hard as they can to make sure that those communication pathways are wide open.

He presented the charges that will be most helpful to have the WHEJAC Climate Resilience Group take on. Of course, it's not solely limited to this, and they welcome guidance in a lot of different places. This is a start and a good place to be able to engage. They've developed seven different charges that they want to walk through that they think are some of the top-tier ways to engage and would love to continue the conversation and get more feedback as far as that goes.

The first of the charges is around community resilience. What steps can the federal government and the White House take to reduce disparities in climate change impacts in communities, and not just limit to the five different areas that Krystal talked about: extreme heat, flood, wildfire. Please keep in mind that most people are living at the intersection of multiple threats. It's not just each one of these in isolation. As you're taking the charge and thinking about community resilience, please think about how one threat impacts the ability to withstand another and how those, of course, exacerbate existing conditions that create threatening environments for various communities. That's going to be really critical in terms of making sure that the recommendations that are coming to them are acknowledging that reality.

The second is federal disaster preparedness and relief. Again, what types of support are needed in disadvantaged communities to participate in those disaster preparedness relief programs? There was the bipartisan infrastructure law funding for broadband expansion. Those are the types of pieces that unlock the ability to participate. Think about access as being a critical piece of how they think about being able to take advantage of relief efforts and, again, how can this federal disaster relief program better serve disadvantaged communities that have historically received fewer benefits, and what steps and information would help to deal with those disparities. Please think about the particular issues, the particular programs, but also the access to those programs and which ways those can be addressed. Those would be really helpful in those two categories.

The fourth issue is thinking about the health impacts. Adverse health impacts from climate change, of course, disproportionately impacts disadvantaged communities. We would like more information and more guidance on the policies and programs that you think can help address some of those adverse health impacts leading up to and planning while extreme events are taking place and then also afterward in recovery. All of those pieces oftentimes require different measures to the extent that there could be thought put into where there are opportunities to address those health outcomes.

Number five is housing. What steps can the federal agencies take to ensure that our nation's housing provides security and climate resilience for all Americans, of course, critically important? How can policies and funding decisions promote better land-use planning, construction codes, standards, renovations of substandard housing, energy efficiency, and lifetime connectivity to critical services? How is it, again, unlocking the potential of those people inside buildings to better participate in the communities in which they exist? A lot of that has to do with the buildings themselves. How are we making sure that through energy codes, through construction codes, through a lot of the levers that we have, they are more resilient and more capable of withstanding a lot of the threats that we're talking about here today.

Number six, relocation, what steps can the federal government and federal agencies take to address community-driven relocation and to support a community's interest to plan for relocation from a natural disaster or climate change? A consistent theme in not only all of the Justice40 work but also this work as we're talking about resilience is making sure that communities and community voices are being centered in the work that impacts them to the extent that they are being able to get guidance or suggestions around relocation. That would be particularly helpful given the needs that a lot of communities have in terms of dealing with these extreme threats that require moving locations.

Finally, clean energy, again, is another one of those unlocking pieces, particularly around the generation of clean energy and storage of renewable energy. On-site storage of renewable energy allows for much more flexibility and islanding of different communities to allow for them to withstand extreme events. Again, it's about keeping the power on. It's about keeping the cooling on when it's extreme heat. It's about keeping clean electricity heat on when it's cold outside. It's about all of these different pieces that allow time for communities to be able to recover and make sure that they're doing that in a way that is fully benefiting from a clean-energy economy that's growing. Think about that, making sure that we're thinking about policies and processes that can go to support that and support that community resilience that takes place at the same time.

One last piece that I'll mention is that all of these pieces have components that are also significant in terms of job creation. As they do look at the pieces around social resilience, as well as community resilience and resilience against natural threats, being able to have the additional capacity that is created by the jobs that go into doing all this work and preparedness is incredibly important and also part of that model. Please, continue to keep that in the framework, too, of where are these options that these efforts and these interventions help create jobs and sustain jobs, lasting jobs, that will allow for, again, more flexibility and more rigidity when it comes to being able to survive and to thrive through many of these climate-induced threats. He turned the meeting over to Chair Moore for questions.

Chair Moore explained the housekeeping aspects of the questions and answers part as to the previous presentations. He reminded them to be cognizant of past discussions that day. The focus is on specifically what was heard around the charge that's being proposed and being discussed within the Council, and the last slides were identifying the pieces of that charge.

Ms. Waghiyi stated that she's excited to hear about the electric grid updates. Being a fossil-fuel industry-dependent state, they are not seeing benefits in these disadvantaged communities that need them the most. As far as the seven programs that were talked about, the federal disaster preparedness and relief benefits, her people are facing food security issues. They are in a climate crisis. As far as health impacts, they are already some of the most highly contaminated populations on the planet because they rely on their subsistence food from persistent organic pollutants that arrive in the Arctic through air and ocean currents. As far as housing, these substandard homes are prefab homes that are not made for the Arctic. They don't have adequate ventilation. There's mold and no adequate insulation. Also, in relocation, communities like Shishmaref, Newtok, and others have been falling into the Bering Sea because of coastal erosion. There is no ice, and we're seeing increased massive fall storms that are creating erosion because there's no ice. Communities need assistance. They have been trying to relocate for decades. They get funding for relocation, but they have to match funding, which they don't have. Our tribes do not have the resources.

Mr. Chambers stated that her concerns are well heard. There are opportunities to make sure that they are centering those voices in the policy development. This is a part of the pathway to do that. There are opportunities to use the different levels of the federal government around code and standards and upgrades that do impact all of how buildings are made and retrofitted and fixed are very important. Right now, there is a process underway for public comment around manufactured housing, to provide voices around improving the efficiency of manufactured housing to make sure that they are providing the level of insulation and thermal performance. He invited her to keep the conversation going and engage to make sure that they are actively soliciting more engagement for all those points.

Mr. Havey stated that rather than just looking at the disaster end of climate change, how do we make the homes more resilient today to economic impacts? One of the things that he's involved in right now is a very, very significant price spike in gas prices due to a five-day runup by thousands of percent created on-the-spot markets in gas. This is going to cost low-income families in Minnesota $100 million more over the next two years. This is something that has a tremendous impact on our low-income communities when 14 percent of our families are already behind in payments, according to our natural gas provider.

How do we tie resilience into more or not addressing hurricanes and coastal flooding, but into improving the everyday lives of people in their homes so they aren't impacted by the significant economic impacts that are interconnected with climate change and our inability to address them in real-time? He offered to work with the resilience subgroup. In Minnesota, they export $18 billion a year to their utilities, and they have a role to play in this. What role do they play in this resilience effort?

Ms. Laymon stated that most people know the energy burden is very much real. Many people spend up to 30 percent of their incomes just paying for their energy bills, and that's not acceptable because that's a huge amount of money that can go into many different pieces of someone's life. There are many different opportunities, especially with some of these charges that were listed, that they can expand into. Energy efficiency is a huge component of that. When a home is energy efficient, it keeps the heat in or keeps the cool in and sometimes even allows for passive survivability, which means, if the power does go off, then someone there can stay there longer as opposed to being evacuated to a shelter or needing to have some sort of amenities or resources. They can actually live in their homes during the storm or after the storm.

There are a lot of external players that'll be important to these conversations. A really good first step is getting deep into what could be really helpful for us to understand. That may be more external stakeholder conversations, especially with utilities. They do play a role in many of these, especially as they saw with the winter storms last year and even with a lot of extreme heat. The power went off, and so being able to show that reliability in what can be done not just on the household level but then also expand that to much more of the sectorial level would be wildly important. It's all very much interconnected. This is just one piece of the pie.

Mr. Chambers added that one of the charges that were laid out was around clean energy. He mentioned battery storage, battery storage not just in a single-family home but at the utility-scale is incredibly important in being able to start to reconfigure how we deal with the energy market and also do it in a way that allows for much more stability for those clean electrons. That's a really particular place in which it helps to deal with rural applications as well as urban applications where we can look at the ability to store clean electrons, clean power, nearby where it's going to be used and not necessarily have to deal with some of the constraints that come from long lines of travel for that energy. As they start to fix the buildings, as they make them more efficient, make them operate better, they also need to let the little bit of energy they are using to operate them as far as it possibly can. Part of that happens through battery storage and making sure they're being thoughtful about how that is set up to empower communities regardless of their size.

Dr. Morello-Frosch lifted a few issues that are going to be relevant to the deliberations of this resilience group. One is thinking through broadly about flooding and sea-level rise threats to housing, not just in government-subsidized housing but affordable housing that's also market-based. They did a study on this a year or so ago forecasting sea-level rise threats to all kinds of affordable housing, and it's going to be important to think about that more holistically.

The other thing is thinking about flooding and sea-level rise in terms of NATAC disasters and really thinking broadly, again, about different kinds of sites, particularly those that are disproportionately located in environmental justice communities, communities of color, both in rural and urban areas. They've seen this in terms of extreme weather events like Hurricane Harvey. There are sea-level rise threats. This is a slow-moving storm that we can forecast better and really plan for. That also includes cleanup sites not only around surface flooding but around groundwater intrusion due to sea-level rise which threatens to spread out contamination from legacy sites. That's, again, much more of a slow-motion silent storm that often I think escapes some of the regular conversations because they're always responding to these extreme weather events. We can plan for these more long-term things.

The other thing is climate-related pressures around displacement, both in terms of the effects of climate change itself but also in terms of new forms of gentrification that are happening as wealthy or coastal communities are seeking to move inland and displace communities of color that are living in higher-elevated areas. This is an issue, for example, that's unfolding in places like Miami right now. It's important to forecast that in terms of resilience.

The last thing is making sure that they're keeping an eye and that conversation around mitigation strategies and ensuring that proposed climate change and litigation strategies don't undermine community resilience. There's a lot of policy tools, things like cap and trade, that have been shown, for example, in California to not necessarily be health beneficial to communities of color that are disproportionately hosting some of these regulated entities, where they're not necessarily reducing pollution locally but purchasing a lot of allowances and offsets and forgoing opportunities to reduce pollution locally. That has implications for climate resilience from an EJ perspective.

Ms. Santiago commented that, on behalf of the eight communities impacted by the Cano Martin Pena, people here are still celebrating that announcement about the dredging project and the resources finally to do that. That is wonderful, wonderful news. She also commended them for the charges for the WHEJAC Resiliency Working Group. She noted that their example of clean energy was exactly the experience they've had in Puerto Rico after the hurricanes. Since then, we've had earthquakes and many more outages since then. The onsite renewables that you mention under clean energy and storage were definitely resilient, and they're seeing it in the studies and literature that that is the way to go to provide community resilience. All of this will depend on the implementation.

Chair Moore stated that, when he was looking at these three charges, they brought him back again to the reality that many of those are issues that have been testified to and that questions have been asked during the public comment periods.

Susana Almanza commented on the whole use of land used in zoning. Because of all the gentrification and the development that's happening, people are seeing areas, especially in single-family home areas, where the impervious cover was at 22, maybe 30 percent or 35 percent. They are now going up to 80 percent of impervious cover. What has been seen in numerous communities is the heat island effect, and not only the heat island effect but also the flooding effect, especially those living downstream from all the impervious covers that are happening. She questioned how the standards can be raised and how they can bring those policies down. They did talk about that in the revision of the executive order; how do we get cities and counties to abide or use a lot of the EJ principles, the screening tools, to make sure that communities of color are not continued to be impacted by the heat island and the flooding and the gentrification that's happening?

The other thing is, when we talk about housing, there is a need for public housing. A lot of public housing hasn't been built since the late '30s. How do we make sure that solar is included or mandated in several areas? That brings me to the issue of ERCOT here in Texas with the electric grid not having to abide by federal regulations and being more intrastate instead of interstate. Last year, this February, they were all on needles and pins from the freeze that hit them and how some people were out for a whole month without electricity and the cold weather. There were a lot of deaths. A lot of things were not reported with the media about the people who were impacted. They were low-income and communities of color, of course, and other people. Still, they need to look at how does the federal government bring Texas into the federal regulations? The housing and the solar all kind of come together when they're looking at how do they make sure that people can sustain themself when they are in these crises if they have solar power in our communities?

Ms. Laymon stated that, in terms of the flood piece, ensuring that they're actually building to a higher flood standard is going to be very important to get a discussion on. They're already doing a lot to that and trying to make sure that it's weaved throughout all of their investments.

Mr. Chambers added that it was a very good point around issues like increased impervious surfaces. That issue that that creates, of course, is double. It's in addition to exacerbating the impact on flooding and overrunning the local systems. When these are dark surfaces, you're increasing the protentional for heat island. You're just making a threat multiplier. It's really important to look at every different opportunity they have to discuss those pieces that impact multiple places so that they're making sure that they're using the levers and they're using other pieces of the government, not only for the federal government but also for how we interact with the subnational government like cities, states, tribes, and areas in which they're able to help local government to be able to also use their policies around zoning and to be able to help us address some of those issues.

Dr. Whyte recommended there'd be clear information about the connection between CEQ's work and the White House Council on Native American Affairs as well as the new White House Initiative on Asian Americans, Native Hawaiians, and Pacific Islanders. He encouraged very clear information about the status of government-to-government consultation related to the areas of the charge so that there'd be precise knowledge of what is happening and the different things that tribal leaders and the federal government are involved in, obviously, that's publicly shareable and available. He emphasized the importance of ensuring the meaningfulness of the work. Many tribal leaders, federal leaders, and other leaders worked very hard to establish the funding levels in the infrastructure spending that you cited. For example, at one level, over 470 million for infrastructural issues tied to climate, relocation, other issues is much, much larger than the previous budget for climate resilience and related areas.

That 470 million gets very small when it's divided by over 570 federally recognized tribal nations. It's important to ensure that there can be meaningful recommendations made that would address that situation, which again from a more factual perceptive, some of the budget levels we're seeing are a drop in the bucket given the trillions of dollars of assists, resources, and territory that tribes had to forcibly yield to the United States over the last several centuries for the U.S. to establish itself as the global superpower that it is.

Mr. Chambers stated that the note of concern and meaningfulness of the work is really taken to heart. They must make sure that they're not only taking that in with what they're doing but also making sure that is a level of engagement that they're asking of all of the agencies that are working on behalf of these products to make sure that money's being spent well. Also, as they continue to work towards the additional funds that are set up in the framework for the Build Back Better plan, that's another place in which there are opportunities to truly engage in that meaningfulness and make sure that there are opportunities to increase the amount of impact that those funds will have.

Ms. Laymon added that ensuring that they leverage not just those funds but additional funds that can be associated with being able to utilize it for climate and also for infrastructure spending is going to be really important for us. Let's ensure constantly that the tribal piece is threaded throughout the conversation.

Beverly Wright reinforced making certain that they look closely at legacy pollution and communities in older housing. Thinking about a problem that was had after Katrina, when they were trying to change windows to hurricane-proof windows, they ran into historic preservation and all kinds of things that would not allow us to bring these houses up to code. Of course, the people who lived there could not afford to make those changes, and many of them could lose their houses because they couldn't keep them up. There are all kinds of issues related to legacy pollution and climate change and impacts on housing, the people who live there, the poverty that's there, and the gentrification that's sitting right outside of those communities, many of whom are close to downtown, those areas where developers are just waiting to gentrify those areas. That's been said in a lot of different ways. In the end, it's all about the process and the structures that you put in place to make sure that people are served who are the neediest.

Chair Moore reemphasized a few points. One is that the legacy piece is crucial as they look at and continue to engage in this charge and the different pieces connected to the charge or anything that could possibly be added to it. There needs to be a major emphasis on climate change, global warming, and the impacts also in rural communities. The other one is the intersection, relocation. As they even talk about sometimes, Build Back Better, some of our communities cannot be built back better. The last point was the consultation between native indigenous, from nation to nation and the question of sovereignty needs to be kept, also, at the highest level.

DFO Martin clarified that they heard some recommendations during this session. Those recommendations are from individuals. The WHEJAC as a body has not voted to submit any recommendations that would come out of our workgroup process. She wanted to make sure that everyone understands that those recommendations are coming from the individuals during this conversation.

Dr. Bullard stated that they should never underestimate the hurdles and the barriers and the difficulty our communities are facing when it comes to ensuring that resources are accessed by our cities, municipalities, and counties when there are mandates and laws and restrictions saying that the money has to go to the state. The fact is that's six percent of funding; that's real dollars. Justice40 is not real dollars when it comes to their benefits. That's still squishy. The fact that, if there's a mandate that says that this program must be mandated to the state and if there are no guardrails, guidances, coming from CEQ or the justice department to mandate that that money has to go to where they need, then communities will not see any changes. Communities don't have access to that. The money will go to some government entity. If we don't organize, mobilize, and shape the power as to how our government officials, city, state, local, and federal, build the justice framework into it, they will not see any benefits that will accrue to our justice communities over and beyond what has happened in the past. He stated that he can say that without any hesitation because what he's seeing now are decisions being made where there's little input from most communities. They call them disadvantaged communities.

If you're talking the legacy stuff, there's no guarantee that one dime will go to those communities that have lived on superfund sites, that have lived with pollution that is disproportionately in our communities or flood zones, flood plains, low-lying areas, the cumulative impact of all this stuff being put in our communities because of racial redlining, zoning, et cetera. That's a political policy and power arrangement that's sending the money to where usually it ends up. It's not generally ended up in our community. This is based on four decades of research and analyzing this stuff. If we don't be careful and we don't emphasize the urgency of getting this right, then all of this money and people that our communities voted for and elected will not be in place. If the money is going into these different departments, then the money will be sent to the states. Next thing you know, the administration's name is off the check. Next thing you know, their people are saying that we got this money, and the money won't come to our communities. We have to be vigilant on this with a laser focus. If we are not, we're going to get what we've always been getting, which is nothing. That's cynical but realistic.

Chair Moore reiterated that they would get nothing unless they're very organized at the grassroots and community level, both rural, urban, and native indigenous.

DFO Martin announced that it was time for a break. She reminded everyone that the public comment period was next, and the commenters should be ready to go.

1.5 Public Comment Period

On January 26, 2022, the WHEJAC held a public comment period to allow members of the public to discuss environmental justice concerns in their communities. A total of 37 individuals submitted verbal public comments to the WHEJAC. An additional 121 individuals had signed up to speak but were not in attendance. Each speaker was allotted three minutes.

1.5.1 Jamie Banks - Quiet Communities, Inc. (Lincoln, Massachusetts)

Jamie Banks stated that she is the founder and president of Quiet Communities, a national nonprofit organization working with communities affected by noise and related pollution. She also chairs the Noise & Health Committee of the American Public Health Association. Today, hundreds of communities in the nation struggle without federal support to confront harmful noise and related pollution. There's an excess also of fossil fuels. Noise is a public health problem, an environmental problem, and an environmental justice problem. Our organization is trying to provide support to these communities in the absence of a federal program, but we can barely scratch the surface.

Noise is not simply an annoyance or nuisance as some believe, but a serious public health risk. Noise damages not only hearing, but cardiovascular, metabolic, neurological, and psychiatric health, as well as children's learning. It interferes with thinking, communication, and socialization. Several national studies in addition to many local studies show that EJ communities bear the brunt of noise from sources ranging from road, rail, and air traffic to construction to industry, predisposing them to poorer health and learning outcomes. Addressing noise now is especially important given that major infrastructure projects, funded by the Infrastructure Act, are likely to impact EJ communities the hardest.

The Noise Control Act of 1972 was never rescinded but was defunded 40 years ago by the Reagan Administration. However, that act is still the law of the land, similar in strength and scope to the other core EPA environmental laws enacted around that same time. The act contains a number of nondiscretionary mandatory requirements for EPA action that are not currently being fulfilled. Addressing them again as they were before the program was defunded would lead to major improvements in public health.

Quiet Communities petitioned the EPA in 2017 to reestablish the Federal Noise Control Program. We've written to members of Congress to provide funding to EPA to do so. We've submitted comments to NEJAC. We are in discussions with EPA and have proposed a modest budget to reestablish the program. Providing assistance and facilitating funding for things like highway sound barriers and sound insulation, enforcing existing noise regulations, and creating quiet green spaces along with infrastructure improvements would go a long way to mitigating the problem in EJ communities. Much of this is low-hanging fruit.

We urge the Council to include noise as an addressable problem. In particular, we ask the White House to request that Congress again fund the EPA to implement the Noise Control Program and ask that noise be integrated into EPA, public health, other relevant programs, and screening tools to protect EJ communities from the unnecessary impacts of excessive noise.

1.5.2 Amber Reimondo - Grand Canyon Trust (Flagstaff, Arizona)

Amber Reimondo stated that she is the energy director for the Grand Canyon Trust, a regional conservation organization based in Flagstaff, Arizona. Our mission is to safeguard the wonders of the Grand Canyon of the Colorado Plateau while supporting the rights of its native peoples.

We're thankful the Biden administration has committed to take action against climate change and to support environmental justice communities, but as a group that works with frontline indigenous communities, we worry that if done improperly, the Biden administration's climate policies could inadvertently worsen rather than ameliorate environmental injustices in tribal communities. With a shift away from fossil fuels, mineral supply chains, mineral recycling, and long-term radioactive waste disposal grow more important by the day. So, it's critical that despite the unspeakable urgency of addressing climate change, the administration moves with great care and attention to the concerns and needs of indigenous communities.

Importantly, past and present mineral extraction, processing, and waste are major perpetrators of environmental injustice on the Colorado Plateau and across the country. Yet, the environmental justice screening tool, for example, doesn't have a layer for hard rock mines or processing mills, which are often near tribal communities. According to one estimate, more than 600,000 Native Americans live within ten kilometers of abandoned mines. The Navajo Nation alone is home to more than 500 abandoned uranium mines and existing operations like the White Mesa Uranium Mill in southeastern Utah and the Pinyon Plain Uranium Mine or the Grand Canyon are long-standing examples of ongoing threats to the health and cultures of indigenous communities.

We'll send more detail of written comments, but to highlight a few risk areas, we hope that WHEJAC can emphasize, or in some cases, reemphasize for the administration. The first is that the world still lacks a safe long-term waste disposal strategy for spent nuclear fuel and the full nuclear fuel cycle has and continues to disproportionately impact indigenous communities, including here in the U.S. These are among the reasons that the European Union, for instance, has not prioritized investment in new nuclear energy.

Second, the White Mesa Uranium Mill in southeastern Utah has long been seen as a threat by the Ute Mountain Ute Tribe, whose White Mesa community lives right next door. The mill was supposed to close in the '90s, but it has repeatedly rebranded itself and now markets itself to the Biden administration as a possible domestic source of rare-earth metals for the buildout of renewable energy. Hundreds of acres of toxic and radioactive waste that the mill produces will remain next door to the White Mesa community in perpetuity.

And lastly, in general, the Biden administration must do more to actively seek out the direct input of tribes and tribal communities who are impacted by hard rock mining, mineral processing, and the toxic and radioactive waste that these operations leave behind. The Havasupai Tribe and Navajo Nation, the Hopi Tribe, and Ute Mountain Ute Tribe are a few among a long list of tribes across the country whose input should be actively, not passively, sought out by the administration.

1.5.3 Emily Tracy - Community Advisory Group (Canon City, Colorado)

Emily Tracy stated that she chairs the Community Advisory Group for the Lincoln Park Cotter superfund site just south of Canon City, Colorado, a rural community. The Cotter Corporation Uranium Mill began operations in the late 1950s. Contamination of radioactive materials in the groundwater, surface water, and soils at the site and a nearby neighborhood was discovered as early as the 1960s and '70s. The site was designated a superfund site in 1984, about 38 years ago. Despite multiple environmental investigations, lawsuits, the involvement of the Colorado Bureau of Investigation, in addition to the regulatory agencies in the '70s, '80s, and '90s, the uranium mill was allowed to continue to operate until about 2010 when it was closed permanently.

Currently, the site is slowly working its way through the superfund clean-up process. Prevention is key. A uranium mill should never have been allowed at this location. It is upstream from the community and a major source of drinking water. In fact, there was a severe flood in 1965, which washed radioactive contamination from the site into the Lincoln Park neighborhood. Property owners there have not been able to use their private water wells for decades due to contamination from the site. The mill site is completely underlain by coal mine tunnels and shafts from a century ago, possible deep pathways for contamination. High winds blew uranium mill tailings for many miles in the past, and there was no uranium mine of any size within 100 miles. Uranium ore was hauled in from far away.

It's important to also note that radioactive waste materials were hauled in from other areas around the country and beyond for processing at the mill, including Manhattan Project Waste. Members of the Community Advisory Group believe that there are substantial environmental justice issues due to the high rate of poverty in the community. The county's median household income is 32.3 percent below Colorado's statewide median household income and far lower than the median household income for larger cities. There's a significant retirement population in the area, and many of them live on very limited incomes.

We believe that hand in hand with a significant level of poverty is limited political power. Most residents do not even attempt to use their voice on critical issues like this one. It has been an uphill battle for a long time. The superfund site Community Advisory Group and the community as a whole would like more assistance in assuring the full investigation of the contamination is completed and a full clean-up of the site is ultimately conducted.

1.5.4 Alicia Scott - Partnership for Southern Equity (Atlanta, Georgia)

Alicia Scott stated that he was speaking up for rural America regarding rural electric cooperatives. Rural electric cooperatives serve 90 percent of the counties, federally recognized for persistent poverty, and yet, the member-owners of those utilities face some of the highest energy burdens in the nation. These persistent poverty counties include broad swaths of the Southern Black Belt Region, Appalachia, tribal lands, and the U.S. border. It is essential that a path to a lower-carbon future does not drive these rural electric cooperatives and the communities to own them further into debt.

Here's a historical fact; rural America required federal investment through FDR's New Deal to bring electrification to the countryside through the Rural Electrification Act of 1936. As a result, rural electrification improved by 70 percent between 1936 and 1950. We are now in an entirely new century, and the only policy concerning rural electric cooperatives that have been submitted or presented in the last 70 years has been only to exclude them. Rural electric cooperatives have been historically disenfranchised from federal incentives for clean energy while investor-owned utilities have been able to leverage robust state and federal tax credits to provide proactive investments. RECs, or electric cooperatives, have not.

So, here are just a few points that we'd like this White House Environmental Justice Advisory Council to recommend to the Biden administration. First, ensure that any debt relief to rural electric cooperatives is conditional on new investments and local energy upgrades in rural communities on broadly equitable terms. Next, ensure that Justice40 recognizes and includes in its definition the counties that are already federally recognized for persistent poverty. Next, ensure that President Biden fulfills his pledge as a candidate to implement the 10-20-30 rule. This is a policy championed in the House and Senate right now to assure that ten percent of federally funded programs be directed to persistent poverty communities.

Next, ensure the Department of Energy actively participates in a whole of government response because it can work with every single rural electric cooperative in the country to chart a just clean energy transition. And lastly, ensure meaningful engagement at the community level for every new application for federal financing from the rural utility service and every issuance of debt for large public utilities, such as Tennessee Valley Authority.

1.5.5 Emma Kurnat-Thoma - Georgetown University (Washington, D.C.)

Emma Kurnat-Thoma stated that she is a registered nurse and adjunct faculty at Georgetown University. She has 24 years of experience and a long history of serving the Catholic church, including environmental health interests. She is commenting on environmental justice for healthcare systems. Pope Francis authored the encyclical document Laudato Si' in 2015. It is a powerful contribution by the Catholic church to the global dialog on the climate crisis. Laudato Si' is based on integral ecology, which defines the nature of shared bonds between humans and the natural world where everything is interconnected.

Laudato Si' consists of two prongs: hearing and responding to the cry of the earth and to the cry of the poor. This means that the earth's resources must be preserved and protected by reducing poverty and lifting up those in marginalized peripheries. Healthcare systems and policy leaders can use integral ecology to complement scientific planning and local communities. Policy, healthcare, and faith leaders working together can address the climate process by linking COVID-19 reforms to design strong, equitable, and climate-resilient healthcare. During crises, integral ecology assists policy leaders to place the strongest science at the source of those who are most profoundly vulnerable. To strengthen healthcare system climate emergency planning and responses, a total of three recommendations are suggested to harness the nursing workforce, which is 4.2 million members strong.

Number one, re-review the joint commission centers for Medicare and Medicaid services emergency management standards to ensure adequate nurse workforce preparedness. This includes healthcare access plans for the inclusion of vulnerable and marginalized populations and critical access hospitals and hospitals in very high-risk climate forecast areas for extreme weather events, such as tornadoes, hurricanes, wildfires, and floods.

Number two, ensure adequate nurse healthcare workforce planning for environmental justice, and integration of social determinacy of health, to better support vulnerable and marginalized individuals and climate crisis impacted communities with documented physician shortages. The "Future of Nursing 2020 to 2030" consensus report provides detailed plans for community and population health nurse models of care for a wide range of health equity applications.

Number three, ensure HHS's climate action plan for climate resilience and readiness, incorporates clinician healthcare workforce planning and collaboration with key healthcare provider advocacy organizations, including American Medical Association, American Nurses Associations, and American Hospital Association. WHEJAC and the Biden Harris Administrations' commitment to the service of all humanity is supremely commendable.

1.5.6 Debra Butler - University of Massachusetts (Boston, Massachusetts)

Debra Butler stated that she is a PhD candidate in the School for the Environment at the University of Massachusetts, Boston. Her research is in partnership and centered in communities along the northern Gulf Coast of Mexico - Alabama, Mississippi, Louisiana, and Texas - the most diverse, ecologically complex, and environmentally exploited land, water, and air in the United States. I'm a native of the Gulf. It's my home.

She stated that her comments are directed to the Build Back Better initiatives that authorize investments of more than $110 billion to roads, bridges, and transformational projects. Build Back Better, in addition to massive investments in steel and concrete, must first acknowledge, mitigate, and repair past and current federal, state, and local adaptations, mitigations, and recovery programs that continue to damage the lives and livelihoods of place-based native Asian and African American communities on the Gulf Coast. These long-tenured communities are not inherently weak, broken, or non-adaptive. They're being systematically marginalized, disinvested, and displaced because they're inconvenient to commercial, economic, and extractive interest.

Where we build is as important as how we build. In particular, she critically examines improvements and expansion of destructive activities such as sand mining beaches and erosion control, exploiting wetlands to transport oil and petrol chemicals, and opening spillways and dams that reduce the salinity and increase turbidity in waters that support marine life and health, the lifeways of fisher focal on the gulf. These interventions along rivers and the wetlands and estuary system and in deepwater ports are activities that exponentially increase vulnerabilities and institutionalize the structural precariousness of place-based communities on the Gulf of Mexico.

We cannot build back in the same places and the same ways. The White House fact sheet specifically states that this infrastructure deal makes the single largest investment in repairing and reconstruction of our nation's bridges and some construction of the interstate highway system where the legacy of that system is to one of displacement, particularly in communities of color, particularly along I-10 from Jacksonville, Florida all of the way across the Gulf to Los Angeles. The 1956 act allows dredging, fill-ins for dams, levees, and fragile estuary systems. With the memorandum on adjusting our nation's and the federal government's history of discriminatory housing practices and policies, the Biden White House 2021 addresses policies implemented by this act.

She suggests that the policies it puts forth be reversed and that we require structural environmental justice regulations and their implementations. It's the only pathway the Build Back Better projects are relevant and transformative.

1.5.7 Dee Allen - t.e.j.a.s. (Houston, Texas)

Dee Allen stated that she will defer to Juan Parras, who will speak later.

1.5.8 Laura Olah - Citizens for Safe Water Around Badger (CSWAB) (Merrimac, Wisconsin)

Laura Olah stated that she is a member of the Mole Lake Sokaogon Chippewa Tribe in Wisconsin and acknowledged her great grandmothers. She is the executive director of Citizens for Safe Water Around Badger and a national organizer for the Cease Fire Campaign. Right now, in communities across America and its territories, the Department of Defense, the Department of Energy, and Industry are permitted by the EPA and authorized states to conduct open-air burning and detonation of hazardous waste causing the uncontrolled release of heavy metals, energetic compounds, perchlorate, nitrogen oxide, hexavalent chromium, PFAS dioxins, and other carcinogens to the environment and permanently polluting our water.

These burn pits are not just overseas. They're here at home. She and her neighbors organized Citizens for Safe Water Around Badger 30 years ago when rural families near the Badger Army ammunition plant learned that their drinking water wells were polluted with chemicals from an open burning area that was more than three miles away. Today, 30 years later, the Army is still testing our drinking water wells for the explosive DMT, having spent more than $250 million on our remediation effort. There are three major groundwater contaminate plumes that have migrated off-site, and two of them are coming from the open burning areas inside the plant. In September 2020, total DMT was detected in groundwater. Yet, concentrations as high as 1.2 million nanograms per liter, which is 25,000 times higher than the groundwater standard of only 50.

In active bases like the Crane Naval Surface Warfare Center in Indiana, the military has been allowed to open burn and open detonate as much as 109 million pounds of hazardous waste every year. Emerging contaminants like PFAS are part of this waste stream. Military countermeasure flares may contain as much as 45 percent PFAS. PFAS are not destroyed in an open fire or even by incineration, instead are continually dispersed to the air, contaminating people, as well as fish and wildlife and permanently contaminating drinking water and groundwater sources.

Even though safer alternative technologies have been successfully deployed by the military in the private sector in some communities, this devastating practice continues almost exclusively in communities without the resources to defend themselves from this relentless source of toxic exposure. These are our domestic burn pits. They're here at home and almost exclusively in communities that are most vulnerable to harm. We need to break the cycle. We need your help to make this happen.

1.5.9 Heather Toney - Environmental Defense Fund (Mississippi)

Heather Toney stated that she serves as vice president for community engagement at the Environmental Defense Fund. I'm coming to you from the ancestral land of the Choctaw and Chickasaw people in the State of Mississippi. As a committee, it's comprised of members under no disillusionment within climate crisis and the disproportionate impact felt by underserved and marginalized communities across the country. For every moment our country delays response to this emergency, we become more and more responsible for the business livelihoods lost and families destroyed because of the failure to act when we have both the science and the resources to back our actions.

What's worse is that inaction will multiply the impacts of communities of color and marginalize communities fighting simply to experience equal access to elements necessary for life: breathable clean air, drinkable water, and nontoxic land. EDF appreciates the commitment of this Advisory Council to hold all of us accountable for equitable solutions and stand as an ally to this work. The all-of-government approach is supported by an all-of-community approach compiled of advocates like local leaders, large environmental groups, and leaders in philanthropy and business to support community-centric responses. We recognize that government alone cannot solve the climate crisis by itself.

Two months ago, EPA administrator, Michael Regan, sat on porches and in front yards of the people in Mississippi, Louisiana, and Texas who are overburdened by air pollution and threatened by climate change. He clearly heard them and the solutions he's put forward to help every community address specific need in each of them. EDF encourages the expansion of these efforts through legislative opportunities like the Technology Assessment for Air Quality Management Act introduced by Senator Markey. The bill would not only strengthen hyperlocal air quality insights but also require EPA to better integrate environmental justice mapping tools focused on cumulative harms and approve the quantities use of low-cost sensors and data from satellites and decision making.

Moving forward, it's critical that the Biden administration seriously considers and implements the White Environmental Justice Advisory Council's recommendations, which outline a strong path for equitable and just climate solutions. She stresses that this is a starting point. There are many climate solutions that still require deep listening and understanding in order to ensure that transformative climate solutions do not sacrifice frontline and fenceline communities. We're committed to a system of implementation of Justice40 initiatives and look forward to working with you on this effort.

1.5.10 June Farmer - Marin City People's Plan (Marin, California)

June Farmer stated that "Just as the water is a foundation of life, there must also be the foundation of design in a built environment." That's a quote that I love to quote when we're talking about climate change. She's the program director for the Marin City People's Plan, which is a grassroot, community-driven project, and she's also a member of the Anthropocene Alliance.

Marin City is a small predominantly black, low-income, unincorporated town in one of the wealthiest counties in America - Marin County in California. Marin City has a history of flooding so much that we are warned that in case of emergency, we should keep enough food, enough water, and enough emergency supplies on hand to last three to seven days because the county emergency departments will not be available for us even though we have a substation here in Marin City. During the heavy rainstorms in an atmospheric river that we had last October, there were threats to our residents' livelihood. Marin City is a town that only has one way in and one way out, and unfortunately, that's where it floods. We have to fight against sea-level rise and flooding issues that have impacted our health for over 75 years, and this problem still exists in this community.

The Marin City People's Plan finds it extremely important to not only address these issues in meetings but start using nature-based solutions and building green infrastructure. We find it very important to use the community as well as part of the solution. By empowering the community, we can design flood adaptation by allowing nature to combat nature, which is a must. Funding a community and building a resilient community is a must. Another extremely important need is that Marin City needs other roads going in and out, and that plan needs to start now, not in the middle of an emergency. And just like the quote by Betsy Damon says, "Just as the water is the foundation of life, it must also be the foundation of any built environment" and not be ignored."

1.5.11 Liz Scott - American Lung Association

Liz Scott stated that she is the National Director of Advocacy for the American Lung Association's Healthy Air Campaign. She talked about regulatory actions that this administration needs to take to protect health and deliver on environmental justice commitments. The former administration rescinded the appropriate and necessary finding that allowed EPA to set standards for the admissions of mercury and air toxins. Those standards were popular and already fully implemented at the time the finding was rescinded. Mercury is a potent neurotoxin that can cause devastating birth defects including brain damage. And other toxic chemicals emitted from power plants threaten the air nearby communities breathe.

Pollution controls that were already implemented were estimated to prevent up to 11,000 premature deaths annually. Due to systemic racism, the communities living within the radius of a powerplant are disproportionately communities of color. Further, elevated levels of mercury in fish can have a disproportionate impact on specific populations, including Native Americans. The reinstating of the appropriate and necessary finding is a justice issue. Despite the president calling for this rule as soon as possible on day one in office, the proposed rule appears to be stuck at the White House Office of Management and Budget going on six months. And the broader health community is very disappointed that we are still waiting for the proposal.

The Biden administration needs to propose this rule, finalize it, and then follow the science and the Clean Air Act to set even stronger mercury and air toxin standards. Another upcoming rule will look at setting pollution standards for heavy-duty vehicles. With communities of color often located near highways, ports, and transportation hubs, people of color bear a disproportionate burden of some of the most visible and dangerous pollution. Setting stronger standards for trucks will yield health benefits across the board, but especially in those areas hardest hit. It is critical that the administration quickly propose this rule so it can be finalized before the end of 2022.

We also encourage EPA to swiftly move through reviewing the 2020 particulate matter in ozone air standards. The 2020 review discounted key science and ultimately finalized standards that were not adequate to protect public health. A study out today from the Health Effects Institute found that if the PM standards were even slightly stronger, as many as 143,000 deaths could've been prevented over a decade. The Lung Association State of the Air report found that people of color were three times more likely than whites to live in areas with the most polluted air. We appreciate that this administration is reviewing the science and we urge the swift, yet robust finalization of stronger standards.

Finally, reducing emissions from passenger cars will provide immediate health benefits, long-term climate benefits, and will accelerate the transition to zero-emission vehicles. The recently finalized standards are an important step, but they must be followed with the proposal to the next round of model years if we're to continue that progress. Everyone's health is impacted by air pollution, but some communities have continued to be left behind on the quest for clean air. We appreciate this administration's commitments to environmental justice, and we look forward to holding the administration accountable to those commitments.

1.5.12 Yvonka Hall - Northeast Ohio Black Health Coalition (Cleveland, Ohio)

Yvonka Hall stated that she is the executive director of the Northeast Ohio Black Health Coalition, but she's also the president of CLASH, Cleveland Lead Advocates for Safe Housing. The Northeast Ohio Black Health Coalition, in our mission, is to address disparities and equities in education, employment, housing, and health and the impact on African American disparities by working to educate, advocate for, and empower the community. Waiting for environmental justice, environmental toxins and pollutants know no class or race. And yet, the government policies and corporate activities place an undue burden on the health of the poor and communities of color.

In Cleveland, Ohio, we talk about Flint, Michigan all the time about lead, but in Cleveland, Ohio, our rates are twice as high as Flint, Michigan. Their rates are about 7.2 percent. In Cleveland, Ohio, our lowest rate is 14.2 percent. We have some communities where 50-some percent of the children are lead poisoned and where 92 percent of kindergarten classes have tested positive for lead. More than 40 percent of the students and some Cleveland schools have elevated levels of lead poisoning. Ninety percent of our homes in Cleveland have lead in them. It so important that we understand that lead correlates the cradle to prison pipeline because of how lead impacts children's brains. Remember that lead goes from your blood into your bones and then to your other organs.

So, it is important that we not only concentrate our efforts and our government efforts on changing lead water pipes in our urban areas but also that we do something about the housing and housing standards to ensure that no child is living in a home that is impacted by lead. So, dollars have to be put into where the problem lies. And in our urban core, the problem lies in older homes, elder stock homes that people are renting. So, for us, we know that a lot of families had that child tax credit, and they don't have it now. Those things were helping us to create equity around housing because it allows families to be able to come up with first-month deposits for rent.

Also, stimulus dollars, some families still have never received them. We need to make sure that we're looking at that, and we also have to understand that if racism is indeed a public health crisis, then the biggest crisis that we have is some of our urban areas is lead poisoning. Lead poisoning is public enemy number one in our urban core. Creating equity and making sure that our communities are safe should be the number one responsibility of all of our government, making sure that people have the dollars that they need to live is important for us.

1.5.13 Susan Liley - Citizens' Committee for Flood Relief (DeSoto, Missouri)

Susan Liley stated that she lives in DeSoto, Missouri, a tiny rural town in Missouri. It flash floods. She does not flood, but our town does. We formed a group in 2016 after we had several people wash away, and we founded the group called Citizens Committee for Flood Relief. She is the co-founder, and we quickly became a member of the Anthropocene Alliance group, the nation's largest flood group. We have had an evacuation every year since 2016, and we had four floods in the three years before that. Not much has changed in the years other than we have gained national attention for flood fighting.

We've had a U.S. AC flood study that designated 229 homes as troubled, 79 for buyouts, and the rest were for elevations or other flood measures. That's out of a town of 6,400. We've also had a U.S. GS study that backed up, but nothing has been done other than we have an emergency plan and a flood gauge. The part that bothers me most is two blocks of their historic Old Main Street are called for a buyout in the five-mile upper watershed. Of the tiny Joachim Creek, 15 dams were used for lead mining. There is lead and arsenic that flow down with every flood. One of the dams is a class two flood hazard, and that terrifies me. It's controlled by a 2 by 8 board. Through our group, we've kept the community aware of the problems, but there have been no real fixes. I worry because as hard as I've tried, our governor will not do anything. Our congressman and senators have been aware of the problem for years.

She looked to D.C. when COVID first started, and she took pictures directly to her congressman. He asked her why we had not helped ourselves. She also watched as the EPA digs the yards of these homes out, knowing that it is costing as much to dig the yard as the home is worth. It will still leave the contaminated foundation and the home for the people that live there. There are few, if any, disclosures when these houses are resold or bought by people to rent out. Some homes are just left behind and a squatter moves in and, eventually, it burns down while trying to stay warm.

Last winter, we lost a building that was almost a city block and the people across from it had their siding and their windows melt. In 2003, we had a young father wash through a drainage tube in his small car in the middle of town. It took ten days for them to find him. In 2016, we had an elderly lady wash down right in front of their rural fire department. They could not help her because they were underwater, too. There's nothing but a safety plan and a water gauge, and we need help as soon as possible.

1.5.14 Lorena Venegas - Moms United in East Haven-Mamas Unidas en (East Haven, Connecticut)

Lorena Venegas stated the land that she's concerned about is indigenous land. It's Quinnipiac and Momauguin land here in East Haven, Connecticut, which is a shoreline suburban town adjacent to New Haven that abuts Long Island Sound. So she lives climate change every day here in her town, and her number one concern is water and flooding. As a member of Anthropocene Alliance and also 10,000 Hawks, we've been able to use media, use public comment, and use our methods to gain information, but we don't have all the information at our disposal. That's where my environmental justice issue is most at stake.

Right now, her 13-square mile town and 29,000 people for over 30 years have flooded. We only have one evacuation route, and that route will flood this Saturday when we have a snowstorm. It's become such a complacent issue that most people just literally go with the flow, but that's not the way it has to be. Our town lacks engineers, lacks scientists, lacks soil scientists to help us mitigate what's happening in my town. Even though there are natural forces such as tropical storms and hurricanes that absolutely impact the flooding that's happening, there are also development and zoning issues where we fill the wetlands and we've been elevating pavement so high that water runoff and displacement is actually causing a lot of our street flooding. We don't have store water management plans in my town. We don't have mayors that go ask for the money at the state level.

What this is coming down to is that the wealthy residents here can stilt their homes. They can pay for the FEMA flood insurance and pay for the FEMA contractors that are required, but the poorer residents cannot. In May of last year, we've been confronting now Tweed New Haven Airport which wants to expand and part of the master plan is really a destruction of our land here. We live in a flood zone, and that flood zone right now has negative impacts including air pollution that are impacting our children and seniors, seen in our asthma and respiratory incidents raised for hospitalization. We have higher noise pollution, light pollution, and traffic.

Statistically, our residents have a higher incidence of hospitalization, and our government officials are presenting contradictions. On one side, they want to conserve and preserve their shoreline, but on the other side, we see that they're aligned with the private investors that are here to make the money off the flights of Florida. That's not enough for us. So, her ask tonight is, number one, she needs help with the NEPA. She needs help with NEPA guidelines so that she can make her town have an environmental impact statement. We don't want just an environmental assessment.

We need to make sure that EPA backs us up so that we have a true community benefits plan, that there's no building on any wetlands anymore, more flood mitigation for local help, and have more information for the public because we have a right to quiet enjoyment.

1.5.15 Brenda Staudenmaier - Clean Water Action Council of NE WI (Madison, Wisconsin)

Brenda Staudenmaier stated that she works in the water industry in Madison, Wisconsin protecting human health and the environment. She has a degree in environmental engineering with a focus on water. In 2017, she, her children, and others filed a federal lawsuit against the U.S. EPA using the Toxic Substances Control Act because fluoride added to the public drinking water supply is an unreasonable risk to the developing brain. We are back in federal court on June 7th of 2022. Science funded by the National Institute of Health published since 2017 finds fluoride at the levels purposely added to the public water supply is equally as toxic to the brain as lead.

If a pregnant mother exposes their fetus to fluoride during pregnancy or through bottle feeding their baby using tap water, they can expect to see a reduction in IQ and an increase in ADHD in their children, similar to what we see with lead exposure. Protecting people from fluoride harm costs nothing. All we have to do is stop the program. You should know that fluoride in the U.S. drinking water is recognized as a water pollutant by the EPA, and fluoridation policy has been recognized as an environmental justice issue by civil rights leadership for over a decade. None of the EPA-approved lead and arsenic-laced fluoridation chemicals originating in the pollution control systems of industry have ever been safety tested.

Water fluoridation began in the early 1940s when we were also singing the praises of asbestos. Fluoride began despite dentists knowing fluoride could stain and pit children's teeth with dental fluorosis. Today, 70 percent of U.S. children and adolescents are afflicted with dental fluorosis on at least two teeth from ingesting too much fluoride. This effect is disproportionate by race. Yet, the oral health report in December of 2021 shows that, despite increases in public water fluoridation, dental visits, sealants, fluoride varnish applications, and significant financial training and program incentives, tooth decay persists, especially in minority populations who are most harmed by fluoride ingestion in ways other than stained teeth.

Fluoride has adverse effects on the kidneys, thyroid, bones, and immunity, but the most appalling effect is fluoride's damage to the developing brain backed by hundreds of animal studies, and 74 human studies link fluoride to lower IQ and increased rates of learning disabilities like ADHD. We are spending over $15 billion to remove lead from drinking water because it harms the brain while purposely adding something just as toxic to the brain. Fluoride is the simplest environmental justice problem to fix and requires no money. All we have to do is turn off the fluoride dosing pumps.

1.5.16 Harriet Festing - Anthropocene Alliance

Harriet Festing stated that she is the executive director of Anthropocene Alliance, which is the nation's largest coalition of frontline communities fighting for climate and environmental justice. We currently represent 110 members in 34 U.S. states and territories, 75 percent of our members represent low-income, black, Latin X, and indigenous communities, and 78 percent of our members are led by women.

The people that our members represent have had their homes and neighborhoods flooded, burned, and contaminated. Many have been displaced as a result and/or faced terrible health consequences, and continuing government inaction puts them in ever greater risk. What's kind of extraordinary as we've set up Anthropocene Alliance is really just how little government support there is out there for the communities we are working with. And so we've been trying to bridge that gap by connecting our members with pro bono services. Our new frontline 360 services bring pro bono scientific, legal, technical, and policy support thanks to partnerships with the Environmental Integrity Project, the Environmental Law Institute, the Environmental Protection Network, and the Thriving Earth Exchange. Thus far, since 2017 when we launched, that pro bono support has been worth 3.3 million.

We also fundraise on behalf of our members, so we both help our members write the funding proposals, we advocate for grant funding for them, and we apply on our member's behalf and distribute the funds. The funds raised thus far has been $16.7 million. She thanked Dr. Bullard, who spoke earlier. Organize, mobilize, shape, that's what our 21 members signed up for. That's what they want, their voices heard.

We're happy to act as a connector between them and the policies that are being shaped. For those of you who have given public comment today who haven't heard of Anthropocene Alliance, please join us. We want you. Your stories are painful and disturbing and powerful, and we need you to join us to organize and mobilize.

1.5.17 Jacqueline Jones - Reidsville Georgia Community Floods (Reidsville, Georgia)

Jacqueline Jones stated that she is the leader of a group called Reidsville Georgia Community Floods, ZIP Code 30453, population just over 2,600. My group is a member of Anthropocene Alliance, a national nonprofit that educates and organizes individuals and communities harmed by environmental abuse and climate change. She's also a member of their mutual aid fund. Reidsville, Georgia is an impoverished community and the people in this area are basically closed off from the rest of society. Many are uneducated and elderly. So, if you ask most people in this area, they will not know what environmental justice is. What is environmental justice? These words can mean many things.

Environmental justice is desperately needed in this area, even though because of generations of oppression, people have normalized the erosion, the devastation, the mold, the sewage, the nuisance, and the inconvenience of flooding. Shockingly, people in this area do not look upon the water that stands for months at a time on their properties as flooding. Drains being 50 years old and inadequate or they're not being a drain whatsoever or being incapable of getting in and out of their homes as being wrong because they have lived with it for decades. She moved there four years ago based on an outdated FEMA flood map and immediately got flooding with three feet of water up to my windows that sat in my yard for months at a time.

In my opinion, it's not the fact that many homes were allowed to be built in this area on top of swamps or that the city and city account officials refuse to do anything, including applying for grants that would help resolve the situation, it is the mentality of the citizens of this area. Although she is not in the best of health, she has passed out flyers to close to 300 people and only two people so far have contacted me as a result of her efforts. There needs to be a federal outreach program to inform people that, yes, there is help and assistance available. This could be accomplished through direct mailing using the U.S. Postal Service.

Reidsville, Georgia is in serious need of environmental justice through educating its marginalized and underserved citizens on the hazards of health and issues that are caused by flooding. Finally, the WHEJAC climate-resilient section can work towards stopping the forcing of individual citizens to go through their county and city and state to get assistance from the federal government because, in her area, city officials refuse to apply for help, leaving many in town exasperated and their hands tied.

1.5.18 Sabina Perez - Office of Senator Perez (Guam)

Sabina Perez stated that she is currently a senator in the 36th Guam Legislature, a native Chamorro woman. She would like to express her sincere appreciation to the Biden administration for their leadership and addressing climate change and recognizing the importance of including those that are most impacted by federal policies and actions on our communities. As tomorrow is our connection to and the protection of our land and environment are central to our culture that has evolved over 3,000 years in Guam and the Mariana Island. One of the biggest issues is the persistent lack of meaningful public input due in large part to our unincorporated status as our island faces the largest militarization since World War II.

Under the framework of NEPA, which serves as an umbrella for environmental laws, environmental impacts of federal actions must be considered before the finalization of plans. If negative impacts are identified, mitigating measures must be analyzed. However, the least damaging alternative is not required. The public comment period for NEPA has often been pacified by the community because the outcome has been privileged and has been the privilege of military projects despite serious community concerns that are either minimized or not responded to. Additionally, the review of voluminous environmental impacts requires a level of technical knowledge and a significant amount of time for review. Under the National Historic Preservation Act, the programmatic agreement workshops are not open to the public, and it is questionable whether consulting and interested parties are adequately sought. In the implementation of the Endangered Species Act, the finalization of the biological opinion occurs under closed doors between federal agencies with minimal, if any, opportunities for public input.

The impacts of militarization are widespread, and it extends beyond the fence and into the community. Two projects that I would like to focus on requires immediate attention. One is the construction of the live-fire training range complex over our northern aquifer, which supplies over 80 percent of our island with drinking water. This firing range consists of five ranges that, in full operation, are expected to release 6.7 million bullets containing lead and other hazardous substances every year for an indefinite period of time. It also entails the creation of the surface danger zone that extends into the Guam National Wildlife Refuge and the adjacent ocean will be off limits to our traditional practitioners, such as our fishermen, and it also limits access to our cultural healers who collect our medicines.

Further, we witness the destruction of our ancestral lands with the establishment of a Marine base. We've seen the destruction of our ancient historical village of Magua being destroyed before our very eyes as well as the destruction of our primary precious limestone forest, which took millennia to develop. She sees it as a natural wonder of the world. In addition, this ecosystem has supported endangered species that are found nowhere else in the world, one of them is a very critically endangered tree, the hayun lagu tree. The largest of the firing ranges will be placed 100 feet away from the mother tree. It's the only reproductive tree found in Guam.

So, that is one project that requires immediate attention. If there's anything that could be done to protect this ecosystem. Additionally, the other project that she's focusing on is the open burn/ open detonation. Legal loopholes exist currently that allow a continuation of toxic releases through this antiquated method, which is the U.S. military's preferred method of disposing of their energetic waste. She will send the rest in written comments.

1.5.19 Lynne Bonnett - 10,000 Hawks (New Haven, Connecticut)

Lynne Bonnett stated that she's part of 10,000 Hawks, which is a group in New Haven that's concerned about the expansion of Tweed Airport, and she participated in the Connecticut state's governor's council climate change meetings that started meeting in 2019. They're very similar to what Krystal Laymon from the CEQ said earlier. They divided into working groups creating reports. All of our reports are final. They're published on the website of the state. They all address climate change, and the reason she's concerned is that the consultants doing an environmental assessment for Tweed have said that there are no formal regulations or standards for climate change at this time, and the FAA-approved process governing the environmental assessment process.

So, here we are, two-plus years that the state has been working on this really hard, producing all of these reports, and the FAA is saying that they don't have anything that they're going to use. So, in order for them to address climate change, they plan to measure greenhouse emissions. She's not sure what protocol they're going to use, but the City of New Haven just gave a report on greenhouse gas emissions using the ICLEI USA protocol and Clear Cuff and they presented it to a small group of citizens that she attended. They totally left out all of the host entities in New Haven, like the power plant, the deep-water port, the sewer plant. All of these heavy greenhouse gas emitters were not included in this report.

The methodology that they're using is something that's supposed to become standardized so cities can compare their values across geographical areas. They can use it as metrics to determine how much progress they make. So, she's hoping that the Council can look into this because, if it's a metric that's going to be probably used, it needs to accurately reflect what's going on in environmental justice communities like New Haven with a lot of host activities that were totally left out of this greenhouse gas emission report. So, she doesn't know what the airport's going to do, but the airport was left out of the city's greenhouse gas emission also.

So, there are some issues that she also included in her written report that had to do with the environmental assessment and her problems with how they're proceeding.

1.5.20 Tina Cordova - Tularosa Basin Downwinders Consortium (TBDC) (New Mexico)

Tina Cordova stated that she is a native New Mexican, a downwinder, and a cancer survivor. She's the fourth generation in my family to suffer from cancer since the Trinity bomb was detonated in South Central New Mexico, on July 16th, 1945. She grew up in a community about 45 miles away as the crows fly from the test site although we've identified families that lived as close as 12 miles. The U.S. government has always controlled the messaging as it relates to the test and has said and continues to this day that the area was remote and uninhabited and that no one lived here, and no one was harmed.

The truth is, according to the 1940 census data, there were about 15,000 men, women, and children living within a 50-mile radius of the Trinity site and close to 500,000 people living within a 150-mile radius. It is obvious from these numbers that it was neither remote nor uninhabited. We've been dealing with the devastating health consequences for 77 years now without aid or assistance from the government. The people of New Mexico are the forgotten collateral damage of the first nuclear test. Most of us are indigenous people of color, Native Americans, or Mexicanos. We bury our loved ones on a regular basis and then another one of us is diagnosed with cancer. It's time to recognize that mistakes were made and correct the injustices perpetrated on American citizens.

To remain complacent in the face of knowing about the injustice of nuclear testing and subsequent damage to the health of human beings renders a person complicit in injustice. There is a moral and ethical imperative to write this wrong. There are bills before Congress now to amend the Radiation Exposure Compensation Act that will expire in July of this year if not amended that would add us to the fund. The bill is House Bill 5338 and Bill 2798. While we bring these bills to the attention of the Council, our recommendations center around a different but related subject matter.

In 2019, a paper was published in the *Bulletin of Atomic Scientists* entitled "Trinity: The Most Significant Hazard of the Entire Manhattan Project." The article focused on the high rate of infant mortality after the Trinity test. Since the article was published, we've been doing research, and the record keeping of infant births and deaths in New Mexico in 1945 is quite incomplete. We have reason to believe that babies that died during this time frame were not well documented, and the lack of accurate records may have understated the number of infant deaths. This might have been much worse than reported. It's time for the U.S. government to thoroughly assess this issue and report on the findings. Did we have casualties from the Trinity test and were they our babies? If so, who will be the voice for these forgotten children potentially sacrificed in the testing of the first nuclear device?

We're here to appeal to the Council to reach out to the Centers for Disease Control who conducted a 10-year study called the "Los Alamos Historical Document Retrieval and Assessment" that addressed the radiation releases from the Manhattan Project and asked that they reopen the study to further research the infant mortality in New Mexico subsequent to the Trinity test. The information about infant mortality was not known to the CDC at the time of their study.

Without addressing infant mortality, the LAHDRA study report is incomplete at best. If babies died while the U.S. government looked the other way, it is the most egregious offense imaginable. The people of New Mexico deserve to know the truth.

Chair Moore interjected that they have heard from numerous people regarding military toxins and the environmental racism that's connected to many of the communities that are surrounding military sites. He suggested that the Council has got to deal with the question of military toxins.

1.5.21 Brenda Vallee - Central LA Coalition for a Clean & Healthy Environment (Colfax, Louisiana)

Brenda Vallee stated that she's with the Central Louisiana Coalition for a Clean and Healthy Environment. She lives near Colfax, Louisiana, in an area that's predominantly African American, and she lives less than two miles from an open burn and open detonation site. In 2007, she had a friend's house who blew up and the household members were killed. She experienced the shaking of my home and the uneasy feeling that followed. Today, people around me are experiencing the shaking of their homes following detonations at Clean Harbors Colfax, which is a hazardous waste facility that's nearby.

Some years ago, a local newspaper published an article about hazardous waste being sent from Camp Minden to Clean Harbors Colfax. We learned that over 300 different types of reactive toxic waste were received, stored, and treated at this open burn and open detonation facility. It is the only commercial facility of its type in America. On an annual basis, the facility is permitted to open burn and detonate over 240 tons of toxic waste that had spewed into the air and goes everywhere, including the soil, groundwater, crops, homes, animals, and humans.

In 2017, our organization conducted a health survey. The Rock community, which is an African American community in our area is less than a five-minute walk from the property there to the property at Clean Harbors. The people within the community and surrounding areas have come to know us and trust us and have joined our coalition. When the health survey was completed, we realized that these people had been suffering quietly and dying, and no one had been there to listen to their story. After reviewing the health survey, we realize that we had a major health crisis on our hands.

The cancer incident rate for the census tracks within our area was compared to the rate in the state of Louisiana. Data came from the Louisiana Tumor Registry. The census track surrounding the town of Colfax and the Rock community indicated major health problems. Elevated cancer rates for lung and bronchial cancer, colon and rectum cancer, female breast cancer, and prostate cancer were found and were high. We are an EJ community that has suffered enough, and too many have died. It is time to stop permanently the open burn and open detonation.

1.5.22 Lydia Ponce - Society of Native Nations (California)

Lydia Ponce stated that she's calling from the unseeded territories of the Tongva people known as Saangna, infamously known as Venice. She is representing the Society of Native Nations. We do not have enough time to solve this climate crisis. We need to act as if every minute counts. As the sister elder spoke before, her heart went to her that her refrigerators are empty, and they are lacking food in her territories. So, she's asking this body, these people, this circle right here, right now, the community that we are to reach out back to her and see if we can offer FEMA or better yet mutual aid because that's who we are and that's what I hope we can be within this space and this time.

We need to include our efforts towards healing our lands, our waterways, and our people, and, in reality, this must include swimmers, winged, four-legged, and other two-legged and creepy crawlers. Healing needs to be part of our vocabulary, of our dialog. It's not simply restoration. It's just a cliché as we continue to use terms as alternatives. This should not be part of our plan. We need to call on this elder, and we need to call her back into our circle right now if we can. I'd like to encourage this body, this community circle as a national circle to look at the cities and counties as a micro. We've already created our climate emergency mobilization ordinances and our plans. We know the lay of the land and the challenges that the climate crisis has caused. We need to utilize that information that already exists to build climate transitions and recovery.

She encouraged everyone to consider the power of moratoriums on permits, permits like desalination. We need to put moratoriums on further excavation and extraction. We need to live in harmony with one another and this new position of power, the power of the people. We need to just place moratoriums on anything or any project that's going to cause harm, to extract, or poison, and these industries that have a profit margin are not relatable to anyone here on this call.

As my relatives have offered, either we are part of the healing or part of the hurting and harming. Moratoriums should be utilized if proposals are not healing.

1.5.23 Jonathan Scott - Waste Analytics, LLC (Longview, Texas)

Jonathan Scott stated that he is the president of Waste Analytics. His topic is about oil and gas drilling waste. To begin with, it's important to understand that the United States has had more oil and gas wells drilled than any other country in the world. These wells have been drilled from New York State to California, from the Gulf Coast all the way to Canada. We're talking about millions of oil and gas wells. Some have been orphaned. Some are still producing. Some are actually idled, and some are actually coal zombie wells.

When you look at what has occurred from oil and gas, clearly, oil production has occurred, natural gas production has occurred. The waste streams that have been generated from those oil and gas wells fall into the categories of produced water, drilling waste, and associated waste. Of these, drilling waste is the largest solid waste stream created. For comparison, drilling waste volumes in ten inches or larger is coal ash, which is a well-known concerning contaminate. When you look at the contaminants that are found in drilling waste, you find that they fall into the four broad buckets: Salts, metals, hydrocarbons, and emerging contaminants like PFAS and PFOA.

Once again, if you compare it to coal ash, coal ash has a heavy metals issue, so does drilling waste oftentimes in the same types of heavy metals and in the same concentrations. But unlike coal ash, drilling waste also has chlorides as a proxy for salts, and hydrocarbons, which are both damaging, as well as the emerging, contaminates like PFAS and PFOA and heavy metals. The management methods that are often used when looking at drilling waste are burial, land application, road application, landfilling, injection, and recycling. Oftentimes, the burial is used in states where it's allowed, such as Texas because it's cheap. But what you find from that is the ability for that material, the contaminants that are in it to leach into the soil and to the groundwater. It also happens from land application and road application and poorly managed landfills.

So, what makes this an environmental justice issue? Drilling waste is a large volume waste stream as we've seen that has multiple contaminants that cover most areas of the United States. Soil and groundwater are being impacted as shown by studies in multiple states. In fact, homes in oil and gas areas are being built on top of old production sites as shown by an Oklahoma Corporation Commission presentation in 2013. Human health is and will be impacted as shown by literature reviews by the National Institutes of Health. Also, the environment is being damaged and will require clean up.

Finally, property values are affected by the pollution from drilling waste as discussed in several articles. Unfortunately, rural communities and communities of color bear the brunt of this damaging waste. Put simply, this is an environmental justice issue that impacts most areas of the country.

1.5.24 Rachel Makleff - Grow Renewable New York: No Canadian Hydro (New York City, New York)

Rachel Makleff stated that it is quite uplifting that justice is on the table for action. She wanted to address some unintended consequences of trade that WHEJAC should address. Continuing a tradition of many U.S. green organizations, Grow Renewable New York recently campaigned against a specific electricity transmission line, which would import hydroelectricity from Canada. The electricity would be used for New York City. This Canadian electricity is generated from mega dams, and it creates environmental justice communities often on first nation land in Canada in violation of treaties with the Canadian government.

As her organization, Grow Renewable New York, increased our sophistication on this particular issue, one of our members began petitioning the federal Department of Energy asking it to rescind the presidential trade permit, which would allow the electricity to come over the border into the United States. We wanted to have this done on humanitarian grounds. We care about everybody in this world, and the fact that the people affected were environmental justice communities in Canadian, it didn't occur to them that this was not important.

Now, the answer from the Energy Department was, "This is not within our purview." Now, she's always felt that the trade law should deal with this, but it's very complicated because sometimes people, like ambassadors, can make an exception to the trade law and give a company status even though they're a foreign company as if they were absolutely an American organization, and this is American trading. She hoped WHEJAC deals with this and changes the policy.

Meanwhile, while you're changing it, she hoped that they'll be able to tell all the departments that can possibly rescind trade agreements that are associated with WHEJAC or that WHEJAC has communication with just to allow them to suggest that the presidential trade permit be rejected on humanitarian grounds with no other comment until they figure out more.

1.5.25 Monaeka Flores - Prutehi Litekyan: Save Riditian (Guam)

Monaeka Flores stated that she is a member of Prutehi Litekyan: Save Riditian, a community-based group in Guam, and I'm a descendant of the indigenous Chamorro people who have lived in the Marianas Islands for over 3,500 years. A specific island people, we truly understand how vulnerable our waters, lands, and sacred sites are. Guam has a long legacy of environmental racism resulting from colonization and militarization. The indigenous Chamorro people continue to suffer several ongoing indigenous and human rights violations at the hands of the United States government and military. Guam is currently a U.S. administered non-self-governing territory whose decolonization process has been stalled for over 123 years.

Our people have suffered numerous harms since the United States took colonial control over the island in 1898, including racist and discriminatory treatment by Naval authorities, negative health outcomes resulting from the storage and usage of nuclear weapons, radioactive vessels, and toxic chemical agents, including Agent Orange and massive land seizures to make way for U.S. military bases and installations among other things. Guam has 19 superfund sites, sites containing substances so hazardous they require a long-term clean-up response, and at least another 70 toxic sites. Far from being remedied, these harms are aggravated today by a massive military buildup and expansion of the U.S. military footprint in Guam.

With insufficient consultation from our community and complete disregard for the Chamorro people's rights to free, prior, and informed consent, plans have proceeded along with the construction of live fire training regions and other installations on sites of great cultural significance to the Chamorro people. The plans include the clearing of over 1,219 acres of limestone forests for constructing the ranges and base, close to seven million ammunitions fired each year over our sole source aquifer, the withdrawal of 1.2 million gallons of water every day from the aquifer, the destruction of endangered and threatened species, including our traditional medicines, and the desecration of several of our ancestral burials.

Furthermore, the U.S. Air Force has applied for an open burn and open detonation permit. This is an extremely antiquated and dangerous practice that is banned in the United States, except for the exemption granted to the U.S. military. Striatum, uranium, magnesium, barium, and lead are just a few of the harmful constituents identified in the permit application. PFOS and PFAS are also contaminants that will be released. The open burn, open detonation pit on Guam is sited along the northern coastline and just above our sole source aquifer that supplies over 85 percent of our island's drinking water. Additionally, it's adjacent to the island's two most populated villages. Multiple production wells accessing the island's aquifer have had to be shut down due to chemical contamination from U.S. government land holdings over and adjacent to the aquifer. Just two days ago, our organization represented by Earth Justice filed a lawsuit in federal court challenging the Air Force's failure to comply with the National Environmental Policy Act for its plan to burn and detonate about 35,000 pounds of bombs and other hazardous waste munitions each year in the open air on Tarague Beach, less than 200 feet from the Pacific Ocean.

We argue that the Air Force violated NEPA without first considering any impacts or looking at better alternatives, and this is taking place on land that was taken from families to hope to see the return of lands including my own family. We will never be able to return to the land if the Air

Force is continually allowed to contaminate the land and water with their toxic waste. We're forced to challenge this in court because the U.S. EPA exempts the Department of Defense. We respectfully ask that you consider all of these issues and request that the Biden administration ends the exemption of this archaic practice and pay close attention to the destruction and desecration of our lands, waters, and all that we hold secret at the very hands of this government.

1.5.26 Catherine Skopic - Sierra Club New York City Group (New York City, New York)

Catherine Skopic began with renewable energy only, protect the Hudson River, reject the Champlain Hudson Power Express. Code red, that's what we're in as we all know as proclaimed by the Intergovernmental Panel on Climate Change, the IPCC. We're in a climate emergency. We know, and that calls for action. She asked for the following actions: One, rescind the presidential permit allowing energy cables to cross our border with Canada. Two, recognize the racial, environmental, and economic injustice of CHPE. Three, reject the CHPE project.

New York State has ambitious climate legislation in the 2019 Climate Leadership and Climate Protection Act, CLCPA. We have active development of on and offshore wind, solar, heat pumps, geothermal, and other renewable projects, all good. However, there was one project, not New York State initiated, that is not good, and it produces methane and CO2, causes racial, environmental, and economic injustice -- the Champlain Hudson Power Express, CHPE. Initiated by the privately owned company Block Stone with over a decade of trying to get it realized. Hydro-Quebec builds mega dams resulting in monstrously sized reservoirs that give off huge amounts of methane and CO2. Their nonrenewable energy would travel by marine and land cable to New York City.

These mega dams are built without permission of indigenous lands and peoples destroying their central means of survival -- hunting, fishing, trapping -- and they cause methylmercury, a poison formed by the submerging of vegetation. One native called it "slow genocide". As fine as Canada is in other ways, we can't have more methane and CO2 or racial injustice to Canadian indigenous. Cables would be laid under Lake Champlain and the Hudson River, the dredging process ruining marine life and the drinking water of the Hudson Southern. For the people of the seven communities along the Hudson who get their drinking water from the Hudson, the PCBs and toxins stirred up would render their water undrinkable.

These cables also give off electromagnetic frequencies that disrupt fish productivity and can harm people as well. New York State would be exporting its jobs and energy dollars to Canada leaving New York State ratepayers responsible for the estimated 3.6 billion cost causing economic hardship for many. Rescind, recognize, reject the Champlain Hudson Power Express.

1.5.27 Courtney Rhoades - Appalachian Citizens' Law Center (Whitesburg, Kentucky)

Courtney Rhoades stated that she is the black lung organizer at Appalachian Citizens' Law Center, a nonprofit law firm based in Whitesburg, Kentucky. Where the Black Lung Disability Trust Fund was established in the 1970s, receiving benefits remains a drawn-out process for insufficient monthly payments and the funding mechanism for this trust fund remains unsustainable as the sole funding source is an excise tax that was once again slashed in half at the start of the new year despite the hard work from groups in the region.

The prevalence of black lung, an entirely preventable disease caused by coal dust, has doubled nationwide since 2000. Minors in central Appalachia have been most affected with 1 in 5 veteran coal miners having black lung disease, and 1 in 20 having the most severe and disabling form of this disease, progressive massive fibrosis. Though our region has been most impacted by this epidemic, other areas with coal could see the same numbers in the next few decades as coal resources decline and the facilitated standard for coal miners are at the highest exposure for the U.S. has not changed. Though black lung is not a traditional environmental issue, it is another example of the glaring negative impact the coal industry has had in our region and the nation for over a century and the ramification of what happens when issues are continually ignored because of the power and industry has.

As you compared recommendations on energy policy, she urged them to remember that those in the communities that have been previously and continue to be negatively impacted by the coal industry. We need this administration to prioritize the reinstatement and extension of the Black Lung Excise Tax that funds the trust fund. Confirmation of a director who will create a silica standard that will decrease exposure, thus, preventing future black lung cases and curing any future miners.

1.5.28 Amanda Woodrum - Re-Imagine Appalachia (Ohio)

Amanda Woodrum stated that she is a senior researcher for Policy Matters Ohio and the director of the campaign to Re-Imagine Appalachia. We are a four-state campaign at the heart of coal country. We are working to find common ground across key stakeholder groups in their region from organized labor to racial justice leaders, faith, environment, local government officials, as well as others to create a vision for what we want Appalachia to look like and a blueprint for how we get from where we are to where we need to go. We are incredibly heartened by the Biden administration's commitment and its Justice40 policy to ensure that communities that have long been left behind or in the process of being left behind are invested in.

As you all probably know, Appalachia is basically an area of concentrated poverty. We have been exploited for more than a century by absentee corporations and the extractive industries that have left our communities impoverished, our workers and neighbors sick, and our lands damaged. We are not going to lift ourselves up by our collected bootstraps. We believe we deserve, and we certainly need federal investments, particularly infrastructure investments. They need to be well designed to essentially build the foundation for a prosperous economy over the long haul, one that is built on local wells. That means creating good union jobs, building pathways into those union jobs for black workers and other people of color and other low-income communities, and giving preference to coal industry workers for new opportunities.

We can learn a lot from the community benefit agreement movement, and we cannot just try to get the money out the door as quickly as possible. These infrastructure investments need to come with federal policy guidance with community and labor standards, transparency in the process. It is worth taking the time to do this right. We know the way we've done development in past doesn't work. We need to do development differently in the future. She encouraged them to work across all departments to make sure that happens and that we're not just funding the same old as we always have.

1.5.29 Nyoka Baker Chapman (Barboursville, West Virginia)

Nyoka Baker Chapman stated that it is well known that West Virginia is one of the top emitters of greenhouse gasses in the nation. We're also at the bottom of the rank used for being a healthy place to live and are at the top rankings in the U.S. for cancer and other diseases related to living and working in the fossil fuel industries. We have thousands of uncapped gas wells that are leaking methane and the fracking of natural gas continues. Our Ohio Valley area has concerns that the Appalachian storage hub will be built along with two plastic-producing ethane cracker plants that will have a devastating impact on the health of the communities who live near them.

Environmental justice for the people of West Virginia is clearly long overdue, and hopefully, some of the Justice40 initiatives will bring change to our communities. West Virginia will be a great state to initiate some pilot programs. By all appearances, West Virginia is a state without a plan to meet a 50 percent reduction in greenhouse gasses by 2030, and zero emissions by 2050. West Virginia is a part of the Paris Climate Agreement by default, and our state must acknowledge that it has a responsibility and an obligation to lead and play in its part to meet our PCC goals. We know there must be just transitions for communities, for the workers who must retrain for greener jobs, and for the industries themselves that all stakeholders must see positive outcomes for the coming decade.

Perhaps, states could be mandated to form planning commissions and provide reporting as to how they will be meeting critical climate change deadlines. We know this will not be easy for West Virginia, but we are a state and not a colony, and federal policies regarding climate change initiatives must be met. Energy profiteers, who utilize our fossil fuels for the purpose of mining blockchain data for proof of work authentication for cryptocurrencies, are infiltrating our state. The resulting damage from pumping even more tons of greenhouse gases into our air, excessive noise, losses to property values would clearly place greed of royalty owners over the welfare of our citizens.

New business applications could just ask a simple yes or no as to whether new businesses would be creating blockchain cryptocurrency. At least this would allow for their activities to be monitored and allow follow-ups for state divisions of banking and divisions of finance to review reporting and registration for the U.S. security and exchange commission. Our state is truly beautiful, and we have so much to offer in terms of tourism and recreation. We want to have a green horizon and see a positive future for the next generations. Federal participation and helping to advance this stream are more than welcome.

1.5.30 Alicyn Gitlin - Sierra Club - Grand Canyon Chapter (Flagstaff, Arizona)

Alicyn Gitlin stated that she represents Sierra Club's Grand Canyon or Arizona Chapter. I'm speaking to you from Flagstaff, Arizona in the Grand Canyon region, the ancestral and current homeland of at least 14 tribal nations. This region is the site of ongoing environmental injustices related to several stages of the nuclear fuel chain from the legacy of abandoned mines in the Navajo Nation, to the Pinyon Plain Mine formerly known as Canyon Mine which is extracting ten million gallons per year of uranium and arsenic-contaminated water from the Havasupai Tribe's watershed despite never producing an ounce of uranium ore, to the White Mason Mill which was just cited for carelessly polluting the air of the Ute Mountain Ute's of radon while their outdated containment cells threaten the Ute's water supply.

These tribal nations bear the brunt of ongoing intergenerational health risks while foreign corporations play a careless money game. We were therefore very happy to see in the Advisory Council's final recommendations plainly stated examples of the types of projects that will not benefit a community. Number four, the procurement of nuclear power. This and all future administrations must operate on the principle that tribes have a right to free prior and informed consent. Consultation is important, but we all know that consultation is not consent. Tribal nations have dealt with enough, and they deserve better.

We, therefore, support the following from the Council's final recommendations. Tribes must be involved as decision-makers, planners, and leaders. Tribal co-management must be an available option. Cultural impacts of infrastructure are critical to consider, and 100 percent of investments must do no harm to environmental justice communities. Green infrastructure must not be built by sacrificing the health of indigenous peoples, destroying their most important religious sites, their water supplies, or their clean air. We are seeing too many examples of mining proposals being marketed as green while they lie within traditional cultural properties and vitally important watersheds threatening to contaminate and deplete the springs and drinking water supplies that affected communities depend upon.

Nuclear power should not be part of any climate solution, and nothing should be part of a climate solution if it harms environmental justice communities. We can find a pathway to a healthy future and tribal nations and other affected communities should be at the forefront of recommending, designing, and benefiting from climate investments.

1.5.31 Shannon Bell - Virginia Tech (Blacksburg, Virginia)

Shannon Bell stated that she's an environmental sociology professor at Virginia Tech in Blacksburg, Virginia. Environmental health research has demonstrated that living near industrial activity is associated with increased stress, depressive symptoms, and feelings of powerlessness. These studies demonstrate that it is not just the magnitude or type of industrial activity that matters. Institutional decision-making processes also play a significant role in the mental health effects of industrial activity on local populations.

U.S. EPA defines environmental justice as the fair treatment and meaningful involvement of all people regardless of race, color, national origin, or income with respect to the development, implementation, and enforcement of environmental laws, regulations, and policies. Her comments focus on the meaningful involvement clause of this definition, which many residents who have been in the path of a new natural gas pipeline feel has not been met by federal agencies. Over the past two and a half years, she and her research team have been studying the social and psychological effects of what we call the disruption phase of new natural gas pipelines, namely the announcement, permitting, and construction of these new fossil fuel infrastructure projects.

Through our analysis of the more than 900 responses we received to two different surveys -- one mail survey and one online survey - we have found that agency public participation processes have not only been disempowering, but they have caused psychological distress and a loss of faith in government institutions among many residents who have participated in these opportunities. Respondents indicated that after engaging in these processes, they felt that their concerns were ignored or completely dismissed by these government agencies, which they came to see as just working for industry rather than for the American people. And many indicated that their inability to influence agency decision-making despite the countless hours they had spent trying, not only led to increased feelings of stress, but it also had consequences for their physical health.

Respondents discuss developing a variety of stress-induced health problems during the pipeline certification and construction processes including high blood pressure, insomnia, immune system disorders, diabetes, and heart problems to name a few. As researchers at Cornell Law School's e-rule making initiative assert, a democratic government should not actively facilitate public participation that it does not value. Encouraging people to engage in participatory acts of low value to government decision-makers is pedaling democratic snake oil. Our findings demonstrate that when people take part in public participation processes that have little or no effect on agency decision-making, it not only disempowers but harms participants and destroys their faith in the legitimacy of government institutions.

As the White House Environmental Justice Council undertakes its important work, I urge the members of the Council to find ways to ensure that agency public participation processes are truly meaningful and that affected residents have the ability to actually influence the outcomes of new energy projects.

1.5.32 Maury Johnson - Preserve Monroe (Monroe, West Virginia)

Maury Johnson stated that he's representing several groups here. The first is located as Preserve Monroe. Monroe County, West Virginia is in Southeastern West Virginia, not too far from where the last speaker spoke from Virginia Tech. We are a small rural county of about 13,000 people from 249 square miles. He's also a member of the Power Coalition, which protects our water, heritage, and rights that formed in the opposition to the Mountain Valley pipeline that's coming through from West Virginia through Virginia and in North Carolina that come through our area. He's also associated with a group of people called Walk for Our Grandchildren that this summer we walked from Scranton, Pennsylvania, met with communities all the way down through Gibbstown, Pennsylvania; Marcus Hook; Chester; into Philadelphia; into Wilmington. I'm also a member of a number of state, local, regional, environmental justice, and social injustice organizations.

Over the past several years, he's met with not just people of West Virginia and Virginia, but Ohio, Nebraska to across the Midwest. People out in the Jordan Cove area, Oregon. He's met with people from the bay of New Orleans, Mississippi Gulf region. The story is the same. Those with power and money and the government who's supposed to be protecting us, we're just in the way. This thing of environmental justice has been lip service. Hopefully, there will be some change. He sees his community being devastated by a pipeline that's not needed in his opinion and a lot of people's opinion. For instance, he's in a very social, economic depressed area. He hasn't been able to turn on his well or use the water in his house for nine months.

When you say something about it, they said to prove it. Well, it cost him $40,000 to try to prove it. He doesn't have that kind of money. He has no government agency willing to come up and help him. The FERC tried to create public participation. He's engaged with that. He's very disappointed in what's happening. He truly hopes that Michael Regan will address the environmental destruction across Pennsylvania, the Midwest, and the Native Americans, the people in Guam. The lady that spoke from Barboursville who's just up the road from me and Shannon Bell from over in Virginia Tech said, we have been screaming at the top of our lungs for the last several years that something must be done.

In closing, a friend of mine in Richmond, Richard Walker, says, "They're hurting my people, and we're not going to take it anymore." The people in this country aren't going to take it anymore. We need your help.

1.5.33 Sarah Merrill - SAVERGV (Rio Grande Valley, Texas)

Sarah Merrill stated that she was calling from the lower Rio Grande Valley on the Mexican border. She had signed up to talk about no more border wall would be a very good solution and save a lot of money. We here have been fighting with the SAVERGV organization. We've been fighting several LNG plans that will greatly increase VOCs and methane emissions and make global warming even worse. We've had many more hundred-degree days than when I first moved here. She had taught environmental ethics and also engineering ethics and have a book on construction ethics. So, the building changes that people have discussed today are very interesting to her, improving ventilation, though, as well as improving with sustainable materials and construction of more sustainable buildings, possibly even regenerative buildings.

Her organization is very much aligned with groups like the Carrizo/Comecrudo Tribe, and we're also fighting the noise pollution and debris of SpaceX. These are all located with planned LNG sites along the Brownsville Ship Channel that is by Route 48 and Route 4 Boca Chica Beach, which Elon Musk is taking over, and her group needs some federal help because our county is not protecting us against SpaceX's debris. I know SpaceX is very popular, and I'm sure the Biden administration doesn't want to speak against SpaceX, but really, they've trashed the region and they did want to buy Boca Chica.

So, her group is urging on the scoping process for more barriers and more blocks. They are happy that the Biden administration is aware of these problems and hopefully does not intend to spend lots of wasteful money destroying habitat instead of restoring the riverine environment that will help with flood prevention and erosion control. So, her group are environmentalists in South Texas. We saved the Santa Ana National Wildlife Refuge and La Lomita Mission, and we're witnessing on the border some positive things. So, we'd like to sort of say that we're glad cumulative is back in the NEPA, which the Trump administration took out. Environmental damage is nothing, and environmental justice is nothing but cumulative. But they also hope that they'll be able to build on the positive cumulative effect of the positive diplomacy that our vice president's been doing and that is happening here with art exhibits and uses of the wall, what exists of it, as a beautiful art project and by cultural art project as in the book *Up Against the Wall: Reimagining the U.S.-Mexico Border* by Casey and Watkins.

So, let's capitalize also on the positive cumulative effects of the diplomacy they've been building with our Mexican brothers, sisters, abuelas, and continue to see that immigration is a real source of hardworking labor for farming, agriculture, healthcare, and construction. So, we urge you not to build any more border walls, and that's a way to save money that can be put into these other programs, helping all the EJ communities. They are a relatively poor population. She's taught there for 20 years, and they have a very needy population, but they're hard workers. They are a peaceful people down here on the border.

1.5.34 Tina Smusz - Preserve Montgomery County, VA (Virginia)

Tina Smusz stated that a full understanding of environmental justice issues is presumably lacking in many state and federal agencies whose leaders approve new infrastructure buildout that often disproportionately impacts rural and lower-income communities. There is a critical need for increasing environmental agency staff's understanding and perception of what constitutes environmental justice. She bases this on a recent statement by Virginia's Department of Environmental Quality director of water permitting who assured state water control board members that environmental justice issues did not pertain to the construction of the huge Mountain Valley pipeline, a high-pressure transmission methane pipeline project, which has spent the past five years bulldozing a hundred miles through rural Virginian's farmland, in addition, many miles in West Virginia as you just heard from Maury Johnson.

It's plowed through water resources, pastures, ancient tree grows, and local heritage sites. Ms. Davenport stated erroneously that there were no, quote, fenceline communities, end quote, impacted by the project. Even more alarmingly, the implication was that there is no such thing as a fence line community when it comes to pipeline projects. This dismissive and misguided statement by a DEQ leader likely impacted the board member's decisions as they narrowly approved the water permit needed for completing this destructive and eventually methane-leaking behemoth.

Linear construction projects like 42-inch diameter, high-pressure gas transmission pipelines create continuous fenceline communities subjected to permit intrusions through their property and peace of mind, loss of use of portions of their land, disruption of household and stock water supplies, and perpetual risk of methane leaks and explosion. The typical routing of large transmission lines disproportionately impacts the elderly, low-income, and medically underserved populations living in more remote areas in many states.

By their nature, fossil fuel transmission lines now epitomize environmental injustice as our planet faces a future forever changed by global warming induced by dependence on fossil fuels.

1.5.35 Sheeah Bearfoot - Anthropocene Alliance (California)

Sheeah Bearfoot stated that she is the program manager at Anthropocene Alliance. Several of our members have spoken here today about a myriad of different environmental justice issues.

We have members who are experiencing lead poisoning, flooding, governmental apathy, a whole host of issues, and they are banding together hoping to be heard. Like Senior Advisor Arsenio Mataka, she grew up in the Central Valley of California, and they still don't drink the tap water there. Because the issues that our organization represents are so numerous, we have over 110 different member organizations, it would be impossible to spend time talking about the different scientific issues that all of our community members are facing.

She has a background in environmental health science, but she wanted to speak more from a place of what to do with anger and frustration that we're all feeling, and she encourages everyone here and any form of decision-making power to remember why they got into this work in the first place and continue doing what they can. We've seen so many people today express frustration at the slowness and seemingly just inability for a change to occur. People have been talking for generations and generations about the impact that they're facing, and it just winds up compounding an intergenerational trauma.

She wants something to be done. Everyone here wants something to be done. We're all a bit confused as to why everything still seems to be at a standstill. She appreciated everyone here taking their time to tell their individual stories. A2 is going to continue working as hard as we can to try and help you individually, one on one to try and work through your various issues, and also to try and make our coalition stronger and have us all band together and speak out against the myriad of environmental injustices with one united and strong voice.

1.5.36 Brandi Crawford-Johnson - EJ Activist (Kalamazoo, Michigan)

Brandi Crawford-Johnson stated that she is an environmental justice activist from Michigan. She got very sick with severe asthma from living in a fenceline community in Kalamazoo, Michigan. She has been fighting, researching, and engaging state and federal agencies to help for years. She also met with MDHHS and ATSDR and they agreed to do a health report on our fenceline community as it relates to toxic exposures from the expanding papermill called Graphic Packaging.

We have a 14-year death gap in our neighborhood. She had to file a civil rights complaint against Eagle with ECRCO for allowing the same polluter to expand despite GPI being out of compliance for violating the Clean Air Act and EGLE receiving hundreds of air complaints from her predominantly African American neighborhood for years. MDHHS will produce their health report along with recommendations for our neighborhood in a few weeks. She still wanted to stress the fact that most people do not want to live next door to toxic polluters. They are sick, stressed, and living next to these polluters because they cannot afford to move. We must use Justice40 funds to relocate these families. She just doesn't see any other option.

The EPA also has funding to relocate residents from superfund sites. These same funds could also be used for relocation because most of the fenceline communities like ours and Kalamazoo are contaminated just as badly as the superfund site even more so because the pollution and contamination never stop. She encourages WHEJAC members, the EPA, and the White House administration to include options for relocation for residents in Kalamazoo, Michigan and all fenceline communities in the United States with EJ scores over 70 percent.

1.5.37 Adriane Busby - Friends of the Earth

Adriane Busby stated that she is the senior food and climate policy analyst with Friends of the Earth. Today, I'd like to talk to you about the negative impacts of concentrated animal feeding operations; otherwise known as CAFOs or factory farms. An issue that has been almost entirely disregarded as an EJ priority for this administration despite the horrible health impacts it has on communities of color and low-wealth communities. Factory farms have been given a free pass to pollute and profit at the expense of EJ communities for decades. There is ample science that demonstrates the major role industrial animal agriculture plays in contributing to climate change. Further, the waste generated by CAFOs can contain pathogens and antibiotic-resistant bacteria in a spray can and often does reach nearby homes and drinking water sources.

The older plumes, which often pervade nearby communities, contain respiratory and eye irritants such as hydrogen sulfide and ammonia. A growing body of research suggests these emissions may contribute not only to respiratory illness in nearby residents, but also decreased quality of life, mental stress, and elevated blood pressure. All of these negative impacts disproportionately affect low-income communities and communities of color because of where factory farms operate. Researchers show a disproportionately high concentration of factory farms in communities of color despite the declining number of black farmers in the southeastern United States. This is targeted and intentional.

One study published last year found approximately 12,700 deaths per year from air pollution in the U.S. that are attributable to industrial livestock production. That is more deaths than occur from pollution from coal plants. Yet, industrial livestock remains largely unregulated, and this administration's policies threaten to expand the industry. President Biden's methane strategy for agriculture, which is the number one source of U.S. methane emissions, relies on investing and production of factory farm methane gas by subsidizing the cost of methane digesters for factory farms. Paying corporations to continue polluting is not the solution to curbing air and water pollution.

In fact, it will have the opposite effect resulting in an expansion of these operations and clustering in overburdened areas to maximize profit. Creating even more pollution for communities of color, especially since digesters do nothing to mitigate coal pollutants like nitrous oxide, ammonia, and heavy metals. We have seen this happen already in places with a lot of methane digesters, such as in California Central Valley. The administration is not just failing to act on factory farm regulations but is actively going in the wrong direction by subsidizing them.

Friends of the Earth ask that WHEJAC work with the Biden administration to uphold its commitment to EJ principles by exercising its authority to meaningfully regulate factory farms to stop further subsidization of factory farms and by supporting a just transition to regeneratively just agriculture instead.

1.6 Closing Remarks - Announcements & Adjourn

Chair Moore reminded everyone that the WHEJAC is made up of various representatives representing broad EJ issues and institutions and organizations. Although they're not representing all organizations, many of them, if not all of them, have been working on environmental justice issues for many, many years. One of their primary themes has been consistently, leave no community and worker behind. They understand the feeling that people want to see the government moving quite faster than what it's been moving. He reminded everyone that there has never been a White House council on environmental justice. This Advisory Council is committed to moving the process forward. The window is open to take advantage of getting as much done as they can get done for the people. He turned the meeting over to Carletta Tilousi. **Carletta Tilousi** stated that she is committed to continuing to voice all their voices. They have been put in this position with the responsibility to continue to speak up against injustice in our communities, and that is what they're going to continue to do. **DFO Martin** closed the meeting for the day.

2.0 Welcome, Introductions & Recap

On Thursday, January 27, 2022, **DFO Martin** welcomed everyone in attendance and explained a few housekeeping items. She noted that Spanish interpretation and closed captioning were available during this meeting. **Chair Moore** reflected on the presentations given, charges given from CEQ, and the public comments. He also reflected on the past. He mentioned that their past sisters and brothers left them with three things in mind: never forget where you came from, always remember whose shoulders you stand on, and always give back to others what’s been given to you. He reminded everyone that the WHEJAC came out of the grassroots movement for environmental and economic justice.

Peggy Shepard informed everyone that the Advisory Council will discuss and offer recommendations for the scorecard that will evaluate the Biden administration’s efforts to advance Justice40 and to advance its priorities on environmental and climate justice. They will discuss core principles that should form the basis of a scorecard. In addition, they’re hosting a panel on Justice40 with several guests from government agencies to help define terms and answer key questions by the Justice40 workgroup. And, lastly, they will hold a business meeting to reflect on yesterday’s public proceedings and issues raised by past public commenters, as well as to discuss new actions and next steps.

She acknowledged that there’s a lot of frustration from the public to see action and progress, but she assured them that the WHEJAC members are meeting in six different workgroups and formations every two weeks. They are working hard and meeting all deadlines given to us by the White House Council on Environmental Quality. They anxiously await and push for status updates on how our recommendations are being considered and implemented. They are here to achieve the best outcomes for the frontline and environmental justice communities in which we live, work, play, and go to school.

Vice-Chair Flowers stated that what they are doing and the work that has been done has been a long time coming to get to this point. This is the first time in her life that they’ve had this type of audience where they have the attention of the executive branch of government as it relates to dealing with environmental justice issues. The WHEJAC represents the various communities that come from and make sure that they are aware of the public's struggles. She was very moved by the testimonies of people from across the country who are dealing with all kinds of environmental injustices that have been going on for years, but hopefully, they can bring the type of attention and action that is necessary to resolve some of the issues that were brought to our attention. Keep in mind that this is a marathon and not a sprint.

Vice-Chair Tilousi stated that she is new to this level of responsibility and task. She's still learning, but as long as she is there, she will continue to speak for the communities that are facing injustices across the country. Water is very important to us, human life is very important to us, and those are the things that they've been discussing these last couple of days. If this body has been formed to make such strong recommendations to the White House, they need to continue to remain on task and set goals and objectives and timelines and pen on paper. She stated that she will continue moving forward as she listens to other testimonies from communities across the country that are continuing to face injustices and water contamination and even human rights violations.

DFO Martin asked the Council members to briefly introduce themselves and afterward informed the Council that the quorum was met.

2.1 WHEJAC Scorecard Workgroup Update & Discussion

Dr. Whyte started by talking about the nature of the recommendations that we're seeking consent from the WHEJAC on. They decided to break up their work into two phases given the complexity of the task of making recommendations for environmental justice scorecard within the whole of government approach which includes many different agencies. In Phase 1, they offer ground-level recommendations about what a scorecard is and what it should do. In Phase 2, they will be focusing on very specific scoring metrics as powerful examples to convey to the federal government about environmental justice scoring. This will take place from now until the next public WHEJAC meeting. This meeting session focused on just Phase 1 and these base recommendations.

He reviewed some of the basic setups for the tasks that they were given and the original strategy that was developed in September. So their mandate from CEQ was to address the following question: what types of indicators or data would be useful in an agency scorecard? There was a reference to the fact that their input can be in the form of general ideas or specific data. This will be a continuing process, and the WHEJAC will have ongoing opportunities for providing further feedback. He highlighted the living nature of this particular project, especially given the complexity of scoring in the context of so many different agencies with very different administrations, programs, and other dimensions. When they took on this task at the end of September, they developed a strategy that they would draw on previous WHEJAC recommendations. They drew on consolations with experts, especially federal agency recommendations, of which they had several key meetings. They drew on the Justice40 interim guidance, and they drew on the Climate and Economic Justice screening tool interim guidance. They didn't have access to any updates from that particular project. So, they were going to identify areas of need for scoring federal agencies on their contributions to environmental justice. To learn what agencies are doing now to track progress on the Justice40 initiative, they're going to form recommendations that ensure the environmental justice scorecard effectively attracts agency accountability.

When they went about this strategy, they learned quite a bit about what it would take to have a scorecard that would have this important grading and assessment function. So, they realized that there needed to be alignment in terms of what the purpose and point of a scorecard would be, and so, the Phase 1 recommendations reflect some of the key aspects of what they think the point of a scorecard is. In another public meeting, they'll present Phase 2, which has to do with the specifics. So, in the next parts that follow, they'll be talking about some of these key aspects regarding what the point of a scorecard is.

The first category is the goals of the environmental justice scorecard. So every agency should have an environmental justice scorecard that tracks the impacts that all government investments have on disadvantaged communities, especially where disadvantage is produced by racial discrimination and economic barriers. By this, they understand that impacts are changes in outcomes in the areas relevant to environmental justice, including outcomes in health, economic opportunity, food security, environmental quality, climate change, maintenance of culture, access to quality housing, energy, transportation, safe, affordable drinking water and sanitation, healthcare, and other physical and social infrastructure. The scorecard must aid federal agencies on access, distribution, and benefits of the impacts according to race, income, as well as other demographic and geographic indicators. In brief, the environmental justice scorecard will follow where and how federal money is spent, and what happens as a result of federal spending to communities disadvantaged by racial and economic inequities. Federal spending and investments will be tracked to evaluate community-level impacts, benefits, improvements, and lessons learned in terms of advancing environmental justice for black, brown, indigenous, and low-income communities.

In terms of goals, the EJ scorecard should ensure just treatment and full protection for disadvantaged communities and advance the application of Title VI and NEPA in agencies. Agency and administrative professional culture should encourage and incentivize staff to reflect and share lessons learned, including mistakes, and to develop processes for incorporating iterative and bidirectional feedback to craft sustainable and socially equitable solutions that avoid repeating and that redress the impact of past mistakes. It should be acceptable for agencies to be forthcoming about where they are, falling short of procedural and outcome goals, and solicit public input on how to address these challenges. He emphasized the importance of this point. That agency culture/administrative culture matters greatly in terms of whether a scorecard will serve the purpose that it can in protecting so many communities that are affected by environmental injustice. Lastly, in places or situations where harm is occurring that is directly and indirectly caused by agency-endorsed programs and activities, the existence of ongoing harm should be included as part of the scoring measures.

Maria Lopez-Nunez said that next, she'll go over the scale and scope of the EJ Scorecard. They don't want to assume that every agency will just be measuring environmental justice programs. They do believe that for the EJ Scorecard to be successful it should include all programs, not just limited to those that are Justice40. That's going to paint a real picture of where the agency is, and not just cherry-pick the programs that might make them score the best. They also think that it's really important that the scorecard must address investment and benefits in terms of racial and income-based equity. They think that that's incredibly important for each agency to keep track of who is being impacted. They want real numbers based on race and income, of outcomes not just process. The EJ Scorecard should assess environmental justice for all federal spending, not just Justice40. The scorecard is not limited to federal investments that are explicitly intended for environmental justice. The EJ Scorecard will address an agencies' specific and interagency investments. Agencies will develop their own version of the EJ Scorecard for application to their specific programs and mandates. So they do understand that each agency might have to be specific, but there should be a generalized approach.

Agency-specific EJ Scorecards should have a data strategy for developing environmental justice indicators that map onto short and long-term environmental justice goals. Scorecards should draw from existing data sources, develop new data, and identify data needs. Data on race and income should be used to identify racial and income inequality. EJ Scorecards should reflect the most up-to-date methods of timely and transparent and accessible data acquisition, storage, analysis, and reporting. Their thinking on that was as much as possible people should be able to look up and be able to see the data, where the money is flowing, where the programs are headed. They would like everything to be accessible to the public so that everyone can participate. Nation-to-nation consultation with tribal nations would be developed for the specification of the purposes and uses of the scorecard for tribal nations. The scorecard should address compliance by states and localities that the funds, investments, and benefits benefit the frontline communities identified by the Climate and Economic Justice Screening Tool. For indigenous peoples and groups with special legal status, adequate engagement will take place to determine specific purposes and uses of the scorecard and data inputs.

The scorecard should track how well-prepared agencies are to understand where their investments go before the investment. That’s key. Before the investment contracts, grants, cooperative agreements, and other forms of federal spending are distributed, scoring for environmental justice, including Justice40, should be both anticipatory and forecast investment distributions, as well as evaluate the impacts of federal spending. They want the scorecard to evaluate not just after the fact and not to do financial autopsies, but also, hopefully, if agencies are trying to keep track before the money goes out. The thinking on this is that there are states that, once the money goes out, might not do the right thing. So, for the scorecard to incentivize good behavior, it should take into consideration how are agencies prepared to track that money before they give up the money that might not get to the places that our environmental justice movement wants it to get to. The scorecard should address community engagement and consultation efforts. Those things should, of course, count towards the outcome of the scorecard.

Then for the purpose of the indicators, agencies should ensure that they recruit staff with relevant expertise in environmental justice assessments who will design agency-specific scorecards and the diverse elements of scorecards, including indicators. Agencies should design scorecards based on explicit EJ goals and outcomes that can be clearly mapped to indicators and data used as inputs. Agencies should include indicators that cross overarching EJ goals across agencies as well as indicators that are specific to each agency program.

Dr. Wright hoped that people can already see how much time, thought, and effort they’ve put, into trying to come up with ways to make the scorecard actually represent what Justice40 is supposed to be doing. There are major areas of need for indicators that they’ve identified. The agencies should have indicators measuring the reduction, prevention, and elimination of pollution, legacy pollution in particular, and cumulative environmental hazards in communities facing racial and income inequalities. They should also have indicators using data that measure whether meaningful participation and community engagement in decision-making are occurring in federal actions. This includes participation that drives decision-making as well as technical assistance, training, cultural and linguistic access, as well as access based on ability and capacity building. Linguistic access must be inclusive of all languages, including African American and Native American languages. Indicators should be tied to agencies' strategic planning, timetables, reports, the establishment and operation of an environmental justice advisory committee (for each agency under FACA), and plans to coordinate with states, counties, and other levels of government.

Regarding Justice40, ensure agencies track how their investments are impacting frontline and environmental justice communities consistent with Justice40, including measuring recipients of and benefits of investments both direct and indirect, and in terms of race and income. Next is to evaluate benefits that include indicators related to outcomes connected to racial equity, economic opportunity, health, environmental quality (for example, air, drinking water, food, land), sanitation, infrastructure, housing, and cultural protection; measuring investments success in cultivating local ownership, contractors, workforce development, poverty alleviation; the establishment of local financial institutions, infrastructure, sustainable technologies, and tools; community-driven recovery, adaptation, and rebuilding, and community ownership of infrastructure. The next is to track whether federal investments are engaging in processes and implementation that incorporates a community-driven, community-controlled approach so that communities most directly impacted benefit as intended.

Vice-Chair Tilousi opened the meeting to comments, questions, and any suggestions related to the draft.

Ms. Santiago inquired about a reference about how the scorecard would work with respect to communities with special legal status. It mentioned tribes and communities with special legal status. Is that referring to the territories like Puerto Rico and Guam, Virgin Islands, et cetera? And how do you envision that if that’s the case?

Dr. Whyte responded with that was exactly how they intended it. They mainly wanted to ensure that communities where their legal status would relate in some way to how agencies relate to them, that that was duly considered. And so, that’s as far as we may have gone with that particular point, but it was just to give a voice that agencies ought to be accountable for ensuring that they’re taking very seriously how their work, their duties, their impacts may differ in cases where you have different forms of legal status.

Ms. Santiago wondered whether they thought about how the special legal status of the territories might make the scorecard process different for the agencies? If somehow there’s a relation to say, representation in Congress or voting for president? Obviously, they don’t have any of those things.

Dr. Whyte stated that they wanted to ensure that what they had was open to other examples and cases that some may be more or less familiar with, but where differences could possibly be important. For example, with regard to federally recognized tribes, you know, tribes are running their own governments, and so, there needs to be something worked out about what does that mean? Because tribal governments that run programs would also be subject to a scoring mechanism potentially. So that’s not really something they can weigh in on here, but something that would need to come out of a specialized consultation process.

In terms of other entities, like, for example, Native Hawaiian-serving organizations that operate under a certain type of business and legal status provide services to Native Hawaiian communities, federal agencies need to relate to those organizations in a particular way. Or those organizations can actually reach out to federal agencies directly. They wanted the scorecard to flag and be something that wouldn't leave any special considerations out. They're certainly not intending to claim that the special legal status might be an excuse for an agency not to treat a community equally. Rather, we're looking at cases where communities, groups, or peoples might actually be overlooked in terms of the scoring mechanism.

Vice-Chair Flowers stated that her concern is making sure that people, organizations, or entities that receive funding are definitely representative of the local community and not people that came in, applied, got the money, and moved on. Maybe that can be one of the indicators, how many people were a part of that process that came from that local community. **Dr. Whyte** appreciated the point and will make sure that some of the specific examples actually demonstrate what that looks like.

Maria Belen Power asked how were they envisioning measuring harm, and how can they make sure that the harm that is being done has an impact? Can we take points off for harm that's done on communities that are already overburdened or vulnerable? How can this scorecard specifically incorporate the harm so that we make sure that that's not happening? The other piece is around accountability; are there consequences or penalties for agencies that don't meet the standards? How do we make sure that the communities most impacted aren't penalized by losing funding?

Dr. Wright responded that this is a place where they all got stuck initially, and that's why they're moving into the second phase of trying to develop a metric that would include that. People were asking, how do we determine the harm that's there? They have a number of members on the committee who are good at developing a matrix so that they could get that done. As a sociologist, we use Likert scales all the time where it goes from very good to awful, and they get a score somewhere in the middle or they get an awful score. Averaging that up to give them an overall score is hard work. They decided that they're not going to leave this up to agencies, and they should take a stab at doing it. That's what we plan to do in the second phase. This suggestion came from Peggy Shepard in the last discussion that they had, so now it's a question coming from the larger group. You have a chance to contribute to the development of that scale moving forward.

Ms. Lopez-Nunez stated that, in terms of a framework, some agencies might actually have a negative score if you consider harm. They might be working towards some good things and have good programs, but then, if you only focus on the Justice40 aspects, an agency might end up with even a B or C if you average it out with not doing enough community engagement. But then, if you take a step back, and you actually look at the whole of an agency and they're actually investing a large chunk of their budget into things that harm our communities, then they might end up with a negative score. That in conjunction with the piece above it in part one about creating a culture that allows agencies to admit mistakes are actually painting a real picture of each agency, where they are, and how far more they need to go instead of always trying to paint the best picture possible. She wants to give people credit for the work that has been done but have a real landscape analysis of how the harm might bring us to a negative and then have to rebuild. Then the scorecard can take into consideration repair and then going above and beyond.

Michele Roberts mentioned that, when we look at the span of time from the TB epidemic to the COVID-19 pandemic, there has been a real severe lapse in data, time, and equations, and all of those things with respect to how do we address the repairable harm? In fact, some of those particular pieces from that of housing infrastructure went from bad to worse. So, with respect to that, how is it that they do make sure that we can look at that repairable harm but then make sure that there is a do no harm and a moving forward of remedy and redemption truly for these communities as opposed to the continuance?

Ms. Almanza asked if the scorecard would also be the one to measure all the programs and not just specifically those that have been sort of e-marked or identified as EJ. She also asked about giving credit for work like repairing or doing some of the changes and how they would do that measurement in all the programs. Or maybe just explain a little further how they envision doing those comparisons.

Co-Chair Shepard thanked Ms. Almanza for bringing up the issue of incorporating metrics around harm. It's such an important contribution to the scorecard. It would have to be very relevant to that particular agency, for example, if their highway air quality increased in pollution. Has water quality increased in places where they are dealing with lead service lines? They might think about some of those kinds of metrics. On the idea of penalties, that would be something they would need to incorporate into the Justice40 Workgroup recommendations rather than necessarily the report card. But the question of, should there be penalties for not ensuring that funding is going to the communities identified by the screening tool is an important principle there.

Dr. Wright stated that, when we think about an environmental justice analysis being applied to all of these programs, that would assure that things like highways being near communities would be a consideration. So she's trying to figure out how do we incorporate in the scorecard whether or not an environmental justice analysis has been done for each project. Because if that analysis is done and done properly, it would eliminate some of the things that we're talking about if they followed it based on the analysis.

Ms. Waghiyi stated that it's so important that they are having these discussions on scorecards, especially with government-to-government legal status and the obligation of the federal government and agencies. Her state doesn't even recognize their 229 tribes. She gave examples with ATSDR not including their community's observation and knowledge and expertise into their health assessment. They are still being discredited. They are not the only community that feels ATSDR health assessments are inconclusive by design. They let the polluter off the hook.

As tribes, they don't want more broken promises; they want real action to achieve environmental justice, enforcement, and to hold industry and military polluters accountable. When they are given these opportunities to meet with the government, these scorecards need to be in place to measure agencies like ATSDR and health assessments. They need to make sure measures are in place to protect their children and future generations. **Dr. Whyte** stated that what she said will be of a huge guiding significance for Phase 2 with kind of understanding the metrics of harm and holding agencies accountable directly in relation to actual harms and their intergenerational aspects.

Dr. Sheats asked, if something's sited in the community and it has harmful emissions, then that certainly should be captured by the scorecard. He also wanted to talk about the potential for harm, for example, a gas line's put through the community. This year there's not a leak, but we all know that in the future you raise the potential for harm in that community. Or a new chemical facility is put in a community, you not only want to measure the emissions but the possibility of fire. How do you capture that, that the community has been placed in more potential harm also? He avoided calling it a high risk because, as EJ folks, they don't normally like normal risk analysis, but they may want to try to capture that potential for harm or an increase in that potential for harm. **Mr. Logan** stated that it's really important to consider incorporating a precautionary principle for assessing in that realm of things, but then also, really thinking about what role the types of analysis that are required through NEPA and other types of NEPA processes to make sure that those don't supersede this scorecard kind of process and/or analysis.

Mr. Cormons stated that these comments are helpful to the Justice40 Workgroup who are thinking about the next set of Justice40 set of recommendations to influence the final Justice40 guidance. He considered, is this helping to hone the tool that they need to create real transformative change in agencies to implement a whole government approach to environmental justice? And to do that, he would ask, how would he want his work to be evaluated? Most people would hope to be evaluated in ways that appreciate and incentivize the very best work that they can do, where the right things are measured, rather than the wrong things. This set of recommendations for the scorecard really moves us in that direction, where they're looking at a scorecard that measures the actual impacts that agency actions have on environmental justice, the effectiveness of community engagement, and the degree of community engagement and participation that occurs.

If community engagement and participation are not occurring, that is likely because the right avenues for that engagement have not been put in place. To move agencies to a transformative place where they are looking at their roles in new ways, engaging with communities in new ways, and looking at the totality of their impact in new ways, they need a really good system of feedback and accountability and positive incentives. In conclusion, if he were serving in a federal agency where they're working on Justice40 or things that impact environmental justice more broadly, he and his agency to be evaluated based on these criteria. He would want to be incentivized to bring in the right kind of expertise to consider impacts fully to be able to engage effectively with communities. If they're creating the right feedback mechanisms and the right incentives for agencies to do that, then they're doing our job well.

Dr. Bullard gave an example of a Justice40 project that's a solar battery facility that's been considered a green facility that may produce a hundred jobs, there may be another side. When they are being scored and analyzed, the location may be the issue. In terms of the siting, the scorecard shows up that it's located next to a black middle-class neighborhood of homeowners. So, when you're scoring it and you're trying to balance out the economics, mental stress, property values, the scorecard won't pick this up. No white communities want this kind of green facility, and so it will gravitate toward the path of least resistance: a black middle-class neighborhood. **Dr. Wright** mentioned that Ms. Flowers asked if a map of who lives nearby could be a part of the scorecard.

Mr. Havey added an example in Minneapolis of how they could assess environmental justice. They developed a racial equity impact analysis that's required for anything that has a fiscal impact or has a policy or ordinance change. It just sort of provides information for council members and the mayor to work from and sort of look at the impacts on the environment. It looks at the economic and racial impacts of those types of developments. One of the things that have been really helpful though is that it's very standardized when it's used. He wondered if there is a way to standardize, at least some high-level portion of this for agencies to start using right away. For example, the mapping, if we have environmental justice maps, can we at least get some semblance of understanding how that money is actually being planned to be invested? Or some sort of standardization as to how they can report it, so you could compare apples to apples sort of across the agencies.

The other thing that would be really helpful is to get an assessment from agencies in regard to what are the types of things that are under executive branch control? There's got to be some way to understand what types of things that the president and his administration can do to make a difference on environmental justice based on what the federal government is currently doing, especially around the topics of harm, like the military's open burn and detonation. He asked how they're thinking about this being phased in? He wants to see something that can be used relatively quickly. Is there a way to get some of this going so that they don't get bogged down by making something perfect or as expansive as possible that it becomes difficult to get implemented? **Dr. Whyte** responded that they did have some meetings with several agencies, and the agencies have already been working on the scorecard approach. Sometimes it's more focused on their Justice40 work, but the workgroup did hear about updates more broadly on an environmental justice scorecard. So the workgroup does know at least that the agencies have been doing some planning work about the scorecard. As they move into Phase 2 and begin to hear more updates about what some of the agencies are doing, they'd be able to figure out that pathway for implementation.

Ms. Adams stated that there was a suggestion around looking at the Civil Rights Report that comes out annually. That's more adjacent to our education system, but it being such a long-standing and robust data set, has there been that type of consideration as far as the breakdown by ethnicity, breakdown by socioeconomic status, and how it could potentially be interoperable. So as opposed to just starting things from scratch, if there was a possibility to actually enhance this tool with things that already exist because there's a multitude of siloed systems. Has there been consideration about interoperability with systems or data sets such as what you would see with the Office of Civil Rights, for example? **Dr. Whyte** responded that they referenced civil rights in terms of Title VI, and they will consider her suggestion in terms of a pathway forward. **Ms. Lopez-Nunez** asked if all of these suggestions can be emailed to them. She also thought that the agencies should figure out the methodology themselves. Give them what data sets and citations they need and let them do it themselves. **Ms. Adams** said she sent over the existing Office of Civil Rights database to use as exemplars, and if there was anything else, to let her know and she'll send it over. **Dr. Whyte** invited her to join the working group. Ms. Adams declined but could send over any information they needed.

Vice-Chair Tilousi suggested, as they're looking at the wording, they add the word "tribes" basically because of the trust responsibility that the federal government has to tribes. As tribes, federal agencies are required to provide education, law enforcement, and health to our people because they took all our land. And, so, they have not provided those proper resources. The tribes that are more near the cities seem to get better benefits than the ones that are out in the really remote areas. So when she thinks about the scorecard, she's thinking about how they would make these agencies accountable to make sure that they're providing these services in their communities. She is trying to understand how tribes can enforce these agencies that are already providing services to our communities and how we envision this scorecard to help our communities make sure they're doing the proper work. **DFO Martin** suggested that they have two options for the wording. They can put make the necessary corrections of what was heard now and move forward. Or they can pass what they have now, and then in the next phase, they could also provide updates to whatever was submitted in the first phase. She stated that, at the last meeting, they did have some adjustments or some additions, and so, the Council voted to move it forward and then took a couple of days after that to make those adjustments before we sent it to CEQ. **Dr. Whyte** thanked her for that clarification. **DFO Martin** also suggested another option. Since they're planning to have a special meeting focused just on the scorecard, they can have an opportunity to incorporate what was heard and then pass it at that special meeting as well.

Ms. Waghiyi stated that she wanted to include a scorecard for the Department of Defense since she wasn't sure if they are part of the Interagency Council. She gave examples of how they have abandoned or buried their waste. There is historic legacy, and they continue today. She also mentioned HIS and their lack of care in the communities. She reminded the Council that she heard that 475 million would be going to tribes, however, there are 573 tribes in the nation, so that's less than a million per tribe. She talked about Veterans' Affairs. Some veterans are not getting adequate health care as well. **Ms. Lopez-Nunez** suggested that a recommendation could be that the scorecard should not just apply to the Justice40 section. They should probably put a bullet in there that the scorecard includes agencies that are not part of the Interagency Council. **Corey Solow** informed the Council that the Department of Defense is a member of the Interagency Council, but the Department of Veteran's Affairs is not currently a member of the Interagency Council.

Ms. Adams asked if there is a list of consequences? Thinking about a scorecard from a rubric perspective, are there concurrent thoughts around what consequences will be for these respective agencies that are included that are violating or not meeting the criteria that are set with the scorecard. Has that been a part of the discussion or is that a better fit for the next phase? **Dr. Whyte** responded that, on one hand, they did discuss penalties, but it's always good to hear anybody's ideas and their language about how best to discuss those issues. He appreciated the suggestion of a rubric. They will take that up in Phase 2 so that the examples provided would have incentives, penalties, or consequences as something they are going to be very focused on.

Ms. Belen Power suggested that, as they think about penalties, let's also make sure that the penalties don’t end up having unintended consequences of taking away funds from the communities that are the most impacted. So, if an agency is not doing its job and there are funds taken away or funds not available, then there's even more harm for the communities that are impacted. There may not be more harm, but there are more funds that are just not available for those that are most impacted. **Ms. Adams** clarified that, with penalties, it doesn't necessarily have to just be associated with funding. It also can be with people being removed from the jobs that they're not doing. That's a good point, but still, if they're talking about justice, justice needs to be served. And we have to think about consequences and not just present data that talks about what people are doing wrong, but then nothing is happening.

Vice-Chair Tilousi recapped the discussion from the workgroup and everyone who made suggestions. There were some questions on the sovereign nation tribes, the accountability, development of a matrix. They also talked about the comparison between the agencies. They discussed an assessment and an analysis that may be developed. She reminded everyone that they still have some time to make recommendations before the business meeting.

2.2 WHEJAC Justice40 Workgroup Update & Discussion

Co-Chair Shepard stated that her group will offer their responses to the significant questions that were submitted to them by the WHEJAC members. The Justice40 workgroup understands the prevailing expectations of WHEJAC members, the public, and those they represent in frontline tribal and EJ communities. Their expectations may not reflect the realities of how government official agencies are setting their goals, procedures, and methodologies. So, as a result, the WHEJAC members of the Justice40 working group invited agency representation from about six federal agencies to be here to respond to the questions they've submitted. She invited Dr. Wright to make comments.

Dr. Wright stated that she's hoping that they can get some clarity as they move forward on just the kinds of questions that community groups ask them so that they can answer with some authority. They are excited about the way forward that's been presented by the president, and they have put their hearts and souls into all of the work that they've been tasked to do. They've tried hard to make all of the deadlines, but where the rubber meets the road, is when they can tell communities that what we are proposing will be implemented and that this will help to solve some of their problems.

2.2.1 Dr. Candance Vahlsing, Associate Director of Climate, Energy, Environment, and Science – Office of Management of Budget; Jahi Wise, Senior Advisor for Climate Policy and Finance – Climate Policy Office; Dr. Phillip Fine, Principal Deputy Associate Administrator for Policy – U.S. Environmental Protection Agency; Dr. Gbenga Ajilore, Senior Advisor, Office of Undersecretary for Rural Development – U.S. Department of Agriculture; Dr. Tony Reames, Senior Advisor on Energy Justice – U.S. Department of Energy; Corey Solow, Deputy Director for Environmental Justice – The Council on Environmental Quality

Ms. Solow invited her colleagues to introduce themselves. She thanked WHEJAC for providing them with this opportunity to discuss the Justice40 Initiative. One year ago today, the president signed an executive order on tackling the climate crisis at home and abroad. It created not only this historic advisory council but established the Justice40 Initiative, which, for the first time, applies an intersectoral approach toward addressing underinvestment. The executive order recognizes the interrelated nature of the climate crisis, and social inequalities require us to tackle them together. It is not an accident that the most significant environmental justice initiatives, including the Justice40 Initiatives, were launched in the presence of a climate executive order. Rather, it is a recognition of the effects of climate change and solutions must integrate the principles of justice into our national agenda. To begin our conversation, she turned things over to Mr. Wise to highlight some of the work the Biden/Harris Administration has done over the past year to meet this commitment.

Jahi Wise hoped to spend just a few minutes framing the conversation within the broader context of the administration's climate policy and environmental justice policy. As Ms. Solow said, a year ago today the president signed Executive Order 14008, which established the WHEJAC and a host of other initiatives to advance justice and climate policy. Since the establishment of these different pieces of infrastructure within the government, the senior administration officials have worked tirelessly to, one, elevate the voices of environmental justice communities into the White House and throughout the administration; two, to secure and ensure a long-overdue investment in environmental justice; three, to advance science-based regulations that reduce environmental pollution; and, four, to strengthen enforcement of the nation's environmental and civil rights laws.

He proceeded to walk through just a few of the accomplishments of the last year as they gear up for the next year of good work. Agencies across the federal family have launched new or strengthened environmental justice offices, enforcements, strategies, and policies. To make sure that they're not just setting good policy out into the world, but that they're enforcing that policy. Two, the administration has taken over 200 distinct actions to repair damage caused by the prior administration's rollbacks and implement some ambitious regulatory agenda to address potent pollutants, including PFAS and others. Three, the administration secured the largest investment in bio and infrastructure law in tackling legacy pollution in American history and that's funded to clean up superfund and brownfield sites, reclaim abandoned mines, and cap orphaned oil and gas wells, among others. We've also launched a lead paint and pipe strategy, an ambitious strategy that marshals the entirety of government to leverage over $55 billion that's in the Bipartisan Infrastructure Law to make sure that disadvantaged communities, underserved communities, overburdened communities are not burdened by the scourge of lead in their water and in the paint in their homes any longer.

And, lastly, among a much larger list, they're going to focus on Justice40, which is the work that's been done through the federal family to improve air quality through a variety of rulemaking. Everything from lead and aviation fuels to heavy-duty vehicles has moved through the administration. We also made historic investments in community monitoring and electrifying ports and waterways while also investing in delivering clean school buses. This work is an important frame to show that Justice40 is a part of the administration's environmental justice and climate policy, a part that they're very proud of. The administration continues to move forward with Justice40 and with our broader environmental justice strategy.

Candance Vahlsing stated that the Justice40 program is part of the president's historic commitment to environmental justice. It's an initiative where certain federal investments are made to reach the goal of directing 40 percent of the overall flow of benefits to disadvantaged communities. The way that we have further specified this is the covered programs. They are programs that fall in one of the following categories: climate change, clean energy and energy efficiency, clean transit, affordable and sustainable housing, training and workforce development, mediation and reduction of legacy pollution, and development of critical clean modern infrastructure. So when they're looking at what types of investments as a whole Justice40 apply to, these are the categories of investments on which we focus.

What types of investments are covered within these categories that were listed? So at least five here are noted: one, federal grant and procurement spending; two, financing, including loans and loan guarantees; three, programmatic federal staffing costs to implement the types of programs that they've outlined; four, direct financial benefits; and five, additional benefits that may help overburdened or underserved communities.

Ms. Solow stated that this is a brand new and historic initiative within the federal government. As this body knows, after it was established in March, it worked tirelessly to provide recommendations on the Justice40 Initiative. Over a few short months, WHEJAC all shared some rich and robust recommendations -- nearly a hundred pages. It helped inform the formal instructions that the Office of Management and Budget, the White House Climate Policy Office, and the Council on Environmental Quality sent to federal agencies in July. Included in that guidance were the Justice40 pilot programs for 24 programs across 9 federal agencies that were required to meet all of the requirements in the guidance but on a faster timeline. These programs were designed to provide a degree of learning and help us develop best practices regarding the implementation of Justice40. By August, these programs developed stakeholder engagement plans, and, in September, they were required to share information with us on how they were going to change their programs to maximize benefits to disadvantaged communities.

Throughout the remainder of last year, the federal family submitted information on their programs that were covered by the Justice40 Initiative, as well as their proposed methodologies for calculating the benefits of those programs. Throughout this, there has been ongoing stakeholder engagement as well because the guidance requires the determination of a benefit of a program that agencies engage with communities.

Dr. Vahlsing stated that the most important thing here is where the rubber meets the road in delivering benefits. And, so, over the course of the year, they wanted to highlight some of the ways that they directly delivered benefits to disadvantaged communities. So, first, I'll mention that just last week, they made the largest investment in resilient physical, natural infrastructure. This was through $14 billion of investments in the Army Corps of Engineers. This included a variety of investments in disadvantaged communities, like Caño Martin Peña, which is helping support an urban tidal canal surrounding the San Juan Bay National Estuary. There was $40 million of investments in Espanola Valley, which is in the Rio Grande in New Mexico. There are $28 million of investments in the Kenai River Bluff in Alaska. And the Army Corps also committed to starting a process with environmental justice and the disadvantaged communities to figure out how to allocate $130 million through two pilot programs as a continued effort. And this is just some of the Bipartisan Infrastructure funding, only the funding for the fiscal year '22. And the Army Corps worked hard to ensure that they are meeting the goals of Justice40 when they were putting out that $14 billion.

She highlighted LIHEAP. Most people are familiar with the LIHEAP program. They wanted to note here that they delivered $8.2 billion in low-income energy systems funds, including 4.5 billion to the American Rescue Plan. There's an additional 500 million in bill funding that's also available. This is more than double the typical annual appropriations in the program's history. Another investment to highlight is they recently announced a historic $1 billion investment through the Bipartisan Infrastructure Law to initiate the cleanup of 49 previously unfunded superfund sites. This is a really big deal. Sixty percent are in historically underserved communities.

She gave a few more examples. They put out a lead pipes and paint strategy part of the funding in this. So, this $15 billion will go towards fixing lead service lines and replacing lead service lines. Within the area or existing programs, we've included funding criteria and solicitation so that benefits are directed to disadvantaged communities and that they get a higher score in the application process. So, a few of those are HUD's Choice Neighborhoods implementation grants, public housing radiation testing, funding for lead paint through HUD. So, there are numerous different programs where, across the government, they've really changed the ball here and they've made sure that different funding solicitations, when they're put out, are prioritizing funding and benefits to disadvantaged communities.

The last thing she noted is, as part of the bill implementation process, they're really working hard, which is one of the lessons learned from the pilot programs, to ensure that the funding, especially for some programs which are state revolving funds or formula grants to states, are maximizing investments to disadvantaged communities. And, so, one of the things that the EPA administrator did last year is he sent guidance to states and a letter to governors directing them to target the EPA's Bipartisan Infrastructure Law funding to disadvantaged communities to maximize and make sure that that funding is directed where it's supposed to be. So, these are just a few examples of how they're ensuring that the benefits are reaching disadvantaged communities in line with the president's Justice40 commitment.

Ms. Solow: Thank you, Karen. Would like to recognize next my colleague, Tony Reames from DOE, but I just want to make sure that his slide is next.

Tony Reames stated that he will give a one-year update on how they've approached the Justice40 Initiative in the Department of Energy. So the mission of DOE is to ensure America's security and prosperity, addressing its energy, environmental, and nuclear challenges through transformative science and technology solutions. This mission is carried out every day by over 60,000 hardworking federal employees and contractors. But they know that our energy system is not fair. They know that they face a climate emergency that continues to threaten overburdened and vulnerable communities, and those same communities have worn the burden of the energy system that they're trying to change. Now, at DOE, as the solutions department, they know that they have the technical tools to avert catastrophic climate change and transition to a system that is far cleaner than it is now. But our grand challenge is how do they undertake this transformation in a way that is fair and equitable? And Justice40 provides us the pathway and also gives them the tool thanks to the great work of the WHEJAC and all the staff that is working on this to begin to do that.

He then talked about how DOE is approaching Justice40. Secretary Granholm has made a commitment to justice through all of our work across the department. One of the first roles of that is to hire the Deputy Director for Energy Justice, Shalanda Baker, the first time this position has ever been hosted in DOE. When Shalanda came on board, they've created a host of ways to really integrate Justice40 and equity and justice and equality into the lifeblood of the department. And, so first, he built a team to focus directly on Justice40 made up of federal employees, contractors, and details from our national labs who are conducting research and writing documents and policy statements to help the program offices implement Justice40. They've also done stakeholder engagement activities, working with community groups, businesses, and other entities to understand Justice40 implementation on the ground. They have a metrics working group that is thinking about how we actually measure Justice40, measure benefits, define benefits, define disadvantaged communities. And then, to make sure that the program offices begin to integrate this into all program design and implementation, we created a Justice40 community of practice from all program offices across the department, representing some 75 programs. So, our calculation looks at Justice40 investments, a host of benefit categories, and looking at where investments are going across disadvantaged communities relative to total DOE investments. That should be greater than 40 percent.

First, they wanted to create a set of policy priorities that every program must consider when they are creating funding opportunities, designing their programs, providing guidance to states and localities. They know that energy burdens are disparate across the country, and so, a goal of Justice40 in DOE is to reduce energy burdens, then to decrease exposure to pollutants and other environmental burdens, to increase parity in the clean energy technology access and adoption, to increase access to low-cost capital, to create minority and diverse business enterprises around clean energy, to work on job pipeline and job training, to increase resiliency and increase energy democracy. Again, these eight policy priorities are baked into all of their guidance to program offices, all of our funding opportunity announcements, and they are very critical to how we're implementing the Bipartisan Infrastructure Bill.

Where are they in this first year of the Justice40 Initiative? On day 100, they wanted to be transparent and look at where dollars are going across the country. That's just one foray into the Justice40 Initiative and how we're doing on equity. So, you can actually go online and see the energy justice dashboard, which was released on day 100 and see the distribution across the country of dollars that DOE puts into the nation. You can look at it by different awardees, by different cities, by different categories. This data is public. It can be downloaded, and it can be analyzed, again, focusing on transparency both internally for them to look at gaps in their funding, but also for others to look at.

He highlighted a few funding opportunities and other activities in the department that are centered on equity and justice. And so, in July, our Office of Indian Energy contributed $12 million to 13 tribal communities to focus on energy affordability and energy burdens. They also launched an inclusive energy innovation prize. The first time they really wanted to bring diverse communities together, HBCUs and other minority-serving institutions to think about how they create clean energy from the ground up. In September, the Communities LEAP, which is a pilot program to provide technical assistance, so environmental justice and fossil dependent communities can create their own energy future and give them a pathway to make that happen. In November, the Energy Storage for Social Equity Initiative, $9 million to help underserved communities create a path to energy storage and increase their resilience. The Infrastructure Bill includes a lot of programs on the energy side that actually focus on disadvantaged communities, weatherization, efficiency block grants, and focusing on public school energy upgrades. They have a major hiring effort. They want our department to look like America, and so they launched the Clean Energy Corps in January with over 1,000 jobs, focusing on diversity, equity, and inclusion. And just yesterday, they created more clean energy access to jobs in diverse communities, the Minority Business Matchmaking Initiative. Over 300 people attended that, and they're going to have a series of webinars to match minority-owned businesses with DOE opportunities.

Gbenga Ajilore stated that USDA is a very large institution. There are nine mission areas, and it covers a lot of issues, a lot of topics, including agriculture but not limited to. So, in the rural development mission area, they touch a lot of aspects of all rural American life, especially rural communities of color. And so, I'm not just going to talk about the work of USDA in this Justice40 Initiative, but also the department's broader work on equity, climate, and justice.

So, in the Justice40 Initiative, they have over 60 covered programs covering a large variety of topics, such as rural development, farming conservation programs, forestry programs, and research programs. Some of the work that they've been doing over the last couple of months is thinking about what constitutes benefits, because as you look at these different programs, you're looking at research, what constitutes benefits from doing research? So they think about the economic research service, all of the agricultural research work that they do. What are the benefits to disadvantaged communities from that, like forestry programs? What are the benefits from those, like farming conservation programs, both for the producer side, like ranchers and producers and farmers, but also from the consumer side?

In rural development, they look at housing programs in rural areas, infrastructure programs, and businesses. The other thing is looking at, in terms of measurement, also identification of benefits. Part of that is doing the stakeholder feedback. Part of it is that as a department, they think about the benefits and what benefits go to certain communities, but we also want to hear from the community, what do they think are benefits? How do they see benefits? And then, how can we incorporate that into their work? As mentioned before, there are 21 pilot programs in the Justice40 Initiative. At USDA, they have one program, that's the Rural Energy for America Program, REAP for short.

So, the program REAP is administered by the Rural Business Cooperative Services Agency, one of three agencies in the Rural Development mission area. REAP provides financial assistance to rural small businesses and agricultural producers for the purchase and installation of renewable energy systems for making energy efficiency improvements. Rural Development provides loans, grants, and loan guarantees for rural communities for housing, businesses, cooperative services, and then for utility. In REAP, they offer loan guarantees up to 75 percent of eligible project costs, grants up to 25 percent of costs, and a combination of grants and loan guarantees up to 75 percent of costs. One of the things he mentioned about the REAP program is that rural development in USDA broadly, they're cognizant of the need to balance important priorities in this work. They want to ensure that small and underserved producers have access to support our renewable energy development, as well as ensure land tenure. So, we don’t see these as competing priorities, and the ultimate recipients of the REAP loans and grants are farmers. So, they're the ones who are in control of their projects. And we always want to make sure that we listen to the ultimate recipients.

In September, they had to submit an implantation plan for the REAP program to how we're going to maximize benefits. So, there are five things that they talked about for REAP, but these five things have applications across the current programs, not just across Rural Development, but across USDA as a whole. So, first, we created this priority points framework for people who apply for grants can get extra points depending on the administration's priorities. So that's COVID recovery, that's equity, and that's climate impact. And so, they measure equity using the CDCs Social Vulnerability Index. Some of the other things that they do for climate impact in measuring disadvantaged communities are using the Distressed Communities Index, which is an index that measures distress based on seven different census factors. So, priority points are kind of similar to what was discussed before in terms of funding prioritization and trying to make sure that money and investments go to the communities that have been historically underserved.

The second part of our implementation plan was to do better outreach, and so that was stakeholder engagement, both internally with our staff and externally with our communities. And, when they talk about better outreach, it's not just going out and trying to see what do they know? It's trying to figure out who are the people that we haven't reached before? What is the best way to reach them? And then, how do they use that to be able to show that we're listening to them? And so, part of that work is figuring out what are the different types, sometimes you say you can do an online survey, but in a lot of rural communities, there's no internet, there's no broadband, so doing that kind of thing doesn't work. So they have to figure out what are other ways that they can reach these communities, listen to them, and then make sure that we're accountable to them. The third thing they did is leverage intermediaries. They have webinars. They have listening sessions with mission-driven lenders, so these are community development, financial institutions, and minority depository institutions, reaching out to say native CDFIs or African American CDFIs or Hispanic CDFIs and actually helping them to access our programs.

Statutory changes, the REAP program provides grants up to 25 percent of costs. Now, for a lot of communities, especially rural communities, that's still not enough to be able to access grants. And so, some of the things that they're trying to do is to engage with the Hill and try to lower the barriers to access for a lot of these rural communities. And so, one of the things that we were able to do in the Build Back Better Bill was to increase that federal cost-share from 25 percent to 50 percent. And so, while the bill did not pass, they're still engaging with the Hill to try to get these things done. Finally, to help maximize benefits, one of the things they did is to map disadvantaged communities using the Distressed Communities Index and the Social Vulnerability Index. He gave an example of what that looks like.

He showed a map of Arkansas and its strategic investment toolkit. It uses information from the census, but then also, information about their obligations, their loans, and grants to these communities. What we do is create these different measures. One, in the pink, these are all the census tracks that are considered distressed by the Distressed Communities Index. With the Social Vulnerability Index, they can look at persistent poverty counties. Then they look at places where we take their obligation data and say, okay, which communities have not received any funds from us for the last five fiscal years? The green is the Distressed Communities Index, the census tracks that are distressed, but then also, census tracks that have not got any money from them. So, what that tells them is a visual kind of representation that these green communities have been underserved. And so, when they think about what place that they need to send their money to, what place that they need to reach out to, that green helps tell them, okay, these are the places that haven't received any money from them. Now, part of this, in terms of doing the work, is that they can't just look at the map and say, okay, this tells them that they need to go there. They need to do stakeholder engagement. That needs to be paired up with this. So that when they look at these places, they say, okay, well, why is it that these places haven't received money from them? This is where they have to reach out to the community, listen to the community, and understand what some of the barriers were.

You know, sometimes the barriers might be they didn't know about their programs. Sometimes the barriers may be that they have poor experiences with them. USDA has a poor history with a lot of communities, and so that might be part of it too. But they have to do better outreach to figure out what is it that causes barriers to access their programs. And then once they do that, then they're able to figure out how best to reach them. Sometimes it might be direct lending to them. Sometimes it might be tapping those CDFIs or minority depository institutions and having them reach out to them. And so, this is part of that example of doing the outreach and maximizing benefits to these communities.

What are some of the things that they're doing? While Justice40 is a very important initiative to have environmental justice issues, it can't be limited to just the Justice40 Initiative to pursue environmental justice. So, they can think about Justice40 Initiative as an accountability framework to help them to do these things. One example of a program that's not part of the Justice40 Initiative, but it has those elements to it, is they have this new Rural Energy Pilot Program, and this is going to provide up to $10 million to assist rural communities to further develop renewable energy. This program will provide grant assistance, particularly to underserved rural communities, to deploy distributed renewable energy technologies. Now, this was just announced a couple weeks ago, but one of the things that came out of this program is that our program REAP has a number of issues in terms of access and barriers. So they have this pilot program to see if this is a better way for us to be able to better reach these communities.

Another thing that they're doing is that USDA is working on procurement roles to ensure that farmworkers have greater workplace protections at companies who procure with the USDA. They're also supporting the White House's efforts to pass the Farm Workforce Modernization Act. The acquisition community is engaging in efforts to strengthen worker rights and protections. USDA, back in November, announced $86 million to improve equitable access to jobs, business opportunities, education, housing in underserved rural communities. And then, another mission area, Natural Resources Conservation Service rolled out $50 million in 118 cooperative agreements specifically focused on working with historically underserved producers. There are other mission areas, like the Risk Management Agency and then the Farm Service Agency, that are also working with historically underserved producers. So, there's a number of initiatives both within Justice40 and outside that are working across USDA to try to reach historically underserved communities.

In addition to this Justice40 Initiative and our environmental justice work, we're also trying to link this up to broader USDA processes. So, one of the things that he mentioned before was the Equity Commission. This is going to be just like the WHEJAC Council. It's going to be established to look at the history of USDA to try to address equity issues, and then, they're going to provide recommendations to the USDA in terms of how to improve equity both to our external stakeholders and internally. It's set up with a steering committee, and then we have several different subcommittees that are going to focus on specific issues because USDA's so broad. Our first subcommittee is going to be on agriculture and what's happened with agriculture producers, particularly black and native farmers. They are going to have another subcommittee that's going to be on rural community and economic development. And then, hopefully, the recommendations and the reports are going to come out this Equity Commission can help inform how USDA can do better and inform things like the farm bill. The farm bill discussions are going to be coming up next year. They're also integrating equity and justice into USDA strategic plan. As part of the other Executive Order 13985 on the whole of government review for equity, they're developing an equity action plan at the department, and our Justice40 and agent parties are going to be intertwined with that. Finally, they're going to be integrating equity and justice priorities into our FY23 budget planning.

Phillip Fine stated that first, he's going to share how EPA is approaching Justice40, and then he wanted to get into some very specific examples of how it's already being implemented. Then get a little bit into the benefits because you had a lot of questions that were submitted on how they're viewing those benefit calculations. A lot of this has been mentioned, but one way to look at Justice40 is, it's sort of two-fold. There's a mandate to improve the way they implement certain existing programs is to ensure that the benefits of those programs are maximized in and delivered to the communities that need them most. But it's also that accountability framework that his colleague at USDA mentioned, by which the level of change that they are able to achieve is quantified in a transparent fashion. They are held to account for meeting those goals. So, at EPA, from the top down, they are fully committed to both those elements, significantly improving the way their funding programs are implemented to advance justice while we also work to build that framework for accountability. Thanks to the infrastructure law, these programs are going to be able to make unprecedented investments in communities that are overburdened by pollution to solve some legacy EJ issues, such as water infrastructure, safe drinking water, and toxic pollution.

They have the political will, clear policy guidance, once in a generation infusion of dollars to make meaningful and lasting improvements in the protection of these communities' health and environment. As these historic investments are flowing into these communities to address pollution, they need to ensure it flows in ways that are rooted in the community's vision for their protection and their future. So, in terms of engagement, they have engaged with the NEJAC frequently. They have, again, talked to the WHEJAC. They also continue to hold multiple national engagement calls to gain feedback from communities and EJ leaders about how to implement the program across a wide range of our programs. They've engaged with thousands of community members in this way over the past several months, and we'll continue these opportunities and hopefully be able to do some of this in person soon.

EPA has six pilot programs. They're reviewing the pilot programs, and a lot of those are in the Infrastructure Law as a way to try to develop better tools and data, resources, and practices by which all of the other covered programs, which are 70-some programs the EPA is going to have, so those other programs can learn. And that's a lot of difficult work both in reorienting their thinking and the way that they implement these programs, but also figuring out how to calculate and track benefits. It's still a work in progress, but they're not going to wait until all that is worked out before they start moving forward and make progress because these resources have just arrived, and they want to be flexible to adapt as they go forward but also make progress where they know we can.

So he wanted to get into some very specific examples of how they're already implemented, and some have been mentioned already by Dr. Vahising. She mentioned the drinking water and clean water state revolving funds. These are in the pilot program, and they both received significant increases in funding under the bill. They represent the largest assets within our portfolio, and these are formula funding pass-through to states, so it will require that EPA work with our states and other regulatory partners to ensure that these resources and benefits flow in the direction that Justice40 wants them to. She also mentioned on December 2nd, Administrator Regan sent a letter to the governor of each state and territory to share. They will be soon issuing national program guidance but gave them a preview that the first priority is going to be ensuring that these resources are targeted for disadvantaged communities. They also are committing EPA to provide technical assistance to help those communities overcome those barriers in getting that funding through the SRF funds.

In addition, they announced our first allocation of the infrastructure funding with the superfund. This will jumpstart remediation at many of the unfunded sites across the U.S. This is very exciting. Initially, 60 percent of the new resources are going to be invested in historically underserved communities, far exceeding the 40 percent goal in Justice40. Going back to American Rescue Plan funding, they got $100 million there from Environmental Justice, $50 million directly in EJ grants to communities. Seven million of those funds are for a new pilot for the Diesel Emission Reduction Act to provide rebates for electric school buses. It's specifically targeted to underserved school districts, tribal schools, and the fleets that serve those schools.

And, again, this experience is helping them implement the infrastructure funding towards school buses in a way that furthers the mandate. Again, our Air Office also launched the $20 million grant program to fund air quality monitoring in communities that need it across the country, and they recently extended the deadline and we're getting a lot of positive feedback there. The Office of Land and Emergency Management issued an EJ action plan. One of the goals in that plan is the Justice40 Initiative. This includes providing direct and indirect benefits to underserved communities with technical assistance, grant application resources, to the extent we can. And then, it also compliments the recommendation for integrating environmental justice in cleanup and redevelopment of superfund and other contaminated sites. It was highlighted in the NEJAC report, so this is directly responsive to a NEJAC report that was asking for these changes in remediation and redevelopment in EJ communities. In addition to sort of this top-down approach the government often takes, they also know that these issues get solved from the bottom up, grassroots.

This past November, Administrator Regan led by example in his Journey to Justice tour, which focused on rebuilding trust with those that have been overlooked and underserved. Just yesterday, EPA released a list of concrete actions that EPA is already taking in response to what their administrator heard on that tour, directly responsive to the issues that were brought up by the community members that he met with. This includes aggressively using our enforcement authorities, implementing unannounced inspections, the launch of the Pollution Accountability Team to advance more aggressive compliance and air monitoring occluded facilities, and affirming the latest science-based regulation at the risk from ethylene oxide. They're going to use the old standard, not the one that was proposed by the Texas Council on Environmental Quality. These are clear examples of how they're centering our mission on equity and justice, and how they are looking deeply across all of our activities to implement Justice40 and our other equity and EJ goals.

They've had some questions on benefits, and he wanted to just touch on that briefly. So, on January 5th, Administrator Regan talked to the National Environmental Justice Advisory Council and committed to providing transparency in the tracking and mapping of where these investments are going alongside the benefits. So while benefits are important, so is tracking the dollars. They have heard that loud and clear. They were asked what types of benefits they were looking at. So they have, again, dozens of additional programs and pilot programs, and the benefits are going to be very specific to the programs. He gave a few examples of what they're evaluating: percent reduction in children's asthma-related hospital visits, tons of various pollutants that were reduced, reduction in hospital visits, reduction in missed days of work or school, number of home health assessments completed, increased access to health providers, job trainings, jobs created, technical assistance given to tribal drinking water systems and other community organizations, a number of deaths prevented. But this is difficult. This is difficult and necessary work they're fully committed to, but it's going to take some time to get it right. In many cases, these outcome-based measures are going to take years to achieve and demonstrate progress. In other cases, they don’t even have the data. They need to figure out how to get the data to calculate the benefits, and they need to figure out how to get the data without adding additional burdens on grant recipients because that would be acting counter to the Justice40 principles. Even tracking where the dollars go is more complicated than one would think at first glance, but they're working hard to make that happen.

But, as I said, and that hopefully illustrated by these examples, we are not waiting to work out all the metrics and calculations before they get started. They're continuing to make significant progress based on the mandate to steer funding to communities that need it. And have been historically underserved. In his conversations all across the agency, every major funding program at EPA is taking this seriously, and it is part of every conversation for every program that they have. This is what Justice40 and Administrator Regan have directed us all to do, and they are doing it, and they look forward to sharing as they go forward as we progress in all these areas.

Co-Chair Shepard thanked the presenters and stated that there was some very interesting information they've never had before, so that's a great thing. She invited the Council to ask questions.

Ms. Roberts asked if the water revolving funds as a Justice40 pilot require matching funds from state and local governments? And, if so, what about the disadvantaged communities who can't provide that match? **Dr. Fine** stated that he's not an expert in the exact matching requirements. He would have to get back to you with the specifics of those matching requirements that are required by statute.

Harold Mitchell stated that historically all the agencies are not as enthusiastic as what they have presented, and he wanted to know, what type of training will be put in place around Justice40 to where we see these efforts executed and implemented with the priorities that they have for Justice40? Because he's seen around the country in many of these efforts that you can hear the presentations like what you're doing, but when you get to the regions and you get to the states, that same attitude of execution is not there. He wondered; what type of training is taken into consideration? **Dr. Vahlsing** replied that they've done several pieces of training for staff across the government because, as they've noted, this is a historic initiative, and they know it's going to take a lot, like moving a ship. And so, they've, across the government, put staff meetings, staff training on those calls. They're taking a whole government approach here. They know that agencies are starting in different places and so, they want to be able to meet them where they are and encourage them to maximize this initiative and the work that they're doing across the government. They've also provided TA on the Development of State Engagement Plans. She invited him to let them know where there are certain areas where they should provide more training.

Mr. Logan stated that he'd like to learn more about the selection process for the pilot projects. How were they selected? Who was engaged in identifying the particular pilot projects, and how you may have used the recommendations from WHEJAC specifically to the Justice40 in the implementation plan rollout on suggestions on the lessons learned? And whether or not, at least within the pilot projects across the different departments, plan to engage with WHEJAC as stakeholders in that process? **Dr. Vahlsing** stated that, in terms of the pilot program selection process, they tried to get a variety of different programs from across the government that represented programs like LIHEAP, where we know there are targeted investments in disadvantaged communities now to programs that are hard and more difficult to work with, like state water revolving funds that Phil noted. Where it's formula funding, they wanted to dig in and figure out how they could develop lessons learned that can be shared with other agencies who also have formula funds. So that was their starting point which is how can they get a broad scope of pilot programs that represent the different challenges and opportunities that they're being faced in implementing Justice40 as a whole, so we could create a set of lessons learned that could be applied to the rest of the programs. As a whole, they were developing the entire guidance, including the pilot programs. The WHEJAC recommendations played a big role, and they made substantial changes in previous drafts in response to the WHEJAC recommendations.

Ms. Solow added that the interim guidance and selection of the pilot programs, as well as the identification of what could be constituted as a benefit within the Justice40 guidance, more than 90 percent of those examples were consistent with the recommendations within the WHEJAC report that was submitted in May. About 85 percent of the pilot programs that were selected were those that were also identified within the WHEJAC recommendations as programs that deserve greater attention or should be considered as part of the Justice40 Initiative. They relied heavily on the recommendations that WHEJAC produced in the development of this guidance.

Dr. Bullard stated that he was trying to get an update on the status of the administration's plan to address the black farmers. He knows there's money. He thought that last December the money was held up in a lawsuit in Texas. Will there be any Justice40 consideration for the black farmers and the other farmers of color that were discriminated against by USDA over decades and have not been made whole? **Dr. Ajilore** replied that he doesn't have an update on that, but he can connect you with people who do. He emphasized that this is also going to be a large part of the work that the Equity Commission, particularly the agriculture subcommittee, to try to find ways of being able to address those issues.

Dr. Bullard stated that, if we had a hundred black farmers on this call, he would suspect they would say, give us the damn money. Don't go to any other committees. They've been waiting since 1996 or somewhere back there. So there are some things that they need to get to and cut to the chase. Racism denied these folks their money, and it was the government, the USDA, and the policies and procedures. There are some things that they just need to own up to and do it, and hopefully, the black farmers can get some justice.

Vice-Chair Flowers had two questions. One is, how do they get copies of the pilot programs? And the second question is, how do they ensure that minority women and veteran-owned businesses in these communities also get a chance to compete for the contracts that will come out of these programs? **Dr. Vahlsing** replied that they're happy to share the list of the pilot programs with the group. With regards to the second question, that's really in the implementation, and so that's one of the things she would raise with the agencies as they are talking with them. It's helpful to hear that, so as they're going through, they will update the guidance. It's helpful to hear that that's a priority as we go forward.

Ms. Solow added that their hope with these presentations within the agencies who do have pilot programs as well as many other covered programs is an opportunity to share a lot of the information and the work that is happening to move those programs forward. They'd be happy to continue to work with DFO Martin and the wonderful folks at EPA to bring together conversations and bring things to provide more information in that space.

Mr. Cormons stated that he's thinking about the interaction between top-down and bottom-up, specifically that agencies need to receive top-down expectations from the White House to do work in a transformatively bottom-up way. They can feel their energy and enthusiasm for this effort. They can see the very innovative approaches they're using. The USDA map showing what areas have never received USDA funds, pointing to where you really need to dig in and figure out why that's the case, that's a really smart and impactful way to analyze where some of the failings are and where some of the transformations are needed. But, to go back to a point that Harold Mitchell made earlier, not everyone in every agency, or every division of every agency is going to be motivated necessarily to do that kind of hard energetic, innovative work to transform how they do things. And it seems like the only way that's going to happen is if there's the right and properly formulated top-down expectation from the White House that that happen.

So, the WHEJAC are advisors to the White House, and on the Justice40 Workgroup, they're going to be working on the second phase of our recommendations to inform the final Justice40 guidance. Thinking about what that guidance should look like and what kinds of protocols and expectations it should contain is top of mind for us in the workgroup and probably for other WHEJAC members as well. So how do they draft that to provide agencies with enough flexibility to get the job done but very clear expectations around excellent stakeholder engagement around reversing rather than just feeding into and perpetuating existing inequities as to who's getting these funding streams and to honor some of the other equity considerations that they expressed in the initial set of recommendations and were expressed in the interim guidance. That's the question before them, and he really wants to have that conversation. He'd love to get perspective from agencies. How do we incentivize agencies to do things like developing that map that you developed at USDA?

Dr. Vahlsing replied that she will go to where they are in the process, and next steps, and that'll help inform your question. So, right now, they're currently reviewing hundreds of federal programs that were submitted to OMB and to the White House representing billions of dollars in annual investments to ensure that they're meeting the goals that they set out in the interim guidance this summer. So that's the oversight process that they're currently going through here from OMB, CEQ, and CPO as a next step. They are also in the process of working on the climate and economic justice screening tool, which will be continuously updated and refined based on public feedback and research and will improve the consistency across the federal government on how agencies implement programs and initiatives that are intended to benefit under-sourced communities. So, the beta version will be released for public review and comment this year. The third thing is we are working on an annual, federal environmental justice scorecard, the first of which will be published this year, which will report on progress from the agencies and implementation of Justice40 and other key environmental justice priorities.

Dr. Fine gave some examples that they're doing in EPA in terms of holding everyone accountable. Some of this may sound bureaucratic, but they integrated environmental justice and equity into our four-year strategic plan, but from that flows all kinds of long-term performance goals, annual performance goals, and trickles down into program guidance that all staff have to follow and can also flow into individual performance evaluations. So there are a lot of accountability mechanisms that are already set up within the government that can be leveraged in this instance. Another thing the EPA did is there's parallel Executive Order 13985 on equity. They submitted that to OMB recently, and, in there, they recognize that building the capacity of EPA internally to do equity and justice work is paramount. So part of that is training is, part of that is, again, accountability through individual performance plans, and also working with our state and local partners in getting them the training that they need as well to be able to carry this out. Those are just some examples that they're already implementing, but obviously, there's more work to do to execute on that.

Dr. Reames added, at DOE, he presented the eight policy priorities. Those policy priorities link directly onto metrics that they're having programs measured through the Justice40 Initiative, but also, our annual FY programming. They have our community of practice where a representative from all of our program offices is learning about energy justice, environmental justice, and they're also including that in our strategic plan and the equity analysis that we've done for the department.

Dr. Ajilore stated that he posted in the chat a link to our strategic investment toolkit. A lot of it is about trying to put in the accountability measures, put it in people's individual performance plans. They've created a key performance indicator about the share of investment that go to distressed communities so that we match that. At Rural Development, also FSA, they have state directors, and, as they get new state directors in, they set up training programs on a lot of our programs, but also, they do training on equity and justice issues. The other thing that they're working on is trying to figure out how do they do, like, how do we diversity, equity, inclusion, accessibility, and train our staff so that the staff understands not just how to do it, but why to do it so that people are centering equity in all the work that we do. **Mr. Cormons** replied that informing things like that could appear in guidance, encouraging, or setting the expectation that other agencies take similar steps.

Ms. Santiago suggested that the slides be shared. She stated that the funding for the Caño

Martin Peña dredging is very much appreciated here in Puerto Rico. It's one of the main issues that's currently being discussed and so, if the dredging does happen more than 27,000 people will be spared from flooding with sewage water regularly. Her first question pertains to the hurricane season that will be starting up in a few months, and so we all know what tends to happen with hurricanes, not just in Puerto Rico, but in the Gulf Coast. Transmission lines get knocked down. Division lines get knocked down. They've seen the resilience. Energy resilience really depends on having distributed energy, especially distributed renewables, like rooftop solar and storage. They know what DOE is talking about, and they're part of the PR100 Study. But that's a hundred percent. That's a two-year study. So, in the meantime, can they see FEMA funding for distributed renewables start right away so that the communities and people can be resilient, and we don’t lose the lives of thousands of people for a lack of energy supply? That's number one.

The other question is there's a concern here, and it has to do with very controversial funding by DOE of a study to site nuclear reactors or a nuclear microreactor in Puerto Rico. As you know, they had earthquakes here. They're still experiencing aftershocks. Groups are being formed. She is not a part of them yet, but there have been groups being formed to oppose this siting of nuclear reactors here. So, what was the thinking? And this all goes to the issue of how the administration is defining clean energy if they're including these microreactors.

Dr. Vahlsing thanked her for the comment. On distributed renewables and FEMA funding, FEMA is not represented on this call today, so Ms. Solow will have to get back on that and then defer to DOE on the other question. **Dr. Reames** stated that they are pushing that stakeholder engagement, recognizing that there is an all above approach to clean energy, but communities should have a say in what comes to their communities. And so, they will continue to follow up on that.

Ms. Waghiyi stated that she has a question for Dr. Reames with the Department of Energy. Are they looking at microplastics in our oceans? Alaska is a heavy fossil fuel industry state with a decline in oil and gas development. There's a push for more plastic production, and we now know that the Arctic Ocean has the most microplastics of all oceans. The other question she had was for Mr. Fine with EPA. Are they looking at and taking into account formally used defense sites? We have been asking for the ranking of Northeast Cape. It ranked high enough for the National Priorities List, but it was never placed. **Dr. Fine** replied that he doesn't have the details on that site, but he'll definitely look into it with the Office of Land and Emergency Management and get back to her. As far as the superfunds, $1 billion that was announced is just the first set of funding that was announced, so there will be more coming forward. But he doesn't have the details on how that office is working down that priority list. **Dr. Reames** replied that he can follow up with her on the microplastics.

Ms. Waghiyi asked about mining lithium for solar panels, and it's done near people of color and low income. **Dr. Reames** replied that there is critical mineral mining for clean energy, and they can share that information with her what critical minerals and where they're coming from.

Dr. Sheats stated that one advantage of Justice40 is that the benefits are going to go to disadvantaged communities if it works properly. Outside of Justice40, the agencies need to ensure that also. Whenever there's going to be some kind of environmental benefit, there needs to be some EJ language in there to make sure that EJ communities get them. We shouldn’t have the assumption that it's going to benefit everybody. For example, the superfund cleanup, even though 60 percent of the communities on there may be low income, we still need to take steps to ensure that the EJ communities are prioritized. It seems like some things have been done with funding but also for benefits outside of Justice40. The flip side is, specifically for EPA and DOE, we need to figure out how to keep some of those things out of our community with the siting. Particularly for DOE, we probably haven't bothered you as much about that as we have the EPA, but that goes for both agencies. They need to address siting issues, and that brings us back to cumulative impacts also.

Co-Chair Shepard asked the Department of Agriculture, how are they going to ensure that the benefits go to those communities? And, then her second question is to Dr. Fine about evaluation taking several years. **Dr. Ajilore** replied that that map is the state of Arkansas, and one of the things that they have is this initiative where they're trying to highlight, at least for now, a maximum of ten communities where that's the case where they haven't gotten any investment. They have a three-phase project where they try to, first, listen to the community, then figure out what they need, and then try to figure out, not just within the Department of Agriculture, but across the federal family, investments to do that. This is kind of a one-year project. Hopefully, they're able to use that as a springboard to ask for more resources and funding to be able to reach other communities too.

Co-Chair Shepard asked if they also mapped where there's a lack of waste systems and electricity in some rural communities in the south? Have they mapped those areas as well? **Dr. Ajilore** replied that he didn't have details about the specific things, but they can do that. So right now, it's just about funding and obligations and investments, but they are working on trying to be more flexible and trying to figure out what type of things they're going to be able to map to be able to address several different issues. **Dr. Fine** replied that there are some metrics they can use right away, and some programs have come up with that. Other metrics are more outcome-based that will take time to get the data on. For instance, if you fund a school bus replacement. You're changing a diesel school bus for an electric school bus; you can calculate right away the emission reductions that were achieved from that action. But if you're going to track a reduction of asthma cases or asthma incidents in those children, you're going to have to set up a data collection system to track those outcomes. That's what they eventually want to get to. They want to track the reduction in disparities of these health outcomes. But it's going to take time, so there are some things they can do in the short run, and it depends on the program. But what they really want to get to is more health outcome-based, and that's going to take a little more work.

Co-Chair Moore reiterated that EJ communities are very diverse, and they include Native Indigenous, Latino, African American, Asian Pacific Islander, and other low-income communities, including white, low-income communities. Additionally, they've had some excellent presentations on Justice40. He's attended some of those in the various workgroups. But when they have staff coming on to request slides of presentations, you can't really share any slides or documentation that's been presented. Many grassroots groups have to almost create their own documentation and presentations on Justice40, and some of the groups out there have already done that. They need to give communities some pieces of material because the community deserves to have them. Justice40 is something new, but the same kind of training that's being discussed in-house should be discussed out-house. The other quick last one is penalties. Penalties are going to keep coming up until they get an answer. Lastly, he's attended several of the Interagency Council's training. Some have done a very good job in terms of explanations and engagement. They could always do better.

Vice-Chair Tilousi noted that, as they're discussing the Justice40 and how funding is going to trickle down to our disadvantaged communities, she wanted to make sure that tribes are not stuck with a cost share or a match. As they're developing this program, all those matches need to be waived. The bigger tribes get more money, and the smaller tribes get less money, and that happened in the COVID as well. So they want to make sure they are looking at the need, and the rural communities are the ones that really need the support, especially during these difficult times.

Ms. Roberts reiterated that they need staff training across the federal family. It is one thing to say they will embrace environmental justice across the federal family. She wanted to make it clear; some have grave disparities and have had some deep trauma and distrust and mistrust, as you have heard time and time again. While those people know that agencies are very well-intended and very well-academically trained, they want to make sure that there is intentionality in undoing racism. So, it is incumbent upon this administration to make sure that they are addressing these disparities and making sure that the staff can clearly hear the challenges to effectively provide the remedy and redress that's needed.

DFO Martin informed everyone that is time for the break.

2.3 WHEJAC Business Meeting Reflection & Conversation

The WHEJAC will use this time to reflect on the meeting proceedings, public comment period, discuss and deliberate action items, and finalize the next steps.

DFO Martin reminded everyone that they needed to talk about the recommendations for the scorecard. They don't have to make decisions on all of these topics, but they definitely needed to decide on what to do with the recommendations that they're going to put forward.

Chair Moore reminded the Council that they need to keep a quorum to make decisions and that the comments need to be brief. The briefer they keep them and get their point across, the quicker they'll be able to move along fairly quickly. The first business discussion is about public comments. During the last public meeting, the WHEJAC started a discussion about what they would like to see happen with public comments. There were three options for discussions. If members have other options to add, they should please raise their hands. Then they'll go through the first two options or requests from CEQ and IAC. These are two requests that could be put forward as recommendations during the business meeting. He invited members to respond. **Dr. Sheats** noted that the first two options are for how agencies will respond. The third option is for WHEJAC.

Mr. Logan offered that as part of how they're going to respond to the commenters, they should also establish a protocol. Right before public comment, they should make folks aware of limitations as WHEJAC members so that there are no unexpected expectations of what their authority is. They have very limited authority if any at all.

Dr. Wright stated that this is a real sore spot for her on public comments and who's responsible for answering since going through the NEJAC. She doesn't believe it's their responsibility to answer these questions; it's the agency's responsibility. She's been in meetings where people were angry with them and thought they were the culprits. She agreed with Mr. Logan about making certain that people understand their role. When you're still trying to answer for the agency, you become the agency. They can ask more questions to assist the agency in getting to the answer, but she wants no parts of answering questions for the agency. That's a precarious place for us to be.

Ms. Lopez-Nunez stated that, as someone that has not always been involved with the political system, it's really hard to determine what to share and where. For instance, with the next public meeting that's going to be specifically on the scorecard, hopefully, they get more comments about the scorecard because, in terms of folks publicly commenting at the WHEJAC, it should be more of a collaboration between communities and the WHEJAC. It sounds like a lot of community folks are speaking to the agencies and using the meeting more as a platform to voice their issues. She wonders if there would be a way to get more directed comments that help them connect between what communities want and the role they can play as WHEJAC to advance those concerns or incorporate those concerns into their recommendations.

DFO Martin stated that, with this special meeting, they will try to tighten that language up a little bit to let people know that that is the focus area of this meeting. For this last meeting, we said we would be discussing Justice40 scorecard, but as you see, communities want to talk about what they want to talk about.

Co-Chair Shepard stated that she sees no reason for them to be responding to questions. They should respond on is what the process is, and if one or two or three of these options that are being shared become the process, then they should be stating that in front of every meeting. Now, the special meeting on the scorecard presents an opportunity to hopefully get comments that might be helpful on the scorecard. It is sort of is frustrating that very few of the comments are about the work of the WHEJAC. If any people think that they should be making comments, she'd like to know what those comments are because they have no authority to do that.

Ms. Adams asked is it not possible even if it has to be some type of support from an external source for us literally to have clean, very legible, well-understood by layman answers in a centralized location? So, just in the spirit of transparency, what's happening is there are duplicative questions that are being asked because there's no clarity on what the answer is. Even if it is specific to an agency, even if they don't know, just say that because it's going to keep coming up. The community and members will continue to be frustrated if it's not addressed.

What happens from a community facing people, if you have folks coming in literally saying the same thing over and over again, it feels like it goes on deaf ears. As a community leader, it feels awful to sit and listen with no point of recourse. People just came, they talked, and then that's it. What happens after that? How are we holding ourselves accountable as WHEJAC and the administration around these existing questions that people have? Make it plain as opposed to honestly making people feel like their time has been wasted.

Dr. Sheats responded that, when he said how they should respond as a council, he didn't mean that they should respond on behalf of the agencies. The things they can do as an advisory group is they can write letters of advisory. They could write letters to the administrator telling them what they think they should do. They could form a workgroup and do a report. Those are the two things we can do. When he was on the NEJAC and people like veterans would testify, they would ask us, will you, NEJAC, write a letter to the EPA administrator saying to do this or do that?" or whatever they were testifying on. They didn't do it very often, but a couple of times they did. After every meeting, they would get a summary of what people said, and they would decide as a NEJAC, do we as a NEJAC want to write a letter in support based on the testimony that people had given to the EPA administrator?

Dr. Bullard agreed that it's very important that people understand WHEJAC's role and the power, or lack thereof, that they have. They do have a position of summarizing what comes in and sending it forward. They do have the authority to pull out, synthesize, and summarize. If there's a pattern, that may rise to the level of the body itself drafting something and sending it forward as a recommendation. They have no power to do anything except to send it and hope it'll land on receiving ears and eyes, then maybe it will be taken into consideration.

A bunch of them have served on NEJAC and they had subgroups and they did reports and they did recommendations. Some of it had an impact; some of it just landed flat. In terms of questions that come up that the administration, the agencies, it's their responsibility to provide answers. Then WHEJAC needs to take the answers and boil them down into what does it mean in terms of their next move? If they get answers that are runaround, then that would trigger something in terms of whether or not the agency is serious or not. When people say, "We can't answer now. I don't have the information," those answers need to come back to us.

Ms. Waghiyi read from the possible options for public comment, "Request CEQ/IAC to establish a point of contact for each agency to attend public meetings to hear public comment." She suggested that there needs to be stronger language than "request." It needs to be stronger language. It's just a request. If we want them there, they need stronger language to make sure that they are there.

Mr. Parras stated that there are ten EPA regions, and he asked if they could get a report on how each of the regions has addressed environmental justice in their region and also identify the communities in their regions? There isn't representation from each of the regions or communities in each of those regions, but he wondered how they can get more participation from them. He suggested that someone go to each region independently and explain what environmental justice is because they're missing out also on a lot of communities that are overburdened but have no idea that the EPA's out there with an environmental justice agenda to help them out. **DFO Martin** reminded Mr. Parras that they are providing recommendations to the entire federal government and not just EPA. The regions are different across all federal agencies. **Mr. Parras** was just wondering how they reach out to all the regions so that they all get the same message about who's available in the federal government to address their concerns on environmental stuff.

Ms. Adams reiterated that, when there's a question that comes up, whether it be from the WHEJAC and/or from public comment, those respective agencies need to be prepared to respond. That's the least thing that they can ask for, and that would mitigate some of the exhaustion from some of the questions that are for people who are already being paid by tax dollars. That it would demonstrate a model of accountability so that they will then go on record to either provide a thorough response or say that they don't know.

It behooves the respective agencies and those who work there to actually be present. Be professional and do a presentation that they're demonstrating their due diligence and incorporating things that are reflective of the community to the people who show up to voice concern as opposed to them putting on dog and pony show. So don't just write it in your notebook; they need to actually go back to their respective team and do their due diligence to respond to the community.

Vice-Chair Tilousi stated that she looked on the website on the public comment and it read, "A public comment period relevant to the specific issues will be considered by WHEJAC during the public meeting." Some of the presenters asked her what are the specific issues that are being worked on, and what are the status so that they can comment? It seems like, when they're discussing issues, the community has the right to contribute what they want them to advise. She also asked if the public commenters, who signed up to speak but couldn't because of time, are going to be given an opportunity to speak again? She doesn't want people to feel left out of the process. Do they get moved to the next public hearing? Their comments should go on record, go to the White House, and someone needs to respond to them. Somehow the WHEJAC has put themselves in this position to become the ears for the voices of the people.

Vice-Chair Flowers stated that they have to be practical. In terms of CEQ, one of the things that they've discussed is how short-staffed they already are and they're trying to make sure that they get more staff so that they can address these issues. The Council didn't get all the answers that they wanted because CEQ is still in the process of trying to develop something new and different. Maybe WHEJAC can use the comments to inform future people they would invite to come to our meetings. Maybe grouping some of the comments that deal with the same issue may be an easier way to address them. Part of that can be to deal with the charges. Maybe they could have some public meetings that would just deal with some of the comments that have been raised about the environmental justice issues and have representatives come and address them.

Mr. Logan said that he heard comments related to the impacts and concerns around mining. He suggested putting together a working group or a form of recommendation to the administration in terms of mining-related procurement of the materials needed for the advancement of zero-emission technology.

Chair Moore clarified that they are the White House Council and not the National Environmental Justice Advisory Council. He encouraged the WHEJAC Council to move forward with the request to bring in additional staff for CEQ. When new staff comes in, some of them are coming from other agencies. That's good because there's government language that carries along. On the NEJAC it was mandated by the administrator at that moment within the EPA that the regions had to have regional representation attending, not only, as we're doing Zooms, but in those moments on face-to-face meetings. WHEJAC has got to figure out a way to make sure on the interagency side that the interagency members are mandated not only to listen to public comment but to report back. They report back to the individuals that made the public comment, and they report back to the WHEJAC Council. So, there's responsibility and accountability at the same time.

He encouraged public commenters to hit the point and then make a recommendation. There were several recommendations made on two previous public comments, and some of them were interagency-related. His last point was that he appreciated that the CEQ staff is looking at the registrations that are coming in for public comment. They're looking at who has testified before and who is new and moving them closer to the top because they haven't testified before. At the end of the day, many of our people see the WHEJAC Council as a place that they can come to and express their frustrations, dissatisfaction, whatever it may be.

DFO Martin stated that the Council did talk early on about how much time to allot for public comment. Right now, the agenda allots at least two hours on the agenda so they could hear from everyone. There were 158 people who registered to speak at this meeting; only 37 spoke. If the Council wants to spend more time on public comment or do additional days of public comment, then they have to consider that with the agenda and that means longer meetings. The Council needs to discuss how long and how often they want public comments.

Chair Moore recalled that, at one point, there used to be a two-minute public comment. Then the community said that wasn't enough time to get their ideas together and make a testimony. Then it was moved to five-minute public comment, and that was too long. So then it was moved back to three minutes.

Ms. Adams suggested requesting hearings with particular agencies that have a large number of comments that are respective to their agency? Can we capture that and say they've had X amount of data comments on this particular topic? Is that something that WHEJAC has the wherewithal or power to do? **DFO Martin** replied that they can make a recommendation, document it, and forward it to CEQ. She noted that there have been five public meetings since their inception with almost 3,500 people attending. They have received at least 500 people submitting either written or oral public comments. So there are quite a bit of public comments that need to be sorted. Some of the staff at EPA started to sort through them, but it's going to be a heavy lift to go through, categorize, see what the things are, and get back answers.

The other thing that they've been looking at is trying to see what areas of the country folks are coming from. There needs to be a space where they need to do some additional engagement to hear from folks in other areas of the country that they're not hearing from. George Ward was starting to group the public comments by regions of the country so that way they can look and see what was going on in particular areas. She asked if anyone has ideas on how they want the information broken out, she can talk with them further.

Dr. Wright stated that she was confused about the charge of making another workgroup. There needs to be one around these legacy issues that just drag on. The workgroup can almost demand that they take a fresh look at this and come back with ideas to resolve it.

Chair Moore expressed his concern that some people in very rural areas have no access to a telephone, cell phone, computer, and any electricity. They have to drive to wherever they need to go to make a public comment. He proposed to the members that they move to the scorecard as the next agenda item because that's one of the points that they're going to have to take a deep dive in here for a minute.

Ms. Belen Power asked about being a conduit between the agencies and the comments. Are they deciding on what they're doing with the comments or are they moving on without making a decision? **Chair Moore** replied that they will look at those recommendations and then home in on as a council how they're going to move forward. He moved on to the next item -- the scorecard.

Dr. Whyte stated that he made some modifications to the document and highlighted them. He stated that the majority of the discussion was extremely informative for how they're going to do Phase 2. The first one was a change in the wording and typos on page 2. He also made changes on page 4 about including organizational structure, program development, and program organization as part of how the indicators are understood and incorporated. **Chair Moore** acknowledged that some of the points that were brought up now have been included in the documents so that they can move to call for a consensus vote on this. He opened the floor for any questions or comments.

Mr. Logan agreed with moving towards consensus on the changes/edits with the understanding that the other considerations that were raised in terms of assessing risk incorporate or consider the other types of principles around precautionary and so forth for the next phase.

Vice-Chair Tilousi suggested that "tribal nations" be changed/edited to say "tribes and indigenous nations" because all our federal contracts, agreements, MOUs, and all that refer to the federally recognized tribes as tribes. She also suggested adding the words "potential harm" to protect against contamination that's about to happen. **Ms. Belen Power** suggested using both and not put the word potential in front of harm, so harm or potential harm.

Dr. Whyte suggested that they put tribes/indigenous people. He agreed with the suggestion of including the words potential harm. So word it "where harm is occurring and potential harm is possible." Although they need to be cautious because potential harm sounds a lot like risk and precaution. **Dr. Wright** stated that she's happy with harms and potential harms.

Mr. Havey cautioned against "agencies will develop their own EJ scorecard for application for their specific programs and mandates." He thought it should add something about bringing it back to WHEJAC or CEQ for approval because, if they're developing their own scorecard, there's no transparency or accountability. His second concern is with saying, "Agency-specific EJ scorecard should have a data developing environmental justice indicators that map under short- and long-term environmental justice goals." Who has determined what those environmental justice goals are?

Dr. Whyte suggested that there should be a sentence referencing that there should be further than just agencies promulgating by themselves but that CEQ would have a role. He asked if his point is that they would have some kind of sentence that would specify how agencies would develop their environmental justice goals and it would include some relationship to the WHEJAC? **Mr. Havey** replied, yes. There just has to be a reference to something that they've been involved in creating the policy or basis around so that it's not just completely left up to the agency. It lacks accountability. **Dr. Whyte** offered that, in Phase 2, they would determine a recommendation for the role of the WHEJAC in the development of EJ goals that agencies develop for the scorecard.

Mr. Havey wanted to make sure there was accountability on the development of the EJ scorecard but also on the EJ goals that flag the scorecard that they're using to develop the policy. **Dr. Whyte** replied that they don't actually know where each agency is. Some may have already done their goals, and so it'd be ready for that further engagement. Others might not be that far along. If people agree with the gist or the spirit or the main ideas of what he is saying, it could be fairly straightforwardly articulated if folks entrusted them to make those revisions. **Ms. Waghiyi** wanted to make sure polluters have to pay for this harm. They need to put money aside for these potential harms. **Dr. Whyte** stated that they'll make an edit to make sure that it's not just read as referring to the past but is also forward-looking.

Co-Chair Shepard asked who will be publishing the scorecard? Who is doing the scoring? **Ms. Solow** responded, as outlined in the executive order, the Office of Management and Budget in consultation with the Council on Environmental Quality and the U.S. Digital Service are responsible for publishing that annual scorecard. **Co-Chair Shepard** asked who's responsible for finalizing and drafting the content of that scorecard? **Ms. Solow** responded that the scorecard is also mentioned in relation to the Interagency Council, which the WHEJAC advises. So, in the development of that scorecard, it is critically important that they get the WHEJAC's recommendations to help inform the development of the forthcoming scorecard. **Co-Chair Shepard** replied that they've had no interaction with the interagency working group. So, that's not in line with the executive order apparently.

Ms. Solow responded that they look forward to receiving the recommendations. Chair Mallory, who attended yesterday's meeting, is the chair of the Interagency Council, and they will work to make sure that the recommendations that are provided are shared with the Interagency Council. She knows about the request for a meeting about the other issues that have been discussed and the desire for more engagement with agencies that will all be returned back. She hoped some of the conversations from this meeting with our agency partners have been helpful in the development of more recommendations and of increasing the access and opportunity for the WHEJAC to engage with these agencies but, most importantly, for our agency partners to hear directly from members of the public.

Dr. Whyte added that, for the record, it's true that besides the fact that they've been told all recommendations have gone to the Interagency Council, none of the WHEJAC or workgroups has any closer engagement with the Interagency Council. He asked if that was correct. **Co-Chair Shepard** said it was correct since the beginning of the WHEJAC. **Ms. Solow** replied that the members who have presented to the workgroups and the members that equally presented at this meeting are all members of the Interagency Council. **Co-Chair Shepard** responded that haven't met as a group. They're not understanding what their concerns are and what the discussions are. **Ms. Solow** reiterated that the individuals that have presented are all members of that council. **Co-Chair Shepard** clarified that, unfortunately, they are not the ones who are going to be publishing the scorecard, so they're going to have to be realistic and thread the needle on what they're really asking them. **Chair Moore** stated that it sounds like they're requesting a meeting. **Ms. Roberts** stated that's why Chair Mallory must be on these calls to provide us with updates given her role on the IAC. **Vice-Chair Tilousi** agreed that she needs to meet with us more often and work with us closely so that we can make the proper recommendations on a timely basis.

Dr. Whyte recommended that they vote on the current draft of Phase 1 as our recommendations for the scorecard with the additional point that we will add in, in good faith, all of the points that were raised in the business meeting. **Chair Moore** asked the members to vote on moving forward with the recommendation. **DFO Martin** said they have a consensus.

Chair Moore asked to move on to the next agenda item. **DFO Martin** stated that the next item would be the climate resilience charge. **Chair Moore** opened the floor for discussion.

Ms. Santiago stated that she doesn't have a clear idea of what it is that the workgroup would be doing specifically as the recommendations on resilience in disaster situations. **DFO Martin** replied that they're going to want to work a little bit to create that chart. CEQ wanted to give us an introduction and let us know that it's coming. So, the workgroup will do a couple of consultations with them to flesh the charge out so WHEJAC will know exactly what the focus will be. And so, the Council needs to agree to start the workgroup and who will be on it so they can start that process. **Ms. Santiago** suggested that, in order to commit to being on the workgroup, they would need to know what's involved. **DFO Martin** replied that the Council can wait until there's more information, or they can start it now and whoever's interested right now that wants to work with them to scope it out can do it that way. She reminded everyone that all charges that are received through CEQ and the White House are charges that they need to start workgroups on to work. The issue is the timing of when they do it and how quickly they get moving on it.

Ms. Almanza asked what the timeline is for the workgroup and what exactly is the charge? **DFO Martin** replied that's something that they can get more details on. She clarified that the WHEJAC was created to provide advice and recommendations to IAC/ CEQ. So, any charges the Council gets for them, that's part of their role. So, the Council will be getting a charge. The presentation was to give you an introduction of what the plan is and how they want to move forward, but they want to do that in consultation with the Council to make sure that they get to or charge that's going to be useful for the outcomes that they want.

Co-Chair Shepard stated that it sounded like they are voting on the committee now or at another time. **Chair Moore** clarified that there are two questions: are they defining and refining the charge itself and who wants to sit on the workgroup? **Ms. Lopez-Nunez** stated that it was a big charge, seven different categories, so she agreed with the refinement. She is willing to work with them on refining it because her concern was that the charge seemed rather large for one workgroup. **Vice-Chair Flowers** volunteered to be in that working group.

Vice-Chair Tilousi commented that she was in a working group, and they worked really hard to meet the deadlines and they never got a response. If they give us a charge and they accomplish it, CEQ needs to respond; otherwise, it's a waste of time.

Ms. Roberts and **Ms. Yoshitani** volunteered to be in the workgroup. **Chair Moore** volunteered to be on the workgroup as well. He called for a vote on the workgroup. **DFO Martin** stated that there was a consensus.

Chair Moore thanked everyone and moved on to the next agenda item. **Co-Chair Shepard** stated that they asked the CEQ to provide timelines on some of the key initiatives that they are waiting for. So, the first one was finalizing the interim guidance. The interim guidance on the scorecard will be finalized supposedly at the end of February, but they're not clear whether that deadline will be met. So, they need that information.

The Council wanted to know when they're going to release the beta version of the climate and economic justice screening tool. At the last workgroup meeting of the screening tool, they could not get a response on whether it would be ready by Earth Day, which is in April. There was no response as to when that would be. However, when they do release something, it will be the beta version, which could be out there for comment and interaction for four to five months or so. So, again, they have to wonder whether they will see a screening tool before mid-fall. Again, they're trying to get information from them on what's their estimate.

Again, as to releasing interim or final revisions to Executive Order 12898, they need a timeline for that. And, once they have a timeline for these activities, it really helps them plan our workload and their meeting schedule. There's no reason for them to work urgently when they won't be ready to even address or consider the information for a while. So, she's been wondering whether they need to scale back some of their meetings. For instance, the screening tool workgroup already meets only once a month. Justice40 has been every two weeks. So, she'd like to have a consensus vote to make a formal request of CEQ for the timelines for those four items. **Vice-Chair Tilousi** reminded Co-Chair Shepard that they wanted to add a budget from CEQ. **Co-Chair Shepard** added that they also asked for the organizational chart. **Chair Moore** also added, what's the time frame for the release of the executive order? **Co-Chair Shepard** asked again for a vote. **DFO Martin** acknowledged that there was a consensus.

Co-Chair Shepard stated that the next item is whether or not they need to reduce the number of workgroup meetings until they get better deadlines. What they're finding is that most workgroups meet twice a month, and perhaps they should meet once a month. Then they always have the flexibility to readjust as needed or if they have a particular deadline. **Dr. Whyte** agreed to default to once a month, and then in special cases, they move to ramp-up meetings. **Co-Chair Shepard** replied that the scorecard group will have to do a little more to get that second phase in. **Dr. Whyte** agreed. **Ms. Santiago** agreed.

Mr. Cormons agree that they're balancing a lot of things here. This is a marathon, not a sprint, so they do have to pace themselves. But they're in a unique window of opportunity that is not necessarily going to last forever, so, he does want to be sure that where they're needed, they're putting in the time. They're going to do what it takes, so he's fine with once a month being the default. But they have to be very ready to go to a greater frequency in periods of need

Co-Chair Shepard suggested that at the chairs and co-chairs meeting or the executive committee meeting they do a schedule maybe from now through April or May so that they can work backward and see where the deadlines are and how they can get there in terms of numbers of meetings. **DFO Martin** agreed. She clarified that the deadline that was set for March for our implementation recommendation was a timeline that was set by the workgroup. They hadn't got any indication from CEQ about a timeframe yet, but that was kind of their timeframe that they set because they wanted to keep things moving.

Mr. Mitchell agreed but stated that they needed to seize this moment and opportunity. After getting it and seeing a lot of the information, this bi-partisan infrastructure bill is already out. Folks are still confused, and he doesn't think now is the time to pump the breaks because they have about a three-month window before mid-terms kick up. There are some things they need to have some clarity on. The President is doing his state of the union in about 35 days, and they're just starting to get clarity on one of his major pillars and promises. For the next two or three months, this is a critical space that they're in right now. Folks are saying marathons and sprints, but as a trackman, you've got to cross that dang finish line one way or the other. Right now, they can see the end, but he cautioned on the time and timeliness of this right now.

Co-Chair Shepard acknowledged that consideration, but she reminded everyone of all the meetings that are scheduled that don't include the workgroups. **Mr. Mitchell** just reminded everyone that there might be a mad scramble in these next couple of months and having the guidance in what they're recommending that folks do for implementation will be required. **Dr. Whyte** announced that the indigenous workgroup will keep the set of meetings on the books every two weeks for now, and the workgroup itself will confer about their decisions. They'll discuss whether they want to just keep it the same or change it.

Ms. Lopez-Nunez stated that she too is a little nervous about the mid-term elections and the political opening might just get shut in November. If that happens, how can we leave as much as possible? She empathized with the chairs with how many meetings they are part of, so questioned how can they shift responsibilities among the members to keep the work moving ahead and not overburden the chairs? This is a substantially large group that they should be then sharing more responsibilities to get as much down on paper before that political opening closes again. **Co-Chair Shepard** suggested that they withdraw the idea of voting on this and that they continue as is. Each workgroup or particular committee will decide on what they need to do, and the timeline will be based on what was put together. **Chair Moore** agreed.

Co-Chair Shepard stated that the WHEJAC wanted to go on record by official letter requesting additional funding for CEQ to have more staff to support the work. Right now, they found out at one of their meetings a couple weeks ago that the CEQ only has 12 staff for the whole organization. And so, realistically they need 12 staff just to implement Justice40, not to mention running the whole organization and the NEPA team and all of the other issues they're working on. So, the WHEJAC really thinks it's important to advocate for additional funding and staffing for CEQ if they're going to advance Justice40 and environmental climate justice. She wanted to put that idea forward on whether the group thinks that they should move forward with writing a letter to the president on this issue.

Mr. Cormons asked would be appropriate to hear from CEQ on that? How that can positively impact the staffing roles. **Co-Chair Shepard** replied that Brenda Mallory confirmed that they have budget issues, and they don't have enough staffing. Everyone knows historically they never have. Yet, they have plopped this amazing enormous issue right in the hands of the smallest office that has no staffing to do it. So, they're either set up to fail or WHEJAC's got to promote more money and more staff because that's only going to help advance everything they're working here for. **Mr. Cormons** said that did seem obvious.

Ms. Santiago clarified that the suggestion that the Council's recommendations to CEQ should wait until the Council knows whether CEQ will think it's a good idea is making recommendations contingent on acceptance, and that's not how it works. The Council makes recommendations, and CEQ may or may not accept them.

Co-Chair Shepard informed everyone that they will see such a letter before it goes out, then she requested to take a vote. **DFO Martin** stated there was a consensus.

Co-Chair Shepard spoke about how the WHEJAC wanted to have a public meeting just focused on the scorecard for our second phase of recommendations so that they can then come back to the whole body for a vote. It'd be great if they can promote getting public comments that are actually on the scorecard, which would help our deliberations. There is a tentative date for that meeting which is February 24th. They are expected to submit final recommendations at the March 30th and 31st public meeting.

Ms. Roberts asked, when the announcement goes out for that, is there some type of way to really home in on the importance of coming to this meeting to participate in that creative process around this scorecard? **DFO Martin** replied that, as far as the request, they can try to strengthen the language a little bit in the federal registry notice and then get the com folks to see if there's something they can put out. **Ms. Roberts** suggested that they include the time period for the public comment in advance because that has been a little challenging with folks who have been seeking to participate as best as possible. **DFO Martin** replied that they do try to get that out there as soon as possible, but the issue they run into is getting the agenda together.

2.4 Closing Remarks - Announcements & Adjourn

Co-Chair Shepard reviewed the action items that were discussed or voted on today. There will be a special scorecard meeting on February 24th. There will be the next public meeting on March 30th and 31st. They will be sending a letter to the CEQ to get the timelines for the key initiatives such as the screening tool release, scorecard release, and all of those executive order revisions. There will be a climate resilience working group. They will get more clarification although many people feel that they have good clarification they just need to refine or condense this scope of work. Lastly, there'll be a letter to the Biden administration on increasing the budget and staffing for CEQ. So those have been the major actions that were talked about during the business meeting.

Chair Moore added that there were recommendations that were made during the public comment. The idea is to pool those public comment pieces together and then pull out the recommendations so that we can move on some of those recommendations. **Co-Chair Shepard** asked if there is a process for prioritizing and who's going to put that together? **Chair Moore** clarified that the recommendations from the public speakers for this meeting need to be moved on. **DFO Martin** replied that they will need a little bit of time, and they have some staff that's going to be out of the office over the next couple of weeks. They can try to have something for you by the next public meeting. **Co-Chair Shepard** asked for any last-minute comments.

Ms. Roberts thanked everyone who stayed online for so long. We are really in some of the critical times and, yes, we've heard that the window is short and small. But we don't want to continue on a path of speaking about history, but we want to be on a path of making history, especially for those people who have been suffering for a very long time. She would be remiss if she did not say she's complained tonight about not having a break but thinking about the many people who haven't had a break in life at all. Be mindful of that and take care of themselves and take care of one another because we are in that small window. But that small window can make a larger window of difference.

The chairs and vice-chairs thanked everyone for their hard work with recommendations on Justice40. They saw some of those recommendations in the implementation plan. They completed their recommendations for the executive order. **Co-Chair Shepard** adjourned the meeting.

[THE MEETING WAS ADJOURNED]

Appendix A. Committee Members in Attendance

- Richard Moore, Co-Chair, Los Jardines Institute
- Peggy Shepard, Co-Chair, WE ACT for Environmental Justice
- Carletta Tilousi, Vice-Chair, Havusapai Tribal Council
- Catherine Coleman Flowers, Vice-Chair, Center for Rural Enterprise and Environmental Justice
- Angelo Logan, Moving Forward Network
- Rachel Morello-Frosch, PhD, UC Berkley
- Viola Waghiyi, Alaska Community Action on Toxins
- Miya Yoshitani, Asian Pacific Environmental Network
- Kim Havey, City of Minneapolis
- Kyle Whyte, PhD, University of Michigan
- Tom Cormons, Appalachian Voices
- LaTricea Adams, Black Millennials for Flint
- Harold Mitchell, ReGenesis
- Beverly Wright, PhD, Deep South Center for Environmental Justice
- Susana Almanza, People Organized in Defense of Earth and Her Resources
- Jade Begay, Climate Justice Campaign Director
- Robert Bullard, PhD, Texas Southern University
- Juan Parras, Texas Environmental Justice Advocacy Services
- Maria Belen-Power, GreenRoots
- Maria Lopez-Nunez, Ironbound Community Corporation
- Michele Roberts, Environmental Justice and Health Alliance for Chemical Policy Reform
- Nicky Sheats, PhD, Kean University
- Ruth Santiago, Latino Climate Action Network
- Hli Xyoog, Advancement of Hmong Americans

Appendix B. Agenda

THE COUNCIL ON ENVIRONMENTAL QUALITY
WHITE HOUSE ENVIRONMENTAL JUSTICE ADVISORY COUNCIL
VIRTUAL PUBLIC MEETING

AGENDA	JANUARY 26, 2022 — 3:00 P.M. – 8:00 P.M. ET
3:00 p.m. - 3:30 p.m.	**WELCOME, INTRODUCTONS & OPENING REMARKS** ○ **Karen L. Martin, *Designated Federal Officer*** – *U.S. Environmental Protection Agency* ○ **Richard Moore, *White House Environmental Justice Council Co-Chair*** – *Los Jardines Institute* ○ **Catherine Coleman Flowers, *White House Environmental Justice Council Vice Chair*** – *Center for Rural Enterprise and Environmental Justice* ○ **Carletta Tilousi, *White House Environmental Justice Council Vice Chair*** – *Havasupai Tribal Council* ○ **Brenda Mallory, Chair** – *The Council on Environmental Quality*
3:15 p.m. - 3:30 p.m.	**WELCOME & OPENING REMARKS** ○ **Brenda Mallory, Chair** – *The Council on Environmental Quality*
3:30 p.m. - 4:30 p.m.	**U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES PRESENTATION & DISCUSSION** ○ **Admiral Rachel L. Levine, *Assistant Secretary for Health*** – *U.S. Department of Health and Human Services* ○ **Arsenio Mataka, *Senior Advisor for Health Equity and Climate*** – *U.S. Department of Health and Human Services*
4:30 p.m. - 5:30 p.m.	**CLIMATE RESILIENCE DRAFT CHARGE PRESENTATION & DISCUSSION** ○ **Mark Chambers, *Senior Director for Building Emissions & Community Resilience*** – *Council on Environmental Quality, Executive Office of the President* ○ **Krystal Laymon, *Deputy Director for Climate Resilience*** – *Council on Environmental Quality, Executive Office of the President*

AGENDA	JANUARY 26, 2022 — 3:00 P.M. – 8:00 P.M. ET
5:30 p.m. – 5:45 p.m.	**BREAK**
5:45 p.m. – 7:45 p.m.	**PUBLIC COMMENT PERIOD** ***Members of the public will be given three (3) minutes to present comments on their issue or concern to the WHEJAC.***
7:45 p.m. - 8:00 p.m.	**CLOSING REMARKS – ANNOUCEMENTS & ADJOURN** ○ **Richard Moore, *White House Environmental Justice Council Co-Chair*** – *Los Jardines Institute* ○ **Karen L. Martin, Designated Federal Officer** – U.S. Environmental Protection Agency

AGENDA	JANUARY 27, 2022	3:00 P.M. – 8:00 P.M. ET
3:00 p.m. - 3:30 p.m.	**WELCOME, INTRODUCTONS & RECAP** ○ **Karen L. Martin, *Designated Federal Officer*** – *U.S. Environmental Protection Agency* ○ **Richard Moore, *White House Environmental Justice Council Co-Chair*** – *Los Jardines Institute* ○ **Peggy Shepard, *White House Environmental Justice Council Co-Chair*** – *WE ACT for Environmental Justice* ○ **Catherine Coleman Flowers, *White House Environmental Justice Council Vice Chair*** – *Center for Rural Enterprise and Environmental Justice* ○ **Carletta Tilousi, *White House Environmental Justice Council Vice Chair*** – *Havasupai Tribal Council*	
3:30 p.m. – 4:45 p.m.	**WHEJAC SCORECARD WORKGROUP UPDATE & DISCUSSION** ○ **Dr. Kyle Whyte, Scorecard Workgroup Co-Chair** – *University of Michigan* ○ **Maria López-Núñez, Scorecard Workgroup Member** – *Ironbound Community Corporation* ○ **Dr. Beverly Wright, Scorecard Workgroup Member** – *Deep South Center for Environmental Justice*	
4:45 p.m. – 6:00 p.m.	**JUSTICE40 WORKGROUP PANEL DISCUSSION** ○ **Dr. Candance Vahlsing, Associate Director of Climate, Energy, Environment, and Science** – *Office of Management of Budget* ○ **Jahi Wise, Senior Advisor for Climate Policy and Finance** – *Climate Policy Office* ○ **Dr. Phillip Fine, Principal Deputy Associate Administrator for Policy** – *U.S. Environmental Protection Agency* ○ **Dr. Gbenga Ajilore, Senior Advisor, Office of Undersecretary for Rural Development** – *U.S. Department of Agriculture* ○ **Dr. Tony Reames, Senior Advisor on Energy Justice** – *U.S. Department of Energy* ○ **Corey Solow, Deputy Director for Environmental Justice** – *The Council on Environmental Quality*	
6:00 p.m. – 6:15 p.m.	**BREAK**	

AGENDA	JANUARY 27, 2022	3:00 P.M. – 8:00 P.M. ET
6:15 p.m. – 7:45 p.m.	**WHEJAC BUSINESS MEETING REFLECTION & CONVERSATION** **The WHEJAC will use this time to reflect on the meeting proceedings and public comment period; provide workgroup updates; discuss action items and finalize next steps.** ○ **Karen L. Martin, Designated Federal Officer** – U.S. Environmental Protection Agency ○ **Richard Moore, *White House Environmental Justice Council Co-Chair*** – *Los Jardines Institute* ○ **Peggy Shepard, *White House Environmental Justice Council Co-Chair*** – *WE ACT for Environmental Justice* ○ **Catherine Coleman Flowers, *White House Environmental Justice Council Vice Chair*** – *Center for Rural Enterprise and Environmental Justice* ○ **Carletta Tilousi, *White House Environmental Justice Council Vice Chair*** – *Havasupai Tribal Council*	
7:45 p.m. – 8:00 p.m.	**CLOSING REMARKS - ANNOUCEMENTS & ADJOURN** ○ **Richard Moore, *White House Environmental Justice Council Co-Chair*** – *Los Jardines Institute* ○ **Peggy Shepard, *White House Environmental Justice Council Co-Chair*** – *WE ACT for Environmental Justice* ○ **Karen L. Martin, Designated Federal Officer** – U.S. Environmental Protection Agency	

Appendix C. Meeting Attendees

FIRST NAME	LAST NAME	ORGANIZATION
Judy	Abbott	NYS Department of Health
Gerardo	Acosta	Office of Communities, Tribes and Environmental Assessment
Francisca	Acuna	GoAustin/VamosAustin
Astrika	Adams	SBA OA
Jamiah	Adams	The Climate Reality Project
Sara	Adelsberg	Deloitte
Maritza	Adonis	MTA Visions Global CSR & Gov't Relations
Amanda	Aguirre	Rooted & Reimagined Strategies
Saran	Ahluwalia	USDS
Lubna	Ahmed	CDPHE
David	Ailor	American Coke and Coal Chemicals Institute -
Kristin	Aldred	Stericycle
Nicole	Alindogan	University of Pittsburgh
Lylianna	Allala	City of Seattle
Dee	Allen	T.E.J.A.S.
Elizabeth	Allen	Private Citizen
Stacy	Allen	Private Citizen
Roxanne	Almodovar	Private Citizen
Dean	Alonistiotis	MWRF
Charles	Alsdorf	Deloitte
Michael	Altman	USDA
Julia	Alvey	Georgetown University
Susan	Alzner	shift7
Aita	Amaize	OurSpace World, Inc.
Alex	Ambrose	ANJEC
Qay-liwh	Ammon	House Natural Resources
Valerie	Amor	Drawing Conclusions LLC
Rhonda	Anderson	Sierra Club
Liz	Anderson	Dakota Resource Council
Jeffrey	Andrilenas	The TBLS Group
Carl	Anthony	Breakthrough Communities
Devin	Araujo	University of Kansas
Reginald	Archer	Tennessee State University
Karol	Archer	FAA
Deyadira	Arellano	The People's Collective for Environmental Justice
Maria	Arevalo	US EPA
Russell	Armstrong	Oxfam America
Dave	Arndt	Private Citizen

FIRST NAME	LAST NAME	ORGANIZATION
Duffy-Marie	Arnoult	The Climate Reality Project
Maya	Aronoff	Private Citizen
Jessica	Arriens	National Wildlife Federation
Alexzandria	ashton	Ashton Wolfe Sustainability Consulting
Ann	Augustyn	US Department of Energy
Mashal	Awais	Bayou City Waterkeeper
Alycia	Bacon	Mothers Out Front
Cimbria	Badenhausen	First Circle
Sarah	Bailey	Bridges Into the Future/ Community Based Organizations Partners/ Flint Public Health Youth Academy
Taaka	Bailey	MDEQ
Treana	Baker	Michael Baker Intl
Sarah	Baker	US EPA
Nyoka	Baker	Private Citizen
Kim	Balassiano	USEPA
John	Balubs	HHS-OASH
Scott	Banbury	Tennessee Chapter of the Sierra Club
Jamie	Banks	Quiet Communities Inc
April	Baptiste	Colgate University
Lakendra	Barajas	Earthjustice
Edlynzia	Barnes	EPA
Chelsea	Barnes	Appalachian Voices
Xavier	Barraza	Los Jardines Institute / F Valle de Oro NWR
Rosario	Barrera	University of Illinois at Urbana-Champaign
Catharine	Bartone	VTDEC
Susan	Bartscherer	Private Citizen
Timothy	Barzyk	US EPA
Ta Misha	Bascombe	HHS
Hailey	Basiouny	Private Citizen
Nikki	Bass	USEPA
Sean	Bath	NOAA Climate Program Office
Stacia	Bax	Missouri Department of Natural Resources
John	Beard	Port Arthur Community Action Network (PACAN)
Sharon	Beard	NIEHS Worker Training Program
Karen	Bearden	350 Triangle
Sheeah	Bearfoot	Anthropocene Alliance
Nancy	Beck	HuntonAK
Samantha	Beers	us epa
Cory	Behroozi	The New School
Shannon	Bell	Virginia Tech
Linda	Belton	NOAA
Felicia	Beltran	Arizona Department of Transportation

FIRST NAME	LAST NAME	ORGANIZATION
Mitchell	Berge	Private Citizen
Carol	Bergquist	Hannahville Indian Community
Peggy	Berry	Thrive_At_Life: Working Solutions
Jessica	Bielecki	NRC
Trina	Bilal	Department of Energy
Stephanie	Bilenko	Nuclear Energy Information Service (NEIS)
Pamela	Bingham	Private Citizen
Hailee	Birdtail	University of Kansas
Michael	Blair	Innovate Inc
DeShawn	Blanding	Rural Coalition
Julie	Bledsoe	Kingston Coal Ash Worker's Wife
Juliet	Bochicchio	FTA
John	Bolender	TBG, llc
Darlene	Bolsover	North Tonawanda Resident
Lynne	Bonnett	Anthropocene alliance and 10000 hawks
Patty	Bowen	Conservation Voters for Idaho
Wesley	Boykin	Duplin County (NC) NAACP
Jennifer	Boyle	Department of Environmental Quality
Sims,	Brad	Exxon Mobil Corporation
Elizabeth	Bradford	Michael Baker International
John	Brakeall	Pennsylvania Dept. of Environmental Protection
Wayne	Branch	Duplin County Commissioner
Anna	Brandes	Private Citizen
David	Brewster	PARS Environmental
Evelyn	Britton	U.S. General Services Administration
Emily	Brooks	USGS
Erin	Broussard	AEPCO
Doris	Brown	West Street Recovery/ Northeast Action Collective
Austin	Brown	The White House
Carlee	Brown	House of Representatives
Alix	Brown	into the storm
Janice	Brown	Private Citizen
Kalina	Browne	Ocean Conservancy
Morgan	Browning	US EPA
Kelsey	Brugger	E&E News
James	Brunswick	Department of Natural resources and Environmental Control
Kyle	Bryant	R4 EPA/ORA/SPO/EJCHS
Charles	Bryson	Civil Rights Enforcement Agency
Marcus	Buchanan	CDC/ATSDR
Stephen	Buckley	OpenGovMetrics.com
Ashley	Bullock	Public Health - Seattle & King County

FIRST NAME	LAST NAME	ORGANIZATION
Kylie	Burkett	USACE
Diane	Burman	NY PSC
Jeremy	Burnworth	ReviveUP
Shondricka	Burrell	Duquesne University
Bryan	Burton	ALA
Lisa	Burton	Board of Legislators
ADRIANE	BUSBY	Friends of the Earth
Debra	Butler	University of Massachusetts Boston
Michael	Buza	Sierra Club: Nepessing Group
Elyse	Caiazzo	University of Montana
Martha	Camacho Rodriguez	SEE-LA (Social Eco Education-LA)
Karen	Campblin	ktcPLAN
Gabriela	Campos	anthropocene alliance
Darshan	Campos	Reparations University: Somos Semillas Antillanas / Fundación Yamoca Opia
Morgan	Capilla	US EPA
Steven	Carbó	Funder Collaborative on Oil and Gas
Jacqueline	Cardoza	Private citizen
Kristie	Carpenito	K2 Communications LLC
Estefany	Carrasco	Chispa LCV
Maryann	Carroll	Croda
Olivia	Carros	Washington State Department of Ecology
Craig	Casper	Corpus Christi MPO
Alison	Cassady	U.S. EPA
Nancy	Castaldo	Private Citizen
Elida	Castillo	Chispa TX
Mary	Casto	WV Community Development Hub
Katherine	Catalano	American Public Health Association
Crystal	CAVALIER	7 Directions of Service
Brian	Chalfant	Pennsylvania Department of Environmental Protection
Angela	Chalk	Healthy Community Services
Elizabeth	Chan	EPA
Kartik	Chandran	Columbia University
Lelina	Chang	Deloitte
Hoshing	Chang	FDA
Sabrina	Chapa	Anthropocene Alliance
Fatiha	Chaudhry	Deloitte
Donna	Chavis	Friends of the Earth
Jean	Chemnick	E&ENews
Annie	Chen	OEHHA
Christina	Chiappetta	GSA

FIRST NAME	LAST NAME	ORGANIZATION
Bernadette	Chiasson	FEMA
Cassidy	Childs	CEQ
Russell	Chisholm	Protect Our Water, Heritage, Rights (POWHR)
Glo	Chitwood	Alaska Just Transition Collective
Lyla	Choi	Zero Hour
Iqra	Choudhry	Environmental Justice Class at Georgetown University
Amanda	Chu	Financial Times
Anna	Chua	The Sierra Club of Hawaiʻi
Zach	Church	U.S. EPA
Glennette	Clark	USDS
Jenn	Clarke	City of Richmond
Nakiya	Clausell	Deloitte
Rachel	Cleetus	Union of Concerned Scientists
Sterling	Clemmons	Deloitte
Shelby	Cline	Clean Water for North Carolina
Kristin	Coats	CMS
Majidah	Cochran	Beveridge & Diamond, P.C.
Deborah	Cohen	USEPA
Elizabeth	Cole	MT DEQ
Christine	Colley	NYSDOT
Emily	Collins	Fair Shake Environmental Legal Services
Bryanna	Conde	University of Kansas
Andrea	Contreras	ORISE EPA
Hayley	Cook	City and County of Honolulu Office of Climate Change, Sustainability and Resiliency
Lyndsey	Cooper	NYS DEC
David	Copper	Reclaim Augusta-- member. Treasurer
Tina	Cordova	Tularosa Basin Downwinders Consortium (TBDC)
Regina	Cornwell	Sierra Club
Drew	Costley	The Associated Press
Farrah	Court	Texas Commission on Environmental Quality
James	Courtney	Arnold & Porter, LLP
Kitty	Craig	the wilderness society
Bria	Crawford	Environmental Protection Agency
Brandi	Johnson	EJ Activist
Kat	Croft	Private Citizen
Heather	Croshaw	St. Croix Environmental Association (St. Croix, USVI)
Leticia	Cruz	Vintage Nurse Groups Research and Consulting Group
Kathryn	Cruz	The Center for Environmental Transformation
Jonathan	Cruz	Multnomah County Environmental Health
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		Development
Sharyn	Cunningham	Lincoln Park Superfund Site CAG - Colorado
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Jessica	Dandridge	Water Collaborative of Greater New Orleans
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Bryan	Davidson	Tennessee Department of Environment and Conservation
Tina	Davis	US EPA
Zachary	Davis	University of Kansas
Cemelli	de Aztlan	La Mujer Obrera
Kathleen	Deener	US EPA
Jesse	Deer In Water	Citizens Resistance At Fermi Two
Chris	DeFreese	USDA Rural Development
Miriam	DeJong	Matrix New World Engineering
Mariana	Cervantes	GreenLatinos
Jamie	DeMarco	Chesapeake Climate Action Network
ERICA	DENHAM	Transcription ETC
LaTonya	Derrick	Stantec Consulting
Zane	DeVitre	Private Citizen
Christine	Diaz	Clean Water for NC
Carolyn	Dick Mayes	U.S. EPA
Russell	Dickerson	UMD
Mariah	Dignan	Climate Jobs NY
Marcia	Dinkins	Black Appalachian Coalition
Robert	Dinterman	USDA
Warner	Dixon	Committee on Oversight and Reform
Eric	Dixon	Ohio River Valley Institute
Margaret	Doman	jsqA
Simone	Domingue	University of Oklahoma
Ali	Dominguez	Deloitte
Patricia	Donohue	Government
Cece	Donovan	EcoLogix Group, Inc.
Sarah	Doran	Corning Inc.
Grace	Dorton	USDA
Jesse	Dowe	Duplin County Government
Lori	Dowil	Corteva
Melinda	Downing	Department of Energy
JOHN	DOYLE	little big horn college
Jacquelyn	Drechsler	Private Citizen
Sydney	Drinkwater	University of Kansas

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Maddie	Duda	Lotus Water
Justin	Dula	PA Dept. of Enviro. Protection
Aaron	Dunbar	Mid-Ohio Valley Climate Action
Alexandra	Dunn	Baker Botts
Carol	Dunn	Colorado Citizens Against Toxic Waste
Alex	Easdale	Southeast Climate & Energy Network
Jacqueline	Echols	South River Watershed Alliance
Nathalie	Eddy	CDPHE - EJ Unit
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Tania	Ellersick	USDA Forest Service
Adrienne	Elliott	Scripps Institution of Oceanography
Ryan	Ellis	Interstate Mining Compact Commission
Salma	Elmallah	Lawrence Berkeley National Lab
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Lena	Epps-Price	US EPA
C. Sequoia	Erasmus	Ctc
Serap	Erdal	University of Illinois-Chicago School of Public Health
Diloola	Erickson	Native Movement
Demi	Espinoza	Erol Foundation
Dennis	Evans	Urban Rain Gardens
Mike	Ewall	Energy Justice Network
Robert	Fanuzzi	Bronx Council for Environmental Quality
June	Farmer	Marin City People’s Plan
Ericka	Farrell	EPA
Mary	Fasano	EPA
Mohsen	Fatemi	University of Kansas Dept of Public Administration - Lawrence, KS
Darryl	Fears	The Washington Post
Rick	Felan	NASA
Adriana	Felix-Salgado	US EPA
Cynthia	Ferguson	US Dept. of Justice, Environment & Natural Resources Division
Todd	Fernandez	Climate Crisis Policy
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Derrick	Figures	sierra club
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Noemi	Florea	The New School
Monaeka	Flores	Prutehi Litekyan:Save Riditian
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Ernie	Francestine	City of Pittsburgh
Sasha	Francis	Galveston Bay Foundation
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Lisa	Frankland	WIPP
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Geri	Freedman	The Peoples Justice Council
Lena	Freij	U.S. EPA - Region 10
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Lisa	Friedman	New York Times
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Genevieve	Gadsden	EPA
Katherine	Gajewski	City Scale
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Sarah	Gallagher	Upper Green Side
Kathryn	Gallicchio	10, 000 Hawks / Anthropocene Alliance
Briana	Garcia	temboo
Frida	Garcia	UTEP
Tracy	Garland	Resilient Virginia
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Venu	Ghanta	Duke Energy

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Matthew	Goetz	Georgetown Climate Center
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Julian	Gonzalez	Earthjustice
Breeana	Gonzalez	Environmental Policy Innovation Center
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Running	Grass	Three Circles Center
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Kelsey	Green	Pace University
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Marcus	Griswold	Calm Waters Group
Alex	Guillen	Politico
Sheila Baker	Gujral	Association of NJ Environmental Commissions
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Marcia	Hale	UNCG

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Michael	Hansen	GASP
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Holly	Henderson	Duke Energy
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Ginny	Hildebrand	Pittsburgh Green New Deal
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Kimberly	Hill	Private Citizen
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Katie	Hoeberling	Open Environmental Data Project
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Brian	Holtzclaw	EPA

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Candis	Hunter	CDC
Lib	Hutchby	WILPF-Triangle
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Lakota	Ironboy	Leech Lake Division of Resource Management
Faith	Iseguede	Jackson State University
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Shannon	Jewell	EPA
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Lauren	Johnson	George Washington University
Arthur	Johnson	CSED
Maury	Johnson	Preserve Monroe
Sabrina	Johnson	US EPA
Bonita	Johnson	USEPA
Dans	Johnson	We act for Environmental Justice
Doris	Johnson	DEEP
Michael	Johnson	NDN Collective, Inc.
Ayanna	Jolivet	Bayou City Waterkeeper

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Matthew	Jurjonas	USGS
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Jennifer	Kanine	Pokagon Band of Potawatomi Indians
Erin	Kanzig	River Network
Amanda	Kasper	Ervin Graves Strategy Group
Kira	Kaufmann	USFS
Amy	Kelly	Sierra Club
Cheryl	Kelly	DOI
Martha	Kelly	CT Zero Waste Coalition, Connecticut Coalition for Environmental Justice, GPUS EcoAction Committee
Kevin	Kephart	NIFA
Kameron	Kerger	U.S. Digital Service
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Chet	Kibble Sr	Memphis & Shelby County Lead Safe Collaborative Inc.
Joanne	Kilgour	Ohio River Valley Institute
Isabella	Killius	Tennessee Valley Energy Democracy Movement
Suzanne	Kim	Motivate Capital
Toshia	King	EPA/OLEM/ORCR
Marva	King	EPA retiree
Michelle	King	Louisville Metro Air Pollution Control District
Monika	King	New York State Department of Health
John	Kinsman	Edison Electric Institute
Clara	Kitongo	Tree Pittsburgh
Sarah	Kleypas	Lone Star Legal Aid
Elizabeth	Kloeckner	Private Citizen
Amy	Klusmeier	US DOE
Kristien	Knapp	House Committee on Oversight and Reform
Jeffrey	Knishkowy	U.S. Dept. of Agriculture
Bri	Knisley	Appalachian Voices
Megan	Kohler	ADEC
Jeannette	Kravitz	sponsorKIDS Charities 501c3
Fran	Kremer	USEPA
Marnie	Kremer	U.S. House of Representatives
Gretchen	Kroh	USDA
Keith	Krosinsky	Association of American Medical Colleges
JOEL	KUPFERMAN	eji nlg
Emma	Kurnat-Thoma	Georgetown University NHS

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Lynelle	Ladd	Kansas State University Pollution Prevention Institute
Osprey	Lake	Women's Earth and Climate Action Network
Clement	Lam	Gerdau
Yukyan	Lam	NRDC
Kim	Lambert	U.S. Fish and Wildlife Service
Katie	Lambeth	EGLE
Meghan	Langley	City Point Partners, LLC
Chad	Larsen	Environmental Protection Agency
Sarah	Lashley	Augustana College
Erica	Le Doux	U.S. EPA - Region 6
Charles	Lee	US Environmental Protection Agency
Paul	Lee	Los Angeles Mayor's Office
Kristin	Lehman	FEMA
Sharon	Lewis	CT COALITION FOR ENVIRONMENTAL JJSTICE
Stevie	Lewis Klass	Public Lab
Gloria	Li	CEQ
John	Liantonio	Port Authority of NY&NJ
Janesa	Licorish	HHS
Peter	Liebowitz	WSP USA Inc
Susan	Liley	Citizens' Committee for Flood Relief
Christopher	Lindsay	IAPMO
Kyle	Lisek	South Shore Clean Cities
C.	Liv	hhs
Sarah	Lobe	Nixon Peabody LLP
Natalie	Longhurst	University of Kansas
Ashley	Longrie	Environmental Protection Agency
David	Lonnberg	shift7
Luis	Lopez	Comite Civico del Valle
Olivia	Lopez	Ocean Conservancy
Bonnie	Lottes	NYSDOT
Jessica	Lovering	Good Energy Collective
Gloria J	Lowe	We Want Green Too
Tianyi	Lu	fordham law school
Yi	Lu	HEI
James	Ludington	IHS
Nikki	Luke	University of Tennessee
Tai	Lung	US EPA
Maureen	Lynch	Wallace precinct/Democratic Party
Susan	Lyons	Groundswell
Latasha	Lyte	USDA

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Akilah	M	USACE
Benjamin	Machlis	Dorsey & Whitney
Caitlin	Macomber	World Resources Institute
Juan	Madrid	Green Latinos
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Mark	Magaña	GreenLatinos
Ed	Maguire	WV Department of Environmental Protection
Lynda	Majors	Preserve Montgomery County VA
Olufunmilayo	Makinde	AAMC
Rachel	Makleff	Grow Renewable New York: No Canadian Hydro
Michael	Malcom	The People's Justice Council
Juan B.	Mancias	Carrizo Comecrudo Tribe of Texas
Christa	Mancias	Carrizo Comecrudo Tribe of Texas
Ellen	Manges	US EPA
Harry	Manin	House Oversight Committee
Denise	Manole	NJ Watershed Ambassador
Jane	Mantey	Ceres
Ellen	Mantus	Health Effects Institute
Olivia	Marine	Livingston Energy Group
Cecilia	Marrtinez	Private Citizen
Janette	Marsh	US EPA Region 5
Carolyn	Marsh	Save Whiting And Neighbors (S.W.A.N.)
Jennifer	Marshall	Florida Department of Transportation
Aurora	Martin	Front and Centered
Akilah	Martin	USACE
Anna	Martin	House Natural Resources Committee
Vincent	Martin	V Martin Environmental Justice LLC
Astrid	Martínez	LCV
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Eileen	Mayer	US EPA
Bud	McAllister	Partners in Healthy Communities
Eugene	McCall	McCall Environmental, PA
Mary	McCarron	Ohio EPA
Christine	McCarthy	Cherokee Nation Fed (Contractor for HQ Dept of Army)
Justin	McCarthy	Congressional Progressive Caucus Center
Tamara	McConnell	Iowa State University
Larissa	McDonogh	EOP
Jacy	McGaw	Private Citizen
Mary	McGuckin	Parent, Grandparent, Citizen, Woman,
Caitlin	McHale	National Mining Association
Shannon	McNeeley	Pacific Institute

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Sandra	Meier	Environmental Energy Alliance of New York
Chris	Melton	Gerdau
Johnny	Mendoza	USDA NRCS
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Danielle	Mercurio	VNF
Sarah	Merrill	SAVERGV
Melissa	Mertz	GSA
Annette	Metzger	AM Notary
C	Meyer	State of Minnesota
Madeline	Middlebrooks	Great Rivers Environmental Law Center
Maria	Mikitka	Department of Energy
Sara	Miller	U.S. EPA
Courtney	Miller	House of Representatives
Pamela	Miller	Alaska Community Action on Toxics
Vernice	Miller	Metropolitan Group
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Aaron	Mintzes	Earthworks
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Katherine	Mlika	U.S. Digital Service
Katie	Modic	Communitopia
Alessandro	Molina	EPA
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Heriberto	Mondragon	Emergency Management Institution
Mia	Montoya Hammersley	New Mexico Environmental Law Center
Chris	Moore	Eastman Chemical Co. - Kingsport, TN
Leslie	Moore	Activist, community organizer, Advocate, Research analysis
Ernest	Mooring	Community Rep
Lucas	Moreau	Hannahville Indian Community
Olivia	Morgan	LSU
Leona	Morgan	Diné No Nukes
Mary	Morrisey	Private Citizen
Leah	Mueller	USACE ERDC
John	Mueller	Supporter of Fluoride Action Network
Omar	Muhammad	Lowcountry Alliance for Model Communities (LAMC)
Matthew	Muirragui	U.S. House of Representatives
Kathryn	Muncil	Fort William Henry Corp
Ilene	Munk	EPA
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Olga	Naidenko	Environmental Working Group
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Anahi	Naranjo	Center for Earth, Energy and Democracy
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Rachael	Nealer	CEQ
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Colleen	Neely	EPA
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Daniel	Nierenberg	NYSDOT
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Elder	Norris	WomEnv Climate Social Justice Marginalized Community Collaborative
Leanne	Nurse	The Nature Conservancy
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Veronica	Oakler	Clean Water for North Carolina
Lauren	O'Brien	Pay Up Climate Polluters
Eddie	Ocampo	Self-Help Enterprises
Tim	O'Connell	USDA Rural Development
Erin	O'Connor	The Washington Post
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Laura	Olah	Citizens for Safe Water Around Badger (CSWAB)
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JOHN	OLUWALEYE	Gender Based Violence as a Public Health Issue
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Annette	Osso	Resilient Virginia
Sheryl	Ott	HLS
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Lesley	Pacey	Eastern Shore Community Health Partners

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Gianna	Parisi	White House Council on Environmental Quality
Jennifer	Park	epa
Grace	Parker	University of Kansas
Ana	Parras	Texas Environmental Justice Advocacy Services
Jimmy	Parrish	DLA
Seton	Parsons	SBA
Mackenzie	Parsons	Georgetown University
Linda	Parsons	Private Citizen
Mackenzie	Parsons	Georgetown University
Heather	Passchier	National Park Service
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Jacqueline	Patterson	The Chisholm Legacy Project
Regan	Patterson	Congressional Black Caucus Foundation
Allison	Patton	Health Effects Institute
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Luciana	Paz	USDA
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Sabina	Perez	Office of Senator Perez
William	Perkison	UT School of Public Health
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Victoria	Persky	University of Illinois at Chicago
Katherine	Peterson	earthworks
Michael	Petroni	SUNY College of Environmental Science and Forestry
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Emily	Phillips	Georgia EPD
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Barbara	Polivka	University of Kansas Medical Center School of Nursing

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Katherine	Quaid	WECAN
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Karla	Raimundi	VT Agency of Natural Resources
Stephanie	Rambo	Tejon Indian Tribe
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Graton	Rancheria	FIGR
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John	Reed	Private Citizen
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Dawn	Reeves	Inside EPA
Oshana	Reich	Private Citizen
Diane	Reilly	TRC
Amber	Reimondo	Grand Canyon Trust
Michael	Reiner	Building Technologies Office, DOE
Forest	Replogle	Mid-Region Council of Governments
Cynthia	Rezentes	Mohala I Ka Wai
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Pria	Rhodes	Climate Justice Alliance/ United Frontline Table
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Jennifer	Richmond	North Carolina State University
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Pettit,	Riley	Deloitte
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Guillermo	Saar	Stateworkers
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Virginia	Sanders	National Sierra Club
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Jamie	Satterfield	Investigative Journalist
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Stephanie	Schlea	Association of State Drinking Water Administrators
Frances	Schlosstein	The TBLS Group, LLC
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Jonathan	Scott	Waste Analytics LLC

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Alicia	Scott	Partnership for Southern Equity
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Quentin	Scott	Chesapeake Climate Action Network
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Michael	Seavey	CGI Federal
Matthew	Sehrsweeney	USDA
Kim	Seigfreid	Private Citizen
Monisha	Shah	NREL
Booker	Shalom	USDA APHIS
Britney	Shaw	Georgetown University
Irv	Sheffey	Sierra Club
Rebecca	Shelton	Appalachian Citizens' Law Center (ACLC)
Natalie	Shepp	Pima County Department of Environmental Quality
Tracy	Sheppard	US EPA
Scott	Sherman	U.S. Army
John	Sherrill	Illinois Department of Transportation
Lindsay	Shigetomi	Private Citizen
Gina	Shirey	Alaska Department of Environmental Conservation
Jacqueline	Shirley	NEJAC and RCAC
Robert	Shobe	Justice for Beniteau
Maggie	Shober	Southern Alliance for Clean Energy
Nick	Short	University of Kansas
Drew	Shumate	Inmar
Matthew	Sidler	OMB
Sarah	Sieloff	Maul Foster and Alongi, LLC
Jorja	Siemons	Medill News Service
Lauro	Silva	South Valley Partners for Environmental Justice 9
Martin	Silva	Private Citizen
Nicole	Simmons	Appian Corporation
Lori	Simmons	PRPC
Georgia	Simpson	HHS OASH R1
Brad	Sims	Exxon Mobil Corporation
Jessica	Sims	Appalachian Voices
Carly	Sincavitch	Arnold and Porter
Diane	Sinkowski	USDA
Marie	Skaf	Deloitte
Robert	Skoglund	Covestro LLC
Catherine	Skopic	Sierra Club
Melissa	Smarr	National Institute of Environmental Health Sciences

FIRST NAME	LAST NAME	ORGANIZATION
Hannah	Smay	NIRS
Gibbs	Smith	USDA - APHIS
Bernice	Smith	EPA
Megan	Smith	shift7
Steven	Smith	Sweetwater Cultural Center
Tina	Smusz	PreserveMontgomeryCountyVA
Julie	Snow	Army
Rosemary	Sokas	Georgetown University
Jennifer	Sokolove	Water Foundation
Karyn	Sonu	BTB Coalition
JULIE	SPAGNOLI	JM SPECIALTY CONSULTING
Mikayla	Spencer	Ocean Conservancy
Chelsea	Spier	Ca depatment of water resources
Patricia A.	Spitzley	RACER Trust
Cheryl	Spivey	Private Citizen
Anna	Spurlock	Lawrence Berkeley National Laboratory
Gianna	St.Julien	Tulane Environmental Law Clinic
Doreen	Stabinsky	College of the Atlantic
Alexis	Stabulas	EPA
Lynn	Stan	Private Citizen
Joanna	Stancil	USDA-FS
Erin	Stanforth	Mecklenburg County
Brenda	Staudenmaier	Clean Water Action Council of NE WI
Kirsten	Stephenson	University of Kansas
Bill	Stevens	USEPA
Simone	Stewart	National Wildlife Federation
Alexis	Stocks	University of Kansas
SHERYL	STOHS	US EPA
Harley	Stokes	BlueGreen Alliance
Linda	Storm	U.S. Environmental Protection Agency
Brynne	Storsved	US EPA
Terri	Straka	A2/Rosewood Strong Community
Amanda	Strawderman	Clean Water for NC
Tracee	Strum-Gilliam	PRR
Melissa	Sturdivant	USDA Rural Development
White,	Summer	Deloitte
Principal	Sung	Greenbank Associates
Kathryn	Super	ejha
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Marc	Swartz	Navy
Bonnie	Swinford	Sierra Club

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Carlos	Swonke	HNTB
Alicia	Sylvester	Dept of Defense
Frank	Sylvester	US Environmental Protection Agency
Tatanya	Szeliga	SAS
Ean	Tafoya	GreenLatinos
Sandra	Talley	NRC
Reginald	Taylor	Department of Health and Human Services
Chandra	Taylor	Southern Environmental Law Center
Patricia	Taylor	Environment and Human Health, Inc. (EHHI)
Chandra	Taylor	SELC
Amy	Teague	USGS
Debra	Tellez	U.S. EPA
Andrew	Ten Eyck	American Petroleum Institute
Andrea	Thi	DOJ
Dragana	Thibault	Livingston Energy Group, LLC
Amy	Thomaides	City of San Marcos
Laura	Thomas	City of Largo
Nadia	Thomas	Yavapai-Prescott Indian Tribe
Tami	Thomas	EPA
Suzanne	Thornsbury	USDA
Jenny	Tompkins	PennFuture
Heather	Toney	Environmental Defense FunD
ROD	TONEYE	IMPASSE 180
Mike	Tony	Charleston Gazette-Mail
Mark	Toohey	Volunteers for Environmental Health and Justice
Gabriela	Torrano	Private Citizen
Christian	Torres	Comite Civico del Valle, Inc.
Kristina	Torres	US EPA
Jackie	Toth	Good Energy Collective
Kaitlin	Toyama	US DOJ, Civil Rights Division
Emily	Tracy	Cotter Mill/Lincoln Park Superfund site
Kristi	Trail	Pontchartrain Conservancy
Kathy	Triantafillou	EPA
Jenn	Tribble	TDEC
Erika	Trinidad	California Air Resources Board
Devon	Trotter	Institute for Sustainable Communities
Eleni	Tsigas	APHIS/USDA
Adam	Tulu	USDA
Sarah	Turner	Private Citizen
Samantha	Twining	Save Edgmont
Nancy	Ulrich	League of Women Voters-Morgantown-Monongalia County,

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Diana	Umpierre	Private Citizen
Molly	Updegrove	ReImagine Appalachia
Kayla	Vackner	Michael Baker International
Jennifer	Valenstein	Brightwater Strategies
Sandra	Valenzuela	City of San Marcos
Brenda	Vallee	Central LA Coalition for a Clean & Healthy Environment
Virginia	Vassalotti	EPA Region 3
Gloria	Vaughn	EPA
Lindsey	Veas	GSA
Lorena	Venegas	10,000 Hawks; Yale Citizen Climate; Moms United in East Haven-Mamas Unidas en East Haven
Drew	Veysey	Georgetown Climate Center
Clifford	Villa	University of New Mexico
Cristina	Villa	Department of the Interior
Leslie	Vishwanath	National Grid
Peter	Wagenius	Sierra Club - North Star Chapter (MN)
Jasmine	Walker	The Ohio State University
Debra	Walker	Sunnyside Community Redevelopment Org.
Mary	Walker	Gulf Coast Ecosystem Restoration Council
Carla	Walker	World Resources Institute
Dana	Walker	nibr
Pearl	Walker	SACE
Tiffany	Wallace	USDA/FPAC
Stephen	Walls	FERC
Michelle	Walper	Heritage-Crystal Clean, LLC
Brett	Walton	Circle of Blue
Michelle	Ward	Private Citizen
Ryan	Ward	KU
Kearni	Warren	Energy Justice Network
Nate	Warszawski	Private citizen
NaTisha	Washington	One PA
Phillip	Washington	USDA
Michon	Washington	FAA
Cheryl	Watson	Blcks In Green
Kayla	Weiser-Burton	Dorsey & Whitney LLP
Nicole	Weymouth	WSP
Aalaya	Wheeler	Marin City Climate Resilience and Health Justice
Herbert	White	Occtox
Chris	Whitehead	ESI
LaToria	Whitehead	HHS
Chad	Whiteman	U.S. Chamber of Commerce
David	Wieland	WORC

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Te Maia	Wiki	Native Sister Circle
Adam	Wilke	USDA NIFA
Donele	Wilkins	The Green Door Initiative
Roman	Williams	Delphinus Engineering, Inc.
Lance	Williams	Nuclear Energy Institute (NEI)
Edward	Williams	Regional Ready Rockaway
Clarence	Williams	Tecolote Perch
Jane	Williams	California Communities Against Toxics
Thomasina	Williams	Concerned Citizens for Duplin Cty.
Hailey	Williams	University of Kansas
Courtney	Williams	Booz Allen Hamilton
Hailey	Williams	University of Kansas
Ahwahnee	Williams	Private Citizen
Deborah	Williams	City of Springfield, Office of Public Utilities
Dana	Williamson	ASPPH/EPA
Sacoby	Wilson	University of Maryland College Park
Marlon	Wilson	AECOM
Annie	Wilson	NYELJP
Alyanna	Wilson	NCSU
Lorna	Withrow	NCDHHS, DPH, OSWP
Christine	Wolfe	California Council for Environmental and Economic Balance
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Ellisa	Wright	Accenture Federal Services
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Collin	Yarbrough	Southern Methodist University
Mara	Yarbrough	NM Permian Basin Climate Justice Coalition
Mara	Yarbrough	NM Permian Basin Climate Justice Coalition
Ben	Yawakie	Environmental Quality Board
Carolyn	Yee	California Environmental Protection Agency, Department of Toxic Substances Control
Connie	Yerby	Private Citizen
Naomi	Yoder	Healthy Gulf
Rachel	Young	CEQ
Dondre	Young	Office of U.S. Senator Debbie Stabenow
Ian	Zabarte	Native Community Action Council
Lynda	Zambrano	NATIONAL TRIBAL EMERGENCY MANAGEMENT COUNCIL
Hilary	Zarin	Dept of Interior

FIRST NAME	LAST NAME	ORGANIZATION
Chris	Zeitler	KCPS
Victor	Zertuche	U.S. EPA
Shawn	Zhang	JPMorgan Chase
Xiang	Zhang	JPMorgan Chase
Rob	Zidek	Private Citizen
Jennifer	Zingone	Private Citizen
Ronald	Zorrilla	Outdoor Promise
Ariela	Zycherman	NOAA

APPENDIX D. Written Public Comments

White House Environmental Justice Advisory Council
Talking Points for Jan 27, 2022

My name is francisca Acuna,

I am a climate resilience community organizer with GAVA (go austin vamos Austin) and a member of Anthropocene alliance, A2 Climagration. I have lived in my southeast Austin neighborhood for 20 years.

First I want to thank you for the opportunity to speak..

I'm here to support the Justice 40 initiative. I live in an area where stormwater infrastructure does not have the capacity to hold the amount of water that comes from localized flooding which is causing flooding just about every 2 years.

Development in the eastern crescent keeps happening because of high density demand in our neighborhoods causing flooding and displacement for neighborhoods that are already heavily populated, and residents heavily impacted by climate shocks and stressors.

I believe a way to contribute to climate justice you need to work equitably and have transparency, Community engagement needs to be inclusive, without big language jarganing and please meet me where I am to really make a difference.

You need to have strict policies in development along creeks and rivers,

Prohibit redevelopment in already flooding areas allowing conservation and restoration of public lands and waters, and increase reforestation.

Prioritize funding for localized flooding by updating old storm drainage infrastructure to mitigate flooding in communities that are more impacted by climate shocks and stressors.

Increase Green infrastructure and nature based solutions to improve access to recreation, reduce chronic illnesses, as well as to reducing or eliminating the risk of repetitive flood damage.

The Risk Rating 2.0 is great but it benefits 23% of the people that live in flood areas. In addition to that you need to put in place the Monthly flood insurance premium so residents can pay on a monthly basis. You are losing money by having too many people without flood insurance because they can't afford it.

There needs to be an increase of resources for chechnology preparedness, language access to information, and disaster preparedness such as standing orders for resilience hubs, and neighbohood preparedness.

Mental health before and after a disaster needs to be addressed by following up to residents to help them relocate, or rebuild. But it needs to be done in an humanistic fair way

Fluoridation Policy:
An Annotated Bibliography of Published Science

A sampling of the scientific studies and reports relevant to water fluoridation published since the HHS 2015 recommendation to lower the fluoridation target to 0.7 ppm is listed below.

I suggest these items provide compelling evidence that 0.7 ppm is neither optimal nor safe and that any claims to the contrary are ill-founded. Moreover, protests that more study is required before banning fluoridation is a tacit endorsement of human experimentation without individual consent which is medical assault - *Karen F. Spencer*

2021

BENCHMARK DOSE ANALYSIS: Using fluoride studies from MIREC and ELEMENT projects as input, the results of which are consistent with other studies, authors identify 0.2 mg/L as having an adverse impact on neurodevelopment. "The prospective studies offer strong evidence of prenatal neurotoxicity, and the benchmark results should inspire a revision of water-fluoride recommendations aimed at protecting pregnant women and young children."
https://pubmed.ncbi.nlm.nih.gov/34101876/

- Grandjean P, Hu H, Till C, Green R, Bashash M, Flora D, Tellez-Rojo MM, Song P, Lanphear B, Budtz-Jørgensen E. A Benchmark Dose Analysis for Maternal Pregnancy Urine-Fluoride and IQ in Children. Risk Analysis. 8 June 2021.

LIFETIME EXPOSURE: Fluoridation is the primary source of fluoride exposure for 1,629 Canadians between 3 and 79 that finds substantially higher lifetime fluoride exposure in fluoridated communities using CHMS data, increasing with age. Vulnerable subpopulations to adverse effects of fluoride noted as the young, those who are iodine deficient, and post-menopausal women. https://www.mdpi.com/1660-4601/18/12/6203/htm

- Julia K. Riddell, Ashley J. Malin, Hugh McCague, David B. Flora, and Christine Till. Urinary Fluoride Levels among Canadians with and without Community Water Fluoridation. Int. J. Environ. Res. Public Health 2021, 18(12), 6203.

KIDNEYS: This study of 1,070 adults found every 1 mg/L increment in the urinary fluoride concentrations was associated with significant increases of 22.8% in the risk of kidney function injury after adjusting for potential confounding factors. Authors conclude that long-term fluoride exposure is associated with compromised kidney function in adults, and that urinary NAG is a sensitive and robust marker of kidney dysfunction caused by fluoride exposure.
https://pubmed.ncbi.nlm.nih.gov/34478979/

- Wu L, Fan C, Zhang Z, Zhang X, et al. Association between fluoride exposure and kidney function in adults: A cross-sectional study based on endemic fluorosis area in China. Ecotoxicol Environ Saf. 2021 Aug 31;225:112735.

BEHAVIORAL CHANGES: Children in Cincinnati Childhood Allergy and Air Pollution Study (CCAAPS) assessed at age 12. Boys in particular did not experience significant anxiety or depression, yet had somatic behaviors based on their childhood urinary fluoride (CUF) concentrations, "seven times more likely to exhibit 'at-risk' internalizing symptomology."
https://pubmed.ncbi.nlm.nih.gov/34755609/

- Adkins EA, Yolton K, Strawn JR, Lippert F, Ryan PH, Brunst KJ. Fluoride exposure during early adolescence and its association with internalizing symptoms. Environ Res. 2021 Oct 29:112296.

Fluoridation Policy:
An Annotated Bibliography of Published Science

CRITICAL WINDOWS: Using urine samples and test scores from 596 mother-child Canadian pairs in the MIREC prospective cohort, researchers found evidence that developmental neurological damage was based on timing of fluoride exposure and gender, "Associations between fluoride exposure and PIQ (performance IQ) differed based on timing of exposure. The prenatal window may be critical for boys, whereas infancy may be a critical window for girls." https://pubmed.ncbi.nlm.nih.gov/34051202/

- Farmus L, Till C, Green R, Hornung R, Martinez-Mier EA, Ayotte P, Muckle G, Lanphear B, Flora D. Critical Windows of Fluoride Neurotoxicity in Canadian Children. Environ Res. 2021 May 26:111315.

GENES: Several genes make individuals more vulnerable to the neurotoxic impact with gender differences, also affecting mitochondria and suggesting vulnerability to dementia. Chinese study of 952 school children between 7 and 13 using water, urinary, hair and nail fluoride identified multiple neurodevelopmental metabolic pathways that result in adverse effects from low fluoride exposures. https://www.sciencedirect.com/science/article/pii/S0160412021003068

- Yu X, Xia L, Zhang S, et al. Fluoride exposure and children's intelligence: Gene-environment interaction based on SNP-set, gene and pathway analysis, using a case-control design based on a cross-sectional study. Environ Int. 2021 Jun 4;155:106681.

GENETIC VULNERABILITY: Dopamine relative genes affect the susceptibility of individuals to fluoride toxicity even in safe water concentrations which result in lowered IQ so that "low-moderate fluoride exposure is inversely related to children's IQ." https://pubmed.ncbi.nlm.nih.gov/33360592/

- Zhao L, Yu C, Lv J, et al. Fluoride exposure, dopamine relative gene polymorphism and intelligence: A cross-sectional study in China. Ecotoxicology and Environmental Safety. 2021 Feb;209:111826.

BRITTLE BONES: "In this cohort of postmenopausal women, the risk of fractures was increased in association with two separate indicators of fluoride exposure. Our findings are consistent with RCTs and suggest that high consumption of drinking water with a fluoride concentration of ~1 mg/L may increase both BMD (bone mineral density) and skeletal fragility in older women." https://pubmed.ncbi.nlm.nih.gov/33822648/

- Helte E, Donat Vargas C, Kippler M, Wolk A, Michaëlsson K, Åkesson A. Fluoride in Drinking Water, Diet, and Urine in Relation to Bone Mineral Density and Fracture Incidence in Postmenopausal Women. Environ Health Perspect. 2021 Apr;129(4):47005.

OSTEOARTHRITIS: Identifies fluoride as an environmental chemical that has adverse effects on articular cartilage and osteoarthritis (OA) risk. "In full sample analysis, a 1 mg/L increase in UF (urinary fluoride) level was associated with a 27% higher risk of OA." https://link.springer.com/article/10.1007/s12011-021-02937-2

- Sowanou, A., Meng, X., Zhong, N. et al. Association Between Osteoarthritis and Water Fluoride Among Tongyu Residents, China, 2019: a Case–Control of Population-Based Study. Biol Trace Elem Res (2021).

NO BENEFIT FOR PRESCHOOLERS: Polish study finds 'optimal' fluoride concentrations in water provide no dental benefit. Dental caries experience depended on oral hygiene and diet. https://www.sciencedirect.com/science/article/abs/pii/S0946672X2100016X

- Opydo-Szymaczek J, et al. Fluoride exposure and factors affecting dental caries in preschool children living in two areas with different natural levels of fluorides. Journal of Trace Elements in Medicine and Biology. Volume 65. 2021.

ALTERNATIVE: This systematic review and meta-analysis concludes that biomimetic hydroxyapatite-containing, fluoride-free oral care products are effective in reducing dental decay, especially in children without the risk of dental fluorosis and neurotoxicity inherent in topical use of fluoridated products. https://files.cdha.ca/profession/journal/2752.pdf

- Hardy Limeback, BSc, PhD, DDS; Joachim Enax, Dr; Frederic Meyer, Dr. Biomimetic hydroxyapatite and caries prevention: a systematic review and meta-analysis. | Can J Dent Hyg 2021;55(3): 148-159.

AMERICAN KIDNEYS: Using U.S. NHANES data from two recent cycles, finds 'optimal' amounts of fluoridated water results in high incidence of uric acid in adolescents suggesting higher risk of kidney disease and other illnesses. Identifies dose-response trend in plasma fluoride of teens.
https://www.sciencedirect.com/science/article/pii/S0147651320315074

- Yudan Wei, Jianmin Zhu, Sara Ann Wetzstein. Plasma and water fluoride levels and hyperuricemia among adolescents: A cross-sectional study of a nationally representative sample of the United States for 2013–2016. Ecotoxicology and Environmental Safety. Volume 208. 15 January 2021.

TODDLERS: The Programming Research in Obesity, Growth, Environment and Social Stressors (PROGRESS) cohort included 948 mother-child pairs from Mexico City. Blinded testing of children between one and 24 months to examine associations between maternal fluoride intake from food and beverages during pregnancy and offspring neurodevelopment in this prospective and longitudinal study found, "higher exposure to fluoride from food and beverage consumption in pregnancy was associated with reduced cognitive outcome, but not with language and motor outcome in male offspring over the first two years of life."
https://fluoridealert.org/wp-content/uploads/cantoral-2021.final_.pdf

- Alejandra Cantoral, Martha M. Tellez-Rojo, Ashley J. Malin, Lourdes Schnaas d, ErikaOsorio-Valencia, Adriana Mercadob, E. Angeles Martínez-Mier, Robert O. Wright, Christine Till. Dietary fluoride intake during pregnancy and neurodevelopment in toddlers: A prospective study in the progress cohort. Neurotoxicology 87 (2021) 86–93.

NO SAFE DOSE: Study of Mexican children and their mothers using measurements of urinary fluoride and water concentrations associated dental fluorosis and lowered IQ with fluoride dose consistent with findings of larger studies in other countries. Authors declare WHO fluoride guidelines are unsafe and hypothesize that 0.045 F^- mg/day is a protective exposure
https://www.mdpi.com/1660-4601/18/21/11490/htm

- Farías P, Estevez-García JA, Onofre-Pardo EN, Pérez-Humara ML, Rojas-Lima E, Álamo-Hernández U, Rocha-Amador DO. Fluoride Exposure through Different Drinking Water Sources in a Contaminated Basin in Guanajuato, Mexico: A Deterministic Human Health Risk Assessment. International Journal of Environmental Research and Public Health. 2021; 18(21):11490.

BABY BRAIN POISON: Exposure to fluoridated water (10 mg/L & 50 mg/L) beginning on the first day of pregnancy and continuing through the last day of breastfeeding shows chemical imbalances, cellular damage and changes in the hippocampus of Wistar rat offspring that would affect neurological development.
https://pubmed.ncbi.nlm.nih.gov/33096359/

- Ferreira MKM, Aragão WAB, Bittencourt LO, Puty B, Dionizio A, Souza MPC, Buzalaf MAR, de Oliveira EH, Crespo-Lopez ME, Lima RR. Fluoride exposure during pregnancy and lactation triggers oxidative stress and molecular changes in hippocampus of offspring rats. Ecotoxicology and Environmental Safety. 2021 Jan 15;208:111437.

GUTS & BRAINS: Memory function was reduced and gut microbiota structure was significantly altered in fluoride-exposed mice.
https://www.sciencedirect.com/science/article/pii/S0147651321002190

- Xin J, Wang H, Sun N, Bughio S, Zeng D, Li L, Wang Y, Khalique A, Zeng Y, Pan K, Jing B, Ma H, Bai Y, Ni X. Probiotic alleviate fluoride-induced memory impairment by reconstructing gut microbiota in mice. Ecotoxicol Environ Saf. 2021 Jun 1;215:112108

INFLAMED GUTS: Exposure to fluoridated water at both doses (10 mg/L & 50 mg/L) inflame guts in rats and alters the gut microbiome as compared to control (0 mg/L).
https://pubmed.ncbi.nlm.nih.gov/33508686/

- Dionizio A, Uyghurturk DA, Melo CGS, Sabino-Arias IT, Araujo TT, Ventura TMS, Perles JVCM, Zanoni JN, Den Besten P, Buzalaf MAR. Intestinal changes associated with fluoride exposure in rats: Integrative morphological, proteomic and microbiome analyses. Chemosphere. 2021 Jan 11;273:129607.

HARMFUL ADEQUATE INTAKE (AI): Study found "the levels of dietary F- intake were below the current AI, were greater towards the end of gestation and in women who were moderately and highly compliant with Mexican dietary recommendation" in ELEMENT cohort and recommended changing future dietary recommendations due to evidence of developmental neurotoxicity at even low dose exposure. https://pubmed.ncbi.nlm.nih.gov/33602354/

- Castiblanco-Rubio, G., Muñoz-Rocha, T., Cantoral, A., Téllez-Rojo, M., Ettinger, A., Mercado-García, A., Peterson, K.E., Hu, H., Martínez-Mier, E. (2021). Dietary Fluoride Intake Over the Course of Pregnancy in Mexican Women. Public Health Nutrition, 1-25.

CALCIUM & FLUORIDE IN PREGNANCY: Calcium intake during pregnancy lowers urinary fluoride (UF) concentrations by some unknown mechanism in ELEMENT cohort.
https://pubmed.ncbi.nlm.nih.gov/34176079/

- Castiblanco-Rubio GA, Muñoz-Rocha TV, Téllez-Rojo MM, Ettinger AS, Mercado-García A, Peterson KE, Hu H, Cantoral A, Martínez-Mier EA. Dietary Influences on Urinary Fluoride over the Course of Pregnancy and at One-Year Postpartum. Biol Trace Elem Res. 2021 Jun 26.

SAFETY: Evidence of dental fluorosis and other adverse effects to bodies and brains from supposed safe concentrations is alarming. "The safety of public health approach of drinking water fluoridation for global dental caries reduction are urgently needed further research."
https://www.sciencedirect.com/science/article/pii/S0147651321005510?via%3Dihub

- Dong H, Yang X, Zhang S, Wang X, Guo C, Zhang X, Ma J, Niu P, Chen T. Associations of low level of fluoride exposure with dental fluorosis among U.S. children and adolescents, NHANES 2015-2016. Ecotoxicol Environ Saf. 2021 Jun 22;221:112439.

SKELETAL FLUOROSIS: This Chinese study of the pathogenetic progression of skeletal fluorosis, details how local signaling pathways, hormones, promoter DNA hypermethylation, RNA expression etc. are affected by fluoride exposure leading to pain and disability.
https://www.mdpi.com/1422-0067/22/21/11932/htm

- Qiao L, Liu X, He Y, Zhang J, Huang H, Bian W, Chilufya MM, Zhao Y, Han J. Progress of Signaling Pathways, Stress Pathways and Epigenetics in the Pathogenesis of Skeletal Fluorosis. International Journal of Molecular Sciences. 2021; 22(21):11932.

DEPRESSION: Animal study finds negative changes in brain structure and behavior with exposure to sodium fluoride (NAF). https://pubmed.ncbi.nlm.nih.gov/34735150/

- Zhou G, Hu Y, Wang A, Guo M, Du Y, Gong Y, Ding L, Feng Z, Hou X, Xu K, Yu F, Li Z, Ba Y. Fluoride Stimulates Anxiety- and Depression-like Behaviors Associated with SIK2-CRTC1 Signaling Dysfunction. J Agric Food Chem. 2021 Nov 4. PMID: 34735150.

DECEPTION: This historical analysis documents how the ADA suppressed the established science that vitamin D was necessary for healthy teeth and bones in order to promote falsely fluoride which was and is more profitable for their membership. "Public health may well depend on looking at professional societies no different than the way we look at the pharmaceutical industry—conflicted organizations with a power to shape conventional wisdom based on fragile evidence." https://www.mdpi.com/2072-6643/13/12/4361/htm#

- Hujoel PE. How a Nutritional Deficiency Became Treated with Fluoride. Nutrients. 2021.

2020

AMERICAN FETAL EXPOSURE: Study on pregnant women in California and Montana find, "Fluoride concentrations in urine, serum, and amniotic fluid from women were positively correlated to public records of community water fluoridation" and that concentration is consistent with findings of Canadian studies that find these concentrations are associated with increased learning disabilities and lower IQ in offspring.
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7132865/

- Abduweli Uyghurturk D, Goin DE, Martinez-Mier EA, Woodruff TJ, DenBesten PK. Maternal and fetal exposures to fluoride during mid-gestation among pregnant women in northern California. Environ Health. 2020 Apr 6;19(1):38.

BLOOD: Canadian Health Measures Survey (CHMS) collects extensive biomonitoring data used to assess the exposure of Canadians to environmental chemicals finds higher fluoride in urine associated with significantly higher blood lead, urinary lead, etc. Also finds urinary selenium is significantly lower in fluoridated Canadian communities, "this is the first study where biomonitoring data from multiple cycles of CHMS were combined in order to generate robust estimates for subsets of the Canadian population. Such assessments can contribute to a regional-level prioritization of control measures to reduce the exposure of Canadians to chemicals in their environment."
https://www.ncbi.nlm.nih.gov/pubmed/31972364?dopt=Abstract

- Valcke M, Karthikeyan S, Walker M, Gagné M, Copes R, St-Amand A. Regional variations in human chemical exposures in Canada: A case study using biomonitoring data from the Canadian Health Measures Survey for the provinces of Quebec and Ontario. Int J Hyg Environ Health. 2020 Jan 20;225:113451.

THYROID & IQ: Concentrations of fluoride in drinking water considered optimal and safe in the US result in altered thyroid function and lowered IQ in Chinese children.
https://www.sciencedirect.com/science/article/pii/S0160412019301370

- Wang M, Liu L, Li H, et al.Thyroid function, intelligence, and low-moderate fluoride exposure among Chinese school-age children. Environment International. Volume 134, January 2020.

OVERDOSED CANADIAN BABIES: MIREC study documents Canadian bottle-fed babies have lower IQ in optimally fluoridated communities while breast fed babies have extremely low F and significantly higher IQ. https://www.sciencedirect.com/science/article/pii/S0160412019326145

- Till C, Green R, Flora D, Hornung R, Martinez-Miller EA, Blazer M, Farmus L, Ayotte P, Muckle G, Lanphear B. Fluoride exposure from infant formula and child IQ in a Canadian birth cohort. Environment International. 2020.

BIASED NARRATIVES: Canadian researchers comment on "expert" attacks on the high quality studies that contradict the dental CWF narrative, i.e. political suppression of scientific facts.
https://www.nature.com/articles/s41390-020-0973-8

- Till, C., Green, R. Controversy: The evolving science of fluoride: when new evidence doesn't conform with existing beliefs. Pediatr Res (2020).

BONE HEALTH: Low to moderate fluoride exposure weakens and damages bones in women.
https://www.sciencedirect.com/science/article/abs/pii/S0147651320308708

- Minghui Gao et al, Association between low-to-moderate fluoride exposure and bone mineral density in Chinese adults: Non-negligible role of RUNX2 promoter methylation. Ecotoxicology and Environmental Safety. Volume 203, 15 October 2020.

BONES: Found an age-specific association between fluoride exposure and altered CALCA methylation in adult women, affecting bone health. https://pubmed.ncbi.nlm.nih.gov/32283421/

- Sun R, Zhou G, Liu L, Ren L, Xi Y, Zhu J, Huang H, Li Z, Li Y, Cheng X, Ba Y. Fluoride exposure and CALCA methylation is associated with the bone mineral density of Chinese women. Chemosphere. 2020 Aug;253:126616.

SEX HORMONES IN FLUORIDATED US: "The data indicated gender- and age-specific inverse associations of fluoride in plasma and water with sex steroid hormones of total testosterone, estradiol and SHBG in U.S. children and adolescents."
https://www.sciencedirect.com/science/article/pii/S0269749119357963

- Bai, R., Huang, Y., Wang, F., & Guo, J. (2020). Associations of fluoride exposure with sex steroid hormones among U.S. children and adolescents, NHANES 2013–2016. Environmental Pollution, 114003

NERVOUS SYSTEM: The enteric nervous system (ENS) is called the second brain and governs the gastrointestinal track. Includes dopamine & serotonin function. Study finds "fluoride exposure during pregnancy and lactation might induce ENS developmental defects."
https://link.springer.com/article/10.1007/s12011-020-02249-x

- Sarwar, S., Quadri, J.A., Kumar, M. et al. Apoptotic and Degenerative Changes in the Enteric Nervous System Following Exposure to Fluoride During Pre- and Post-natal Periods. Biol Trace Elem Res (2020).

ENDOCRINE SYSTEM REVIEW: The endocrine system includes the pineal gland, hypothalamus, pituitary gland, thyroid with parathyroid glands, thymus, pancreas (partial endocrine function), adrenal glands, as well as male and female gonads (testes and ovaries) which are adversely effected by exposure to fluoride.
https://www.sciencedirect.com/science/article/abs/pii/S0045653520317604

- Marta Skórka-Majewicz et al, Effect of fluoride on endocrine tissues and their secretory functions -- review. Chemosphere, Volume 260, December 2020, 127565.

WHO IGNORES KIDNEYS: WHO guidelines of safety below 1.5 ppm fluoride concentration is wrong. "The available guidelines for drinking water are solely based on healthy populations with normal renal function. But, it is evident that once the kidney function is impaired, patients enter a vicious cycle as fluoride gradually accumulates in the body, further damaging the kidney tissue."
https://www.sciencedirect.com/science/article/abs/pii/S0045653520313795

- Shanika Nanayakkara, et al. The Influence of fluoride on chronic kidney disease of uncertain aetiology (CKDu) in Sri Lanka. Chemosphere. Volume 257, October 2020, 127186

PEDIATRIC BONE DISEASE: Identifies fluoride concentrations in water above 1.2 ppm as "dangerously high" that can cause pediatric bone disease. Urine measurements of fluoride in those afflicted are below the fluoride concentrations in women living in optimally fluoridated communities per 2017 Canadian study by Green et al.
https://pubmed.ncbi.nlm.nih.gov/32692054/

- Nipith Charoenngam, Muhammet B Cevik, Michael F Holick. Diagnosis and management of pediatric metabolic bone diseases associated with skeletal fragility. Curr Opin Pediatr. 2020 Aug;32(4):560-573.

EPA ON ENVIRONMENTAL STRESS: EPA authors find that exposure to fluoride has the greatest adverse impact on cognitive ability in children, even more than lead.
https://www.mdpi.com/1660-4601/17/15/5451/htm

- Frances M. Nilsen, Jazmin D.C. Ruiz and Nicolle S. Tulve. A Meta-Analysis of Stressors from the Total Environment Associated with Children's General Cognitive Ability. Int. J. Environ. Res. Public Health 2020, 17(15), 5451.

SOURCE: Compared MIREC, ELEMENT & PROGRESS data. MIREC & ELEMENT differed from PROGRESS in that "daily food and beverage fluoride intake was not associated with CUF in PROGRESS" but study "found that CUF (child urinary fluoride) levels are comparable among children in Mexico City and fluoridated Canadian communities, despite distinct sources of exposure. " https://pubmed.ncbi.nlm.nih.gov/33233802/

- Green, R., Till, C., Cantoral Preciado, A. D. J., Lanphear, B., Angeles Martinez-Mier, E., Ayotte, P., Wright, R. O., Tellez-Rojo, M. M., & Malin, A. J. (2020). Associations between urinary, dietary, and water fluoride concentrations among children in Mexico and Canada. Toxics, 8(4), 1-11. [110].

DENTAL FLUOROSIS & CWF CESSATION: Dental literature review by dentists finds "a significant decrease in the prevalence of fluorosis post cessation or reduction in the concentration of fluoride added to the water supply."
https://pubmed.ncbi.nlm.nih.gov/32598322/

- Nor Azlida Mohd Nor, Kuala Lumpur, Barbara L. Chadwick, Damian JJ. Farnell, Ivor G. Chestnutt. The impact of stopping or reducing the level of fluoride in public water supplies on dental fluorosis: a systematic review. Reviews on Environmental Health. 2020.

2019

SLEEP & PINEAL GLAND: "Chronic low-level fluoride exposure may contribute to changes in sleep cycle regulation and sleep behaviors among older adolescents in the US."
https://ehjournal.biomedcentral.com/articles/10.1186/s12940-019-0546-7

- Malin, A.J., Bose, S., Busgang, S.A. et al. Fluoride exposure and sleep patterns among older adolescents in the United States: a cross-sectional study of NHANES 2015–2016. Environ Health 18, 106 (2019)

ADHD: Youth in optimally fluoridated Canadian communities are almost 3 times more likely to be diagnosed with ADHD and have significantly higher rates of other learning disabilities as compared to their counterparts in non-fluoridated communities on a dose-response trend line.
https://www.sciencedirect.com/science/article/pii/S0160412019315971

- Riddell JK, et al. Association of water fluoride and urinary fluoride concentrations with attention deficit hyperactivity disorder in Canadian youth. Environment International. Volume 133, Part B, December 2019.

ASD: Increased exposure to fluoride is associated with higher incidence of ASD in regions with fluoridated water or endemic fluorosis. Based on biological plausibility and incidence, authors hypothesize that increased fluoride exposure is an environmental risk factor for autism.
https://www.mdpi.com/1660-4601/16/18/3431/htm

- Strunecka A, Strunecky O. Chronic Fluoride Exposure and the Risk of Autism Spectrum Disorder. Int. J. Environ. Res. Public Health 2019, 16(18), 3431.

PRENATAL: Three measurements in high quality NIH sponsored prospective cohort study (MIREC) found significantly lowered IQ in offspring of mostly white, well-educated Canadian women living in 'optimally' fluoridated communities.
https://jamanetwork.com/journals/jamapediatrics/fullarticle/2748634

- Green R, Lanphear B, Hornung R, et al. (2019) Association Between Maternal Fluoride Exposure During Pregnancy and IQ Scores in Offspring in Canada. JAMA Pediatrics. 2019.

KIDNEY & LIVER: Researchers at Mt. Sinai Medical School find American teens in optimally fluoridated American towns have markers for altered kidney & liver parameters that puts them at higher risk for kidney & liver disease as adults.
https://www.sciencedirect.com/science/article/pii/S0160412019309274

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GUTS: Animal study on microbiome health and immunity documents fluoride causes serious damage to rectal structure and significantly inhibits proliferation of rectal epithelial cells.
https://www.ncbi.nlm.nih.gov/pubmed/31885060/

- Wang H., Miao C., Liu J. et al. Fluoride-induced rectal barrier damage and microflora disorder in mice. Environ Sci Pollut Res (2019).

TEETH: An analysis of the dental fluorosis data in three U.S. NHANES reports noted that more than half of American teens have fluoride damaged teeth as the result of too much fluoride consumption during childhood. This results in costly cosmetic dentistry in young adulthood for millions as well as increased decay in the more severely affected.
(20% very mild + 15% mild + 28% moderate + 3% severe = 65% afflicted per 2011-12 data)
http://fluoridealert.org/wp-content/uploads/neurath.2019-1.pdf

- Neurath C, Limeback H, Osmunson Bm et al. (2019) Dental Fluorosis Trends in US Oral Health Surveys: 1986 to 2012. JDR Clinical & Translational Research.

ALZHEIMER'S: Even low concentrations of fluoride in drinking water at or below concentrations deemed optimal or safe by the WHO result in a pattern of increased dementia.
https://www.ncbi.nlm.nih.gov/pubmed/30868981

- Russ TC, Killin LOJ, Hannah J, Batty GD. Aluminium and fluoride in drinking water in relation to later dementia risk. The British Journal of Psychology. March 2019.

DNA DAMAGE: Mitochondrial dysfunction associated with dental fluorosis observed in Chinese children with fluoride concentrations in water identified as optimal or safe per U.S. authorities. Gender differences to the fluoride induced oxidative stress also noted.
https://www.sciencedirect.com/science/article/pii/S0160412018326291?via%3Dihub

- Zhou G, Yang L, Luo C, et al. Low-to-moderate fluoride exposure, relative mitochondrial DNA levels, and dental fluorosis in Chinese children. Environment International. Volume 127, June 2019, Pages 70-77.

DEMENTIA: Describes mechanism by which the effectiveness of the two most popular drugs used to treat Alzheimer's & other neurodegenerative dementia disease is reduced or blocked by fluoride. https://www.mdpi.com/1660-4601/16/1/10/htm

- Marta Goschorska, Izabela Gutowska, Irena Baranowska-Bosiacka, Katarzyna Piotrowska, Emilia Metryka, Krzysztof Safranow, Dariusz Chublek. Influence of Acetylcholinesterase Inhibitors Used in Alzheimer's Disease Treatment on the Activity of Antioxidant Enzymes and the Concentration of Glutathione in THP-1 Macrophages under Fluoride-Induced Oxidative Stress. Int. J. Environ. Res. Public Health, 2019, 16(1), 10.

ADULT BRAINS: First long term NaF animal study (10 weeks) using moderate levels of fluoride finds a number of histological changes including in parts of the brain associated with memory and learning. https://www.sciencedirect.com/science/article/pii/S0045653518317508

- Pei Jiang, Gongying Li, Xueyuan Zhou, Changshui Wang, Yi Qiao, Dehua Liao, Dongmei Shi. Chronic fluoride exposure induces neuronal apoptosis and impairs neurogenesis and synaptic plasticity: Role of GSK-3b/b-catenin pathway.Chemosphere. Volume 214, January 2019, Pages 430-435.

DELAYED MALE PUBERTY: This 4th study from the NIH sponsored ELEMENT investigation of the prenatal impact of low-dose prenatal exposure found a significant pattern of delayed puberty for boys associated with maternal fluoride as measured in urine samples. Female data showed non-significant trend towards earlier menarche. More study needed to determine the impact on sexual development. https://www.ncbi.nlm.nih.gov/pubmed/30922319

- Liu Y, Téllez-Rojo M, Hu H, et al. Fluoride exposure and pubertal development in children living in Mexico City. Environ Health. 2019 Mar 29;18(1):26.

ANXIETY & DEPRESSION: Both rats and children experience changes in brain chemistry from extended exposure to fluoride which affects mood. Serotonin and the prefrontal cortex are impacted. Studies that only examine short-term exposure are inadequate to detect these changes which are more pronounced in females.
https://www.sciencedirect.com/science/article/abs/pii/S0031938418309375

- Lu F, Zhang Y, Trevedi A, et al. (2019) Fluoride related changes in behavioral outcomes may relate to increased serotonin. Physiology & Behavior.

EYE DISEASE: Fluoride is a poison that has biological impact on consumers in any dose, contributing to the development of cataracts, glaucoma and macular degeneration.
https://www.mdpi.com/1660-4601/16/5/856

- Waugh DT. The Contribution of Fluoride to the Pathogenesis of Eye Diseases: Molecular Mechanisms and Implications for Public Health. Int. J. Environ. Res. Public Health. 2019, 16(5), 856.

BONES & GENES: This 30 day animal study at 8 mg/L fluoride documents DNA & RNA damage that inhibits gene expression which can be passed on through generations affecting bone development and contributing to weak bones, blood & bone cancers and skeletal fluorosis.
https://www.sciencedirect.com/science/article/pii/S0147651318311734?via%3Dihub

- Atule P. Daiwile, Prashant Tarale, Saravanadevi Sivanesan, et al. Role of fluoride induced epigenetic alterations in the development of skeletal fluorosis. Ecotoxicology and Environmental Safety. Volume 169, March 2019, Pages 410-417.

BRAIN INJURY: Fluoride interferes with calcium metabolism which impacts brain chemistry and poisons the hippocampus. "The imbalance of calcium metabolism caused by fluorosis may be a pathogenesis of brain injury induced by fluoride."
https://www.sciencedirect.com/science/article/pii/S0045653518324007

- Qiuli Yu, Dandan Shao. Rui Zhang, Wei Ouyang, Zigui Zhang. Effects of drinking water fluorosis on L-type calcium channel of hippocampal neurons in mice. Chemosphere. Volume 220, April 2019, Pages 169-175. [Online Ahead of Print]

BRAIN DAMAGE: Prenatal & postnatal animal experiment using 10, 50 and 100 mg/L to simulate human experience documents mitochondrial damage and neuronal death as mechanism that result in learning and memory impairments.
https://www.ncbi.nlm.nih.gov/pubmed/30659323

- Zhao, Q., Niu, Q., Chen, J. et al. Roles of mitochondrial fission inhibition in developmental fluoride neurotoxicity: mechanisms of action in vitro and associations with cognition in rats and children. Arch Toxicol (2019).

IODINE: Identifies and discusses the biochemical and hormonal impact of fluoride and fluoridation policy on iodine metabolism with consideration of related neurodevelopmental and pathological disorders. https://www.mdpi.com/1660-4601/16/6/1086

- Waugh DT. Fluoride Exposure Induces Inhibition of Sodium/Iodide Symporter (NIS) Contributing to Impaired Iodine Absorption and Iodine Deficiency: Molecular Mechanisms of Inhibition and Implications for Public Health. Int. J. Environ. Res. Public Health 2019, 16, 1086.

BIOLOGY OF POISON: Deep dive into the biological impact of fluoride that affects metabolism, hormones, immune function, etc. "Moreover, the findings of this study further suggest that there are windows of susceptibility over the life course where chronic F exposure in pregnancy and early infancy may impair Na+ , K+ -ATPase activity with both short- and long-term implications for disease and inequalities in health." https://www.mdpi.com/1660-4601/16/8/1427

- Waugh DT. Fluoride Exposure Induces Inhibition of Sodium-and Potassium-Activated Adenosine Triphosphatase (Na+, K+-ATPase) Enzyme Activity: Molecular Mechanisms and Implications for Public Health. Int. J. Environ. Res. Public Health 2019, 16(8), 1427

DOSE RESPONSE: Three month study on adult rats found "fluoride can impair the learning ability of rats, which may be related to the induction of autophagy in rat hippocampal neurons." https://www.ncbi.nlm.nih.gov/pubmed/31111310

- Zhang C, Huo S, Fan Y, Gao Y, Yang Y, Sun D. Autophagy May Be Involved in Fluoride-Induced Learning Impairment in Rats. Biol Trace Elem Res. 2019 May 20.

GENETIC SUSCEPTIBILITY: Review of recent scientific literature on biological impact. Same exposure in same population affect individuals differently, suggesting genetic vulnerability. https://onlinelibrary.wiley.com/doi/full/10.1111/jcmm.14185

- Wei, W, Pang, S, Sun, D. The pathogenesis of endemic fluorosis: Research progress in the last 5 years. J Cell Mol Med. 2019; 23: 2333– 2342.

MITOCHONDRIA: Prenatal and postnatal exposure to fluoride results in mitochondrial abnormalities, autophagy and apotheosis contributing to neuronal death. https://www.NCBI.nlm.nih.gov/pubmed/30659323

- Zhao, Q., Niu, Q., Chen, J. et al. Roles of mitochondrial fission inhibition in developmental fluoride neurotoxicity: mechanisms of action in vitro and associations with cognition in rats and children. Arch Toxicol (2019).

NUTRITION: The f-ion is a poison but the bioavailability of CaF is different than NaF as calcium is the antidote to fluoride poisoning. In addition to being in water and dental products, 20% of pharma and 40% of agrichemicals have a fluoride base. Consequently, people are exposed to excessive amounts of fluoride which contributes to chronic disease. https://journals.matheo.si/index.php/ACSi/article/view/4932/2095

- Stepec D, Ponikvar-Svet M. Fluoride in Human Health & Nutrition. Acta Chim Slov. 2019, 66.

2018

THYROID: 18% of people drinking 'optimally' fluoridated water in Canadian communities have a heightened risk of low thyroid function because fluoride interferes with iodine metabolism. Many of them will be sub-clinical and not know they are mildly hypothyroid, which nevertheless increases their risk for diabetes, high cholesterol, and other problems. Study excluded those already diagnosed with thyroid disease. (CHMS)
https://www.sciencedirect.com/science/article/pii/S016041201830833X

- Ashley J. Malin, Julia Riddell, Hugh McCague, Christine Till. Fluoride exposure and thyroid function among adults living in Canada: Effect modification by iodine status. Environment International. Volume 121, Part 1, December 2018, Pages 667-674.

THYROID: Even 0.5 ppm fluoride in water has an adverse impact on thyroid hormones. Water is currently fluoridated to 0.7 ppm, a reduction from up to 1.2 ppm in 2015.
https://www.NCBI.nlm.nih.gov/pmc/articles/PMC5805681/

- Z. Kheradpisheh et al. (2018) Impact of Drinking Water Fluoride on Human Thyroid Hormones: A Case-Control Study. Scientific Reports. volume 8.

OVERDOSED BABIES: Over one third of babies (37%) in fluoridated American communities consume amounts of fluoride in excess of the upper limits of fluoride considered safe per government regulations. Even 4% of babies in non-fluoridated communities are overdosed on fluoride due to consumption of products made with fluoridated water. At the very least, this puts these children at high risk for developing dental fluorosis. Dental fluorosis is associated with increased incidence of learning disabilities, broken bones and kidney disease.
http://jocpd.org/doi/10.17796/1053-4625-43.1.7

- Claudia X Harriehausen, Fehmida Z Dosani, Brett T Chiquet, Michelle S Barratt, and Ryan L Quock. Fluoride Intake of Infants from Formula. Journal of Clinical Pediatric Dentistry. 2018.

GOVERNMENT BIAS: A National Toxicology Program animal experiment studying the impact of fluoride consumption used the wrong rats, the wrong dose, and the wrong study design in order to manufacture a finding of no prenatal or postnatal effect.
https://www.sciencedirect.com/science/article/pii/S0306987718308600

- Karen Favazza Spencer, Hardy Limeback. Blood is Thicker Than Water: Flaws in a National Toxicology Program Study. Medical Hypotheses. Volume 121. December 2018. Pages 160-163.

PREGNANT WOMEN: Pregnant Canadian women drinking 'optimally' fluoridated water had twice the fluoride exposure per individual testing as compared to pregnant women in non-fluoridated Canadian communities - and consistent with the range in the Mexican women in the ELEMENT cohort whose children had up to 6 points lowered IQ based on prenatal exposure to fluoride (from salt). The Canadian study excluded those with health conditions such as kidney disease as well as considered confounding factors such as tea consumption.
https://ehp.niehs.nih.gov/doi/pdf/10.1289/EHP3546

- Christine Till, Rivka Green, John G. Grundy, Richard Hornung, Raichel Neufeld, E. Angeles Martinez-Mier, Pierre Ayotte, Gina Muckle, and Bruce Lanphear. Community Water Fluoridation and Urinary Fluoride Concentrations in a National Sample of Pregnant Women in Canada. Environmental Health Perspectives. October 2018.

LEARNING DISABILITIES: Over 200 children were individually tested. Study found attention deficit disorder apparently caused by their prenatal exposure to fluoride specific to dose. This is the 3rd report out of the NIH sponsored 12 year ELEMENT project that has confirmed low dose prenatal exposure to fluoride consistent with exposure in 'optimally' fluoridated communities causes subtle but permanent brain damage for many consumers. Excluded those with history of mental illness or conditions such as diabetes and renal disease. https://www.sciencedirect.com/science/article/pii/S0160412018311814

- Morteza Bashash, Maelle Marchand, Howard Hu, ChristineTill, Angeles Martinez-Mier, Brisa N. Sanchez, Niladri Basu, Karen Peterson, Rivka Green, Lourdes Schnaas, Adriana Mercado-García, Mauricio Hernández-Avila, Martha María Téllez-Rojo. Prenatal fluoride exposure and attention deficit hyperactivity disorder (ADHD) symptoms in children at 6–12 years of age in Mexico City. Environment International. Volume 121, Part 1, December 2018, Pages 658-666.

ALZHEIMER'S DISEASE: Describes impact of fluoride-induced stress and inflammation in the development of Alzheimer's disease and demonstrates the mechanism for cell death in its worsening over time. https://www.mdpi.com/1422-0067/19/12/3965

- Goschorska M, et al. Potential Role of Fluoride in the Etiopathogenesis of Alzheimer's Disease. Int. J. Mol. Sci. 2018, 19 (12), 3965.

CANCER: Researchers who include an IARC scientist find esophageal cancer is 9.4 times more prevalent among those with dental fluorosis in the endemic fluorosis regions of Kenya. Provides biological plausibility that inflammatory fluoride affects microbiome and other biological mechanisms. Recommends more study. https://www.ncbi.nlm.nih.gov/pubmed/30582155/

- Menya D, Maina SK, Kibosia C, Kigen N, Oduor M, Some F, Chumba D3, Ayuo P, Middleton DR, Osano O, Abedi-Ardekani B, Schüz J, McCormack V. Dental fluorosis and oral health in the African Esophageal Cancer Corridor: Findings from the Kenya ESCCAPE case-control study and a pan-African perspective. Int J Cancer. 2018 Dec 23.

KIDNEYS: Fluoride is a common exposure that is selectively toxic to the kidneys. https://www.sciencedirect.com/science/article/pii/S0270929518301827

- Lash LH. Environmental and Genetic Factors Influencing Kidney Toxicity. Seminars in Nephrology. Volume 39, Issue 2, March 2019, Pages 132-140.

IQ & DF: Between 0.5 and 3.9 mg/L, found every 0.1 mg/L increased dental fluorosis by 2.24% and every 0.5 mg/L decreases IQ by 2.67 points. Also found half as many kids with high IQ children with higher F- dose. https://www.NCBI.nlm.nih.gov/pubmed/29870912

- Yu X et al. Threshold effects of moderately excessive fluoride exposure on children's health: A potential association between dental fluorosis and loss of excellent intelligence. Environ Int. 2018 Jun 2;118:116-124.

2017

REVIEW: Concludes that fluoridation schemes whether from water, food or salt programs "pose risks of various diseases in the asthmatic-skeletal, neurological, endocrine and skin systems. Dental and skeletal fluorosis are signs of chronic and excessive ingestion of fluoride." https://www.NCBI.nlm.nih.gov/pubmed/28453591

- Verena Romero, Frances J. Norris, Juvenal A. Ríos, Isel Cortés, Andrea González, Leonardo Gaete, Andrei N. Tchernitchin. The impact of tap water fluoridation on human health. Rev. méd. Chile vol.145 no.2 Santiago Feb. 2017.

DOSE-RESPONSE: Validated that IQs of children are lowered on a dose-response trend line correlated with the amount of fluoride exposure as measured via urine tests of their mothers during pregnancy and individualized IQ tests of offspring. In the range consistent with doses in optimally fluoridated communities, there was up to a 6 point difference in IQ. This NIH sponsored 12 year longitudinal study conducted by researchers at world class American & Canadian universities excluded diabetics as well as those with kidney disease or pregnancy complications and allowed for many confounders.
https://www.sciencedirect.com/science/article/pii/S016041201830833X

- Morteza Bashash, Deena Thomas, Howard Hu, et al. Prenatal Fluoride Exposure and Cognitive Outcomes in Children at 4 and 6–12 Years of Age in Mexico. Environ Health *Perspect*. Sept 2017. Vol 125, Issue 9.

IQ & DF: Every 0.1 mg/L increased dental fluorosis by 2.24% and Every 0.5 mg/L decreases IQ by 2.67 points. There are half as many high IQ children in higher fluoride dose group.
https://www.NCBI.nlm.nih.gov/pubmed/29870912

- Yu X et al. Threshold effects of moderately excessive fluoride exposure on children's health: A potential association between dental fluorosis and loss of excellent intelligence. Environ Int. 2018 Jun 2;118:116-124.

GENES & BONES: "This study provides evidence that chronic oxidative and inflammatory stress may be associated with the fluoride-induced impediment in osteoblast differentiation and bone development." http://link.springer.com/article/10.1007/s12011-016-0756-6

- Gandhi, D., Naoghare, P.K., Bafana, A. et al. Fluoride-Induced Oxidative and Inflammatory Stress in Osteosarcoma Cells: Does It Affect Bone Development Pathway? Biol Trace Elem Res (2017) 175: 103.

PRESCHOOL DIET: Diet of two year olds contain unsafe levels of fluoride.
http://onlinelibrary.wiley.com/doi/10.1111/cdoe.12283/full

- Martinez-Mier EA, Spencer KL, Sanders BJ, Jones JE, Soto-Rojas AE, Tomlin AM, Vinson LA, Weddell JA, and Eckert GJ. Fluoride in the diet of 2-years-old children. Community Dent Oral Epidemiol. 2017;00:1–7.

APOPTOSIS: "Enamel fluorosis is a developmental disturbance caused by intake of supraoptimal levels of fluoride during early childhood.The enamel defects consist of horizontal thin white lines, opacities (subsurface porosities), discolorations, and pits of various sizes. The molecular mechanism underlying enamel fluorosis is still unknown….. We can hypothesize that fluorosis is due to a combination of direct cytotoxic effects causing cell death, the delayed development of tight junctions, which are necessary to form a sealed barrier between apical and basolateral surfaces, and a direct inhibitory effect of fluoride on vectorial calcium and/or bicarbonate transport." https://www.NCBI.nlm.nih.gov/pmc/articles/PMC5770627/

- Rácz, Róbert et al. "No Change in Bicarbonate Transport but Tight-Junction Formation Is Delayed by Fluoride in a Novel Ameloblast Model." Frontiers in Physiology. 2017; 8: 940.

DNA: Finds that "prolonged fluoride intake at chosen concentrations caused imbalance of the cellular oxidative state, affected DNA and disrupted cellular homeostasis… It is recommended that fluoride supplementation requires a fresh consideration in light of the current study."
https://www.NCBI.nlm.nih.gov/pubmed/28089781

- F.D. Campos-Pereira, L. Lopes-Aguiar, F.L. Renosto, et al. Genotoxic effect and rat hepatocyte death occurred after oxidative stress induction and antioxidant gene downregulation caused by long term fluoride exposure. Chem Biol Interact. 2017 Feb 25;264:25-33.

PRENATAL POISON: "F can pass through the cord blood and breast milk and may have deleterious impact on learning and memory of the mouse pups."
http://journals.sagepub.com/doi/abs/10.1177/0960327117693067

- Y Zhang, X Xue, R Niu, J Wang. Maternal fluoride exposure during gestation and lactation decreased learning and memory ability, and glutamate receptor mRNA expressions of mouse pups. Z Sun, Human & Experimental Toxicology. February 13, 2017.

IMMUNITY: Prenatal and early postnatal exposure to fluoride impairs spleen function and development which damages spleen and lifelong immunity.
https://www.NCBI.nlm.nih.gov/pubmed/28846973/

- Yanqin Ma, Kankan Zhang, Fengjun Ren, Jundong Wang, Developmental fluoride exposure influenced rat's splenic development and cell cycle via disruption of the ERK signal pathway, In Chemosphere, Volume 187, 2017, Pages 173-180

NEUROINFLAMMATION: Toxic effects of fluoride on the central nervous system and immunity.
https://link.springer.com/article/10.1007/s10753-017-0556-y

- Chen R, Zhao LD, Liu H. et al. Fluoride Induces Neuroinflammation and Alters Wnt Signaling Pathway in BV2 Microglial Cells. Inflammation. 2017;40: 1123.

2016

CRITIQUE HHS RECOMMENDATION: Pro-fluoridation team of dental researchers determined that the Department of Health and Human Services reduction of the optimal fluoride concentration to a single 0.7 ppm target is lacking in sound science, i.e. that "policy need to be cognizant of the balancing of risk and protective exposures across the entire population and potentially all ages and to be based on recent data that are purposefully collected, critically analyzed and carefully interpreted… (the recommendation seems) premature in terms of its rationale and its use and interpretation of sometimes dated data." These authors' bias is to maintain 1 ppm; nevertheless, their rationale against the HHS document is appropriate. The HHS document is political, not scientific.
https://www.NCBI.nlm.nih.gov/pubmed/26710669

- Spencer AJ, Do LG. Caution needed in altering the 'optimum' fluoride concentration in drinking water. Community Dent Oral Epidemiol. 2016 Apr;44(2):101-8.

OSTEOPOROSIS: "Consequently, although the World Health Organization continues to support F schemes for caries prevention despite a lack of scientific proof, the F schemes are not able to improve the crystal quality but rather contribute adversely to affect tooth development and increases the risk of developing postmenopausal osteoporosis."
http://dx.doi.org/10.4172/2379-1764.1000170

- Mitsuo Kakei, Masayoshi Yoshikawa and Hiroyuki Mishima. Fluoride Exposure May Accelerate the Osteoporotic Change in Postmenopausal Women: Animal Model of Fluoride-induced Osteoporosis. Adv Tech Biol Med 2016, 4:1

DIABETES: Fluoridation policy significantly increases incidence of age related type 2 diabetes.
https://www.NCBI.nlm.nih.gov/pubmed/27740551

- K. Fluegge. Community water fluoridation predicts increase in age-adjusted incidence and prevalence of diabetes in 22 states from 2005 and 2010. Journal of Water and Health, 2016.

IBD: Crohn's disease and ulcerative colitis increases after fluoridation begins in multiple countries. http://www.NCBI.nlm.nih.gov/pubmed/27199224

- Follin-Arbelet B, Moum B. Fluoride: a risk factor for inflammatory bowel disease? Scand J Gastroenterol. 2016 May 19:1-6.

PROPAGANDA: Assisted by the media, fluoridationists misrepresent historical and scientific fact in order to achieve a political end. https://www.researchgate.net/publication/305985332

- Anat Gesser-Edelsburg and Yaffa Shir-Raz. Communicating risk for issues that involve 'uncertainty bias': what can the Israeli case of water fluoridation teach us? Journal of Risk Research. August 2016.

2015

COCHRANE CWF REVIEW: Estimates that 12% of the children living in fluoridated communities with 0.7 ppm fluoridation have aesthetically objectionable dental fluorosis with a total dental fluorosis effect of 40%. The effects were 47% & 15% for 1 ppm, only a minor impact on incidence of dental fluorosis and consistent with the findings of the 2000 York Review. http://www.cochrane.org/CD010856/ORAL_water-fluoridation-to-prevent-tooth-decay

- Iheozor-Ejiofor Z, Worthington HV, Walsh T, O'Malley L, Clarkson JE, Macey R, Alam R, Tugwell P, Welch V, Glenny A. Water fluoridation for the prevention of dental caries. Cochrane Database of Systematic Reviews 2015, Issue 6.

THYROID: Diagnoses of low thyroid significantly higher in ‘optimally’ fluoridated regions. https://www.NCBI.nlm.nih.gov/pubmed/25714098

- S Peckham, D Lowery, S Spencer. Are fluoride levels in drinking water associated with hypothyroidism prevalence in England? A large observational study of GP practice data and fluoride levels in drinking water. J Epidemiol Community Health. 24 February 2015.

ADHD: Researchers found between 67k and 131k more 11 year olds with ADHD in fluoridated regions of the U.S.
http://www.ehjournal.net/content/pdf/s12940-015-0003-1.pdf

- A Malin and C Till. Exposure to fluoridated water and attention deficit hyperactivity disorder prevalence. Environmental Health 2015, 14:17

CWF INFLAMMATIONS: Found that “even in small concentrations fluoride changes the amounts and activity of COX-1 and COX-2 enzymes taking part in the initiating and development of inflammatory process.”
http://www.sciencedirect.com/science/article/pii/S0887233315001605

- I. Gutowskaa, et al. Fluoride as a factor initiating and potentiating inflammation in THP1 differentiated monocytes/macrophages. Toxicology in Vitro. Volume 29, Issue 7, October 2015, Pages 1661–1668.

NEUROTOXICANT: EPA scientists classify fluoride as a ‘gold standard’ developmental neurotoxicant with substantial evidence of harm.
http://www.sciencedirect.com/science/article/pii/S0892036215300362

- William R. Mundy, Stephanie Padilla, Joseph M. Breier, at al. Expanding the test set: Chemicals with potential to disrupt mammalian brain development. Neurotoxicology and Teratology. Volume 52, Part A, November–December 2015, Pages 25–35.

PROPAGANDIZING: The proponents of fluoridation ignored concerning evidence and did not deliver on their promise of dental benefit then, and now. Neither did they do the expected due diligence re harms. https://doi.org/10.2105/AJPH.2015.302660

- Carstairs C. (2015). Debating Water Fluoridation Before Dr. Strangelove. American journal of public health, 105(8), 1559–1569.

NOT COST EFFECTIVE: Reveals errors in cost-benefit analysis (CBA) used by CDC. Best case scenario after corrections is a $3 benefit which is more than wiped out by any consideration of dental fluorosis. Fluoridated drinking water results in an economic loss to communities. http://www.NCBI.nlm.nih.gov/pubmed/25471729

- Lee Ko & Kathleen M. Thiessen (2015) A critique of recent economic evaluations of community water fluoridation, International Journal of Occupational and Environmental Health, 21:2, 91-120

Additional items of note:

2017 IAOMT Position Paper: https://iaomt.org/iaomt-fluoride-position-paper-2/
2018 Open Letter: http://www.multibriefs.com/briefs/icim/nutrition.pdf
2019 Children's Health Defense Statement: https://childrenshealthdefense.org/news/u-s-water-fluoridation-a-forced-experiment-that-needs-to-end/
2020 Expert Opinion: https://www.ehn.org/fluoride-and-childrens-health-2648120286.html

"…fluoride is presumed to be a cognitive neurodevelopmental hazard to humans…"
- Draft Monograph from National Toxicology Program, "Systematic Review of Fluoride Exposure and Neurodevelopmental and Cognitive Health Effects" (Sept 6, 2019)

Fluoridation policy poses a hazard to an unsuspecting public

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DEFINITIONS:

- **Endorsement**: An endorsement is an authoritative statement reflecting a point of view for the purpose of exerting influence. An endorsement is ***not*** an expert opinion.
    - **Authoritative statement:** An opinion that interprets a rule, law or policy for the purpose of guiding, influencing, or mandating action. Authoritative statements are not inherently trustworthy or reliable, but they are inherently manipulative. "Testimonial propaganda" utilizes authoritative statements in marketing and in politics. The slogan "question authority" was intended to encourage critical thinking in order to combat the blind acceptance of biased authoritative statements that endorse policy and/or sanctioned narratives. (*Logical Fallacies: Appeal to Authority*)
- **Expert Opinion**: An expert opinion is dependent on evidence and the due diligence of someone with substantial study in a field. The Daubert Standard is a legal process that validates the trustworthiness of experts offering opinion in a court of law.

EXAMPLES:

ENDORSEMENT: The April 2015 HHS statement recommending 0.7 ppm fluoride concentration in drinking water for 'safe & effective' prevention of tooth decay promoted the long standing fluoridation policy of the agency.
vs.
EXPERT OPINION: The June 2015 Cochrane report finds no reliable evidence of dental benefit to adults or low income children, but documents substantially higher rates of dental fluorosis, some of which will likely result in costly cosmetic dentistry.
The 2019 National Toxicology Program systematic review offered an expert opinion based on the evidence that fluoride is a presumed hazard to human health specific to neurotoxic impact when exposure is pre- or post-natal.

Hello. My name is Dave Arndt, a Baltimore Maryland resident and a Climate, Environmental and Social Justice advocate. These three areas have a lot of overlap and I am going to focus on topics at the intersection of these areas. Unfortunately, by plan, all of this injustice is burdened on Black, Brown and low-income areas. Let me repeat that – this was and is all done by design.

I sorry to say, just five years ago I did not know this. Sure, I knew that air pollution and environmental and social in justice was bad, but I never saw the design. My eyes have been opened; however, we need to open everyone's eyes. This advisory council is a good start in doing that, thank you for your work and this opportunity.

Let's look at Baltimore. What neighborhoods do the highways go through? Where is the trash incinerator? Where is the coal terminal? Where are the chemical plants? Or let's look at this another way, where is there a lack of reliable mass transportation? Where are the no grocery stores? Where are all the schools being shut down or not being repaired? Where are the high asthma rate? Please note Baltimore City has asthma at twice the rate of the rest of the country.   We all know the answers to my questions, Black, Brown and low-income neighborhoods. In Baltimore it is called the Black Butterfly. This is environmental and social injustice is replicated all across the US – all by design.

An example of this is that we are trying to stop a crematorium from being built in a Black residential neighborhood. The EPA has allowed the funeral industry to change the classification of these incinerators to a non-clean air act regulated industry, for marketing reasons. At this point, there is nothing we can do to prevent this- every government agency says it's ok. We see it, but can't prevent it. If this was located in the Baltimore's White "L", it just wouldn't happen.

Recognition is the first step; however, we need action now. People are suffering and dying. Which is bad enough, but the cumulative effects last for decades for the survivors. They are trapped in a downward spiral of poor health, no jobs, poor schools, no generational wealth transfer.

There are four major reasons for this that have to be addressed:

1. Individual regulations are not strict enough, they put profit and jobs over the health of residents
2. Regulations are done individually, there are no cumulative effects. When people breath, they can't choose which pollution source they are breathing in, they get it all.
3. Monitoring and enforcement. It has been shown repeatably that industry can't self-regulate. Monitoring needs to be funded and community driven. And without "a stick" and I mean a big stick, most fines are just a small expense in doing business.
4. Environmental injustice. This was all done by design. We must keep asking, If this was a wealthy white neighborhood, would it be allowed?

Today so far, we have heard hundreds of stories like this all across this nation. It is time for the EPA and all federal agencies to act like agencies for all the people and a healthy environment. Not corporate profit supporters

Thank you,

Dave Arndt

WHEJAC and NEJAC Public Comment (January 26, 2022)

Assault from Within, EPA's Attempt to Topple the Clean Water Act

South River Watershed Alliance has been engaged in a legal fight to protect the CWA since 2019. Playing out under the cover of the DeKalb County Consent Decree is an unprecedented attempt by EPA to upend the Clean Water Act (CWA). The case was argued before the 11th Circuit Court of Appeals in mid-December 2021 and we await the court's decision.

SRWA's lawsuit seeks to uphold the most important provision of the CWA – compliance.

The district court found that "the premise of Plaintiffs' argument is undisputed — unlike priority areas, the Consent Decree does not establish a deadline for DeKalb to stop spills, or rehabilitate the [sewer system] in non-priority areas."

The district court also held that "compliance means an *end* to violations, not merely a reduction in the number or size of them." No technical expertise is required to understand that a consent decree that "does not establish a timeline" to stop spills or rehabilitate the sewer system in non-priority areas is not capable of *requiring* compliance with the Clean Water Act in those areas.

Non-priority areas consist of approximately 69 percent of the County's sewer lines, more than 1,750 miles of sewer pipes, most of the sewage spilled into streams and the South River, and where more than 400,000 black residents live, one of the largest enclaves in the United States.

DeKalb County doesn't get a pass simply because it reached a settlement with EPA that "addresses" the entire sewer system. The settlement will not eliminate sewage spills in non-priority areas. EPA's "efforts" are not relevant here.

Furthermore, DeKalb County doesn't cite any case with similar facts, i.e., where a court found diligent prosecution (aka EPA's "efforts") even though the defendant could violate the Clean Water Act in perpetuity without violating a consent decree.

It is hard to reconcile reliance on EPA's "efforts to require compliance with the Consent Decree" with the district court's finding that the County "is admittedly not complying with the Consent Decree — with the express permission of" EPA.

Our hope is that the 11th Circuit Court of Appeals will follow the law rather than re-write it.

PO Box 1341 | Decatur, GA 30031 | southriverga.org

An adverse decision will effectively topple the CWA and set a nationwide precedent with potentially devasting impacts on environmental justice communities most impacted by poor water quality.

Selecting the South River, a small obscure urban river in metro Atlanta, plagued by pollution and impacting almost exclusively underserved and over-burden communities, for EPA's power grab and test case was not an accident. Why is EPA attempting to gut the CWA is the question we all should be asking and actively opposing? For certain, the efforts of EPA are not and never will be a replacement for achieving the clear intent of the CWA which is compliance with the law.

Jacqueline Echols, PhD
Board President
South River Watershed Alliance
Southriverga.org
jechols@southriverga.org
(404) 285-3756

WHITE HOUSE ENVIRONMENTAL JUSTICE ADVISORY COUNCIL VIRTUAL PUBIC MEETING

January 26, 2022, Lynne Bonnett, New Haven CT, USA

10,000 Hawks is an empowered group of neighbors, professionals, and organizational allies who collaborate on research, action, and education to address public health and ecological issues in support of the Land and all future generations. We envision that all Southern CT communities enjoy a healthy environment that leads to a high quality of life, teeming with (bio)diversity.

I. Current EJ Issues regarding the proposed expansion of Tweed New Haven Airport

The consultants stated at the Nov. 18, 2021 meeting at Nathan Hale School in New Haven that there are no formal regulations or standards for climate change at this time in the FAA approved process governing the EA process. 1050.1F and 5050.4b.

- The State of CT had and has a robust public participatory process to help communities adapt and mitigate the effects of climate change using an environmental justice and equity lens due to the recognition that the brunt of climate change will affect vulnerable communities the hardest. DEEP.climatechange@ct.gov: **The FAA needs to adopt what our state has already created since this proposal takes place in the State of CT and use our guidelines for climate change in their EA process.**

- Air pollution from expansion: people taste jet fuel after jets take off, increased traffic, acute exposures from take offs and taxiing- they plan to use 24 hr averages that will not capture the acute exposures that we know are linked to inflammatory circulatory events such as heart attacks and strokes. How do we measure jet fuel?
- How far do air pollutants travel on the wind? **A citizen science study showed air pollution in this area traveled 3 miles during a specific three week period in 2016**: Peralta*, R., Mendoza*, A., Levin, E., Mann, E., Carpi, A. "Using moss as a biomonitor to detect atmospheric sources of mercury in New Haven, CT," International Conference on Mercury as a Global Pollutant, Providence, RI, July 16- 21, 2017. Poster is attached. New Haven Tweed Authority does not plan to address cumulative air emissions outside of their immediate area instead saying that the already polluted air is from other sources. True, why then are they allowed to add to it? Why shouldn't they address the cumulative effects of what they are proposing in a community that is already overburdened with polluting facilities?
- Flooding: already terrible problems for residents in the area. Rising sea levels up to 20 inches by 2050, modifications to the wetland storage and drainage systems: they may be planning to design an expansion that allows for certain levels of flooding, the lease agreement lasts 43 years.
- Flights taking off cause vibration that I feel in my dwelling, plaster cracking off of the inside window sash, cracks in walls, visible water shaking in a glass during take off from vibration.

Priorities of the GC3 Climate Change working group on Environmental Justice and Equity, page 47: https://portal.ct.gov/-/media/DEEP/climatechange/GC3/GC3-working-group-reports/GC3_Equity_EJ_Final_Report_111320.pdf

" EEJ also expressed concern about cumulative impacts of environmental and climate health risks. Plans must assess cumulative impacts of existing and proposed sources of pollution in order to understand which communities are already overburdened and where mitigation efforts should be targeted and funding for adaptation prioritized. This issue should also be considered when developing indices for the Environmental and Climate Justice Mapping Tool. Please see Appendices C (Review of Financing Adaptation and Resilience Working Group Report), D (Review of Infrastructure and Land Use), and E (Review of Public Health and Safety) for additional feedback and preliminary EEJ Subcommittee recommendations. Notably, Infrastructure and Land Use took significant steps to prioritize equity and environmental justice, recommending the establishment of a statewide climate adaptation implementation committee focused on vulnerable communities, calling for an assessment of the vulnerability of transportation infrastructure to climate change, and relying on mapping to identify vulnerable communities. The report also recognizes the need for hyper-local planning to increase the effectiveness and likelihood of implementation of climate adaptation and education efforts."

I ask that the FAA recognize our state's work on climate change and respect the need for evaluating climate change in Tweed New Haven's Environmental Assessment. There is a great gap in what our state has created and what the FAA says it needs to do in an environmental assessment.

II: The consultants plan to address climate change by measuring greenhouse gas emissions.

- City of New Haven's recent study using an ICLEI protocol concentrated mainly on local contributors giving a very false impression of the true nature of GNG emissions in New Haven.

- New Haven is a host city for regional infrastructures such as deep water port offloading fossil fuels, fossil fuels tank farms, pumping fossil fuels to Hartford area (aviation fuel), train hub where engines are converted from diesel to electric, waste water treatment plant with a sewage sludge incinerator from other cities (up to 45% of incinerated sludge comes from outside our region). Trucking, an integral part of sludge incineration, is not a contributor to GHG emissions from sewage sludge incineration but rather lumped in to transportation. The GNG emission evaluation only recorded the portion of sludge that new Haven generated (less that 50% of the total and did not include the 15 semi truck loads hauling the sludge. How can our state evaluate GNG emissions from this sector if the regional aspect is not accounted for? It is not appropriate to only evaluate each city; there are 169 towns/cities in CT and only a few host cities that incinerate sludge. 67% of sewage sludge is trucked to one of the few incinerators.

- ICLEI protocols are an exciting new measurement tool that will allow standardized evaluation and comparison across different geographical areas. As such, it is important that environmental justice cities such as New Haven be evaluated in such a way that their function as host cities for regional activities reflect what actually occurs. https://icleiusa.org/ghg-protocols/.

WE NEED STANDARD PROTOCOLS FOR ENVIRONMENTAL JUSTICE COMMUNITIES THAT ACCOUNT FOR GNG EMISSIONS THAT ARE REGIONAL. The City of New Haven pushed through an incomplete 43-year lease for airport expansion but chooses to not add its expansion to greenhouse gas inventory.

Thank you,
/Lynne Bonnett/
January 25, 2022
New Haven CT

Adriane J. Busby
Sr. Food and Climate Policy Analyst
Friends of the Earth
1101 15th St NW, Washington DC 20005

February 8, 2022

Peggy Shepard, Chair
Richard Moore, Chair
White House Environmental Justice Advisory Committee

Re: Harmful Health Impacts of Industrial Animal Agriculture on EJ Communities

Dear Ms. Shepard, Mr. Moore, and Members of the WHEJAC:

On behalf of Friends of the Earth and our more than 3 million U.S. members and supporters, thank you for your service and dedication to the environmental justice movement and for this opportunity to provide comments.

The Biden Administration has committed to environmental justice in climate policy. On January 27, 2021, President Biden signed the Executive Order on Tackling the Climate Crisis at Home and Abroad, and section 219 of that Order commits the Administration to placing environmental justice at the center of climate policy.

Friends of the Earth commends the Biden Administration for this commitment, but the Administration is failing to uphold this promise as it relates to the development of its methane reduction plan and oversight of concentrated animal feeding operations (CAFOs). CAFOs, or factory farms, are a major driver of the climate crisis. Animal agriculture accounts for 37% of US methane emissions and at least 8% of overall emissions. Further, these highly concentrated animal operations produce massive quantities of waste and toxic pollutants that degrade community air and water resources and destroy the quality of life for the communities in which they are located, which are disproportionately communities of color and low-wealth communities. In order to protect the health and well-being of these communities, Friends of the Earth respectfully asks WHEJAC to make the following recommendations to the Biden Administration:

1) Use its existing legal authority to track GHG emissions and require methane reductions from industrial dairy and hog operations;
2) End its financial support for anaerobic methane digesters and manure-to-energy projects, which will further incentivize the growth of destructive factory farming production systems; and
3) Urge the EPA to act swiftly to reinstate the requirement that CAFOs report on their hazardous air emissions from animal waste under the Emergency Planning and Community Right to Know Act (EPCRA).

There are approximately 450,000 AFOs operating in the U.S., and many of them have been given a free pass to pollute and profit at the expense of EJ communities for decades. There is ample science that demonstrates the major role industrial animal agriculture plays in contributing to climate change.[1] Further, the waste generated by factory farms often contains pathogens, antibiotic-resistant bacteria, and heavy metals, and the spray from land application of manure can, and often does, reach nearby homes and drinking water sources.[2] The odor plumes, which often pervade nearby communities, contain respiratory and eye irritants including hydrogen sulfide and ammonia.[3] A growing body of research suggests these emissions may contribute not only to respiratory ailments in nearby residents but also decreased quality of life, mental stress, and elevated blood pressure.[4]

All of these negative impacts disproportionately affect low-income communities and communities of color because of where factory farms operate. Research has shown a disproportionately high concentration of CAFOs in communities of color despite the declining number of farmers of color in the southeastern United States.[5] This is targeted and intentional.

One study published last year found approximately 12,700 deaths per year from air pollution in the U.S. are attributable to industrial livestock production.[6] That is more deaths than occur from pollution from coal plants. Yet industrial livestock remains largely unregulated, and this administration's proposed increased investment in anerobic methane digesters and other subsidies for large CAFOs threaten to expand the industry.

President Biden's methane reduction strategy for agriculture - which is the #1 source of U.S. methane emissions – relies heavily on expanding public investments in the production of factory farm methane gas by subsidizing the cost of manure digesters and other infrastructure to enable manure-to-gas projects. While these policies are designed to reduce methane emissions, increased subsidization of these projects is likely to have the opposite effect. While more research is needed, evidence from California's Dairy Digester Programs shows how methane digester subsidies primarily benefit large-scale operators and incentivize the expansion of their herds and clustering of environmentally harmful fossil fuel infrastructure in already overburdened areas to maximize profit.[7] [8] In other words, companies will choose to produce more animals to get more manure – not just to get more meat. As one CAFO operator recently noted "We used to joke about how funny it would be if we could make more money off the poop than the milk, And now we're essentially here." This expansion will create even more

[1] *See* Intergovernmental Panel on Climate Change. Climate Change 2021: The Physical Science Basis, August 7, 2021, available at https://www.ipcc.ch/report/sixth-assessment-report-working-group-i/; *see also* EPA, *Overview of Greenhouse Gases, Methane,* available at https://www.epa.gov/ghgemissions/overviewgreenhouse-gases#methane.

[2] Hribar, C., *Understanding Concentrated Animal Feeding Operations and Their Impact on Communities*, National Ass'n of Local Boards of Health (2010). Available at https://www.cdc.gov/nceh/ehs/docs/understanding_cafos_nalboh.pdf.

[3] Nicole, W., *CAFOS and Environmental Justice: The Case of North Carolina* (2013) Environmental Health Perspectives, available at https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3672924/.

[4] Id.

[5] Mirabelli, M.C., et.al., Race, *Poverty, and Potential Exposure of Middle School Students to Air Emissions from Confined Swine Feeding Operations* (2006), available at https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1440786.

[6] Domingo, N.G., et. al., *Air quality-related Health Damages of Food* (2021), available at https://www.pnas.org/content/118/20/e2013637118/tab-article-info.

[7] https://leadershipcounsel.org/wp-content/uploads/2019/04/A-Working-Paper-on-GGRF-Dairy-Digester-Program.pdf

[8] https://leadershipcounsel.org/california-air-resources-board-program-incentivizes-greenhouse-gas-emissions-and-pollution-from-dairy-and-pig-farms/

pollution for communities of color, especially since digesters do nothing to mitigate co-pollutants like nitrous oxide, ammonia, hydrogen sulfide and heavy metals that are directly harming the health of surrounding communities. Some studies have shown that biogas digestors actually increases emissions of other toxic air pollutants such as NOx.[9] With expanded herd sizes, toxic air pollution from CAFOs will generate increased cases and severity of respiratory illnesses, as well as nausea, headaches, and other health conditions, especially in BIPOC communities. We have seen this happen already in places with a lot of methane digesters, such as in California's central valley.[10]

This Administration is not just failing to act on factory farm regulation but is actively going in the wrong direction by subsidizing CAFOs. Friends of the Earth asks that WHEJAC work with the Biden Administration to uphold its commitment to EJ principles by: (1) exercising its authority to meaningfully regulate CAFOs, (2) stopping further subsidization of CAFOs; and instead, (3) support a just transition to ecologically regenerative and just agriculture.

Thank you for your commitment to environmental justice and your service on the Committee. Please contact me with any questions or requests.

Sincerely,

Adriane Busby
Sr. Food and Climate Policy Analyst
Friends of the Earth, U.S.

9 https://www.liebertpub.com/doi/10.1089/env.2021.0025
10 https://leadershipcounsel.org/tell-the-board-of-supervisors-no-more-dirty-fuels-in-kern-county/

New York State Department of Environmental Conservation
Region 3 Office/Solid Waste Program
21 South Putt Corners Road, New Paltz, New York 12561-1620
Phone: (845) 256-3126 $ **FAX:** (845) 255-3414
Website: www.dec.ny.gov **E-mail:** sxparisi@gw.dec.state.ny.us

Joe Martens
Commissioner

October 5, 2011

Burt McCullough
Manager of Environmental Projects
Mirant Corporation
1155 Perimeter Center West
Atlanta, GA 30338-5416

Re: Mirant-Lovett LLC, T-Stony Point, Rockland County - Consent Order No. 3-20080414-15
Site Characterization Report

Dear Mr. McCullough:

The Department has reviewed the May 15, 2010 Site Characterization Report (the Report), prepared by HDR, and submitted on behalf of Mirant Lovett LLC, in connection with the Mirant-Lovett site in the Town of Stony Point, Rockland County and Order on Consent # 00320080414-15 (dated July 17, 2009). The Department's findings relative to the report are outlined below.

In general, the investigation was carried out in conformance with the approved site investigation work plan (the Work Plan) previously approved by the Department. Deviations from the Work Plan are discussed in section 2.2 of the Report and, in most cases, concurrence from Department staff is documented in the Report. Significant exceptions include monitoring well MW-J2 which was located approximately 25 feet northeast of the location shown in the approved Work Plan, MW-L which is located 85 feet to the east of the location shown in the approved Work Plan, MW-H2 which is located 25 feet to the northeast of the location shown in the approved Work Plan and MW-K2 which is located approximately 30 feet south of the location shown in the approved Work Plan. These deficiencies notwithstanding, the Report is considered to be adequate to serve as a basis for identifying environmental damage caused by the facility which will require remedial action and is therefore approved. Specifically, the following are the environmental issues identified by Department staff based on review of the report and/or observations made at the site which require remedial action or additional investigative work to serve as a basis for remedial design:

1. A plume of groundwater contamination including sulfate, boron, selenium, arsenic and other coal or coal ash derived contaminants which is migrating southward from the former coal storage pile area, the former coal ash settling lagoons and the CAMF to where it may enter the capture zone of the dewatering system of the Tilcon mine;

2. Soil contamination including PAHs, PCBs and toxic metals associated with several AOCs;

3. A groundwater contamination plume of petroleum-related contamination migrating southward from the former PBS area under the influence of the Tilcon mine dewatering system;

4. A residue of C&D debris, other than the waste types allowed at exempt C&D debris disposal sites, which is observable at the surface throughout the areas where facility buildings were demolished (see August 12, 2009 inspection report, attached);

5. Pockets of waste coal which were not removed due to proximity to the active railroad tracks or other reasons; and

6. Release of contamination from the CAMF to surface water as indicated by the increase in sulfate between the upstream sample (17 mg/L) and downstream sample (67 mg/L).

The Report is hereby approved and, in accordance with paragraph II.D.3, is incorporated into the Order on Consent as Appendix B. Mirant has already satisfied the requirement of paragraph II.D.5 to submit the report is an suitable electronic format.

The investigation which has been completed was extensive in scope and represents an important milestone in the process of addressing the various environmental impacts resulting from a long and varied site history. The next step in the process will be the development of a proposed plan of remedial action to mitigate the impacts which have been identified and to monitor the effectiveness of the remedial measures.

Department staff would like to continue our cooperative working relationship as we move forward into the remedial design phase at the Site.

Please indicate whether Mirant Lovett LLC is willing to develop a remedial action plan to address the environmental issues outlined above. To accomplish this we will need your continued cooperation and we request your response within thirty (30) days. A meeting can then be scheduled to discuss the details and timeframe for development of an approvable site remediation plan and an Order on Consent to provide the necessary legal framework. I look forward to your response and to working with you to build on the progress which was made during the investigative phase of the project.

Sincerely,

Steven Parisio

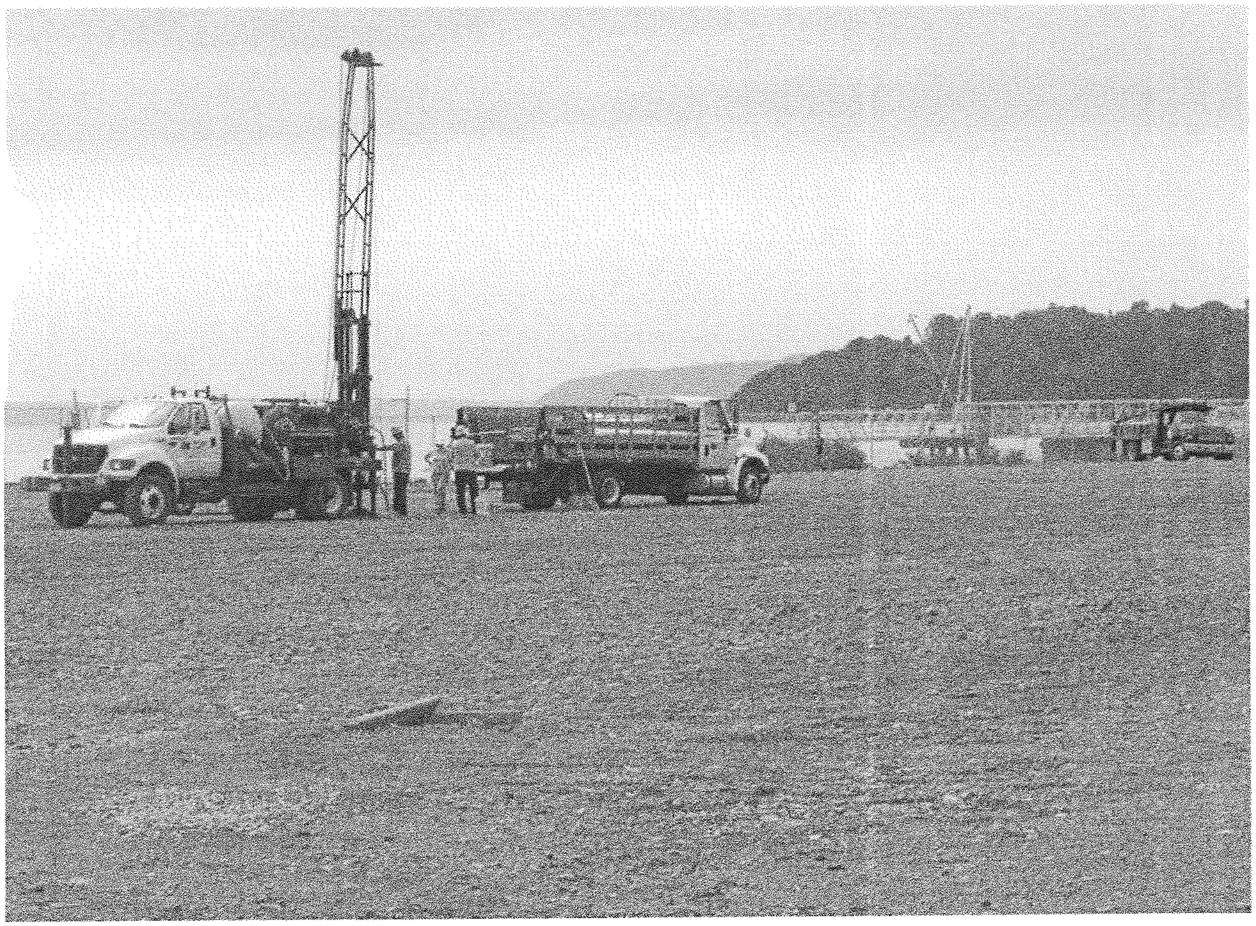
Steven Parisio
Regional Solid Waste Geologist

Attachment

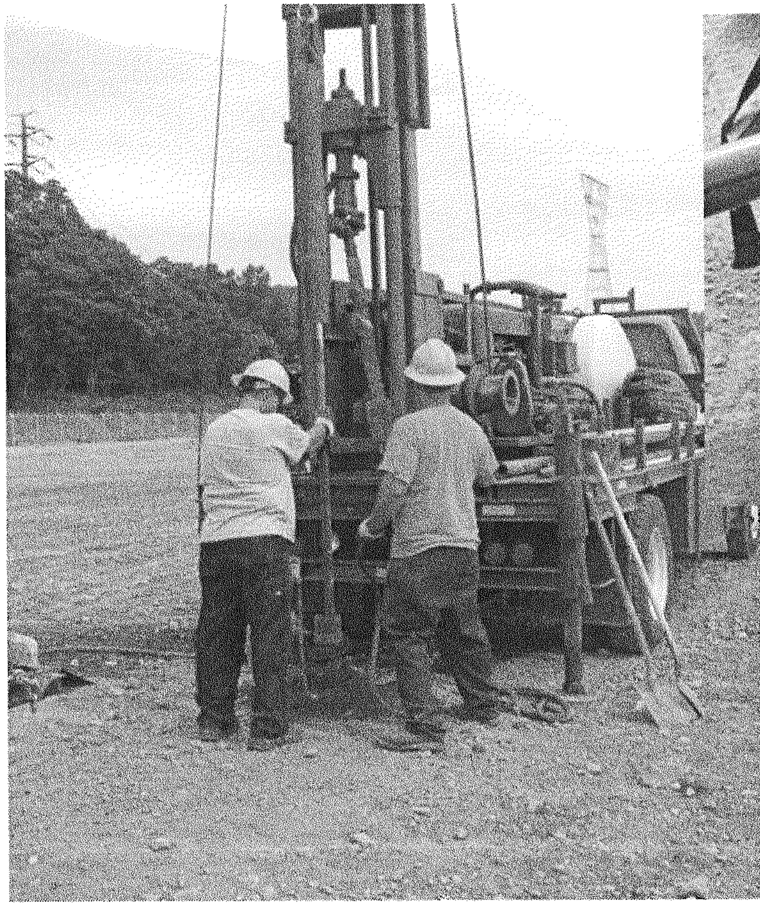
Ecc w/attachment:
Kevin P. Maher, Town Engineer, T-Stony Point
Maureen Leary, Office of the Attorney General
Mauricio Roma, Office of the Attorney General
W. Janeway
M. Caruso
S. Crisafulli
J. Parker
M. Brand
D. Pollock

Region 3/Solid Waste Program
Solid Waste Management Facility Site Visit Report

Facility Name/Location:	Mirant-Lovett former power plant, Town of Stony Point, Rockland County
Date of Site Visit:	August 12, 2009
DEC Staff Present:	Steven Parisio Mauricio Roma, Office of the Attorney General
Facility Representatives:	Burt McCullough, Mirant Corp Michael Pantliano, HDR Tanya Goehring, HDR
Background Information:	The power plant has been demolished and removed from the site. The Consent Order requiring a remedial investigation of the site was executed on July 16, 2009. Installation of groundwater monitoring wells is underway. Several of the monitoring well locations shown in the approved investigative work plan need to be modified due to power lines and other physical constraints encountered in the field.
Purpose of Site Visit:	To discuss changes in monitoring well locations and oversee monitoring well installation.
New Issues and Follow-up Required:	Modified locations for monitoring wells MW-C2, MW-E (couplet), MW-P and MW-O were agreed to in the field. The consultant will need to verify the feasibility of these locations with the driller and obtain permisson for those wells not on Mirant property. (MW-E, MW-P, MW-O). The modified locations for MW-E, MW-P and MW-O are not significantly different from what is shown in the approved work plan. The new location for MW-C2 could be either 75 feet to the east or 150 feet to the south. If the latter location is selected, a deeper well would be needed to reach the target depth because of the higher ground elevation and because of groundwater flow lines which are presumed to be plunging to the south. HDR will provide a GIS shape file showing the modified monitoring well locations. After demolition of the power plant buildings, C&D debris was used as grading material throughout the site. Inspection of the ground surface shows minor but consistent amounts of waste types which are not acceptable for this purpose. This presence of this material is a violation of applicable 6 NYCRR Part 360 regulations which will need to be resolved, along with any soil and groundwater contamination identified during the investigation, when remedial measures are negotiated.
Report prepared by:	S. Parisio
Report Date:	August 12, 2009

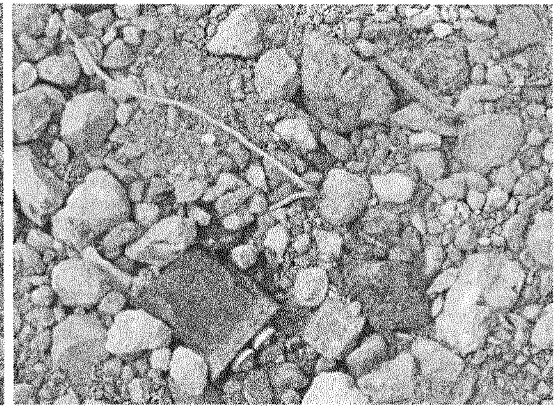


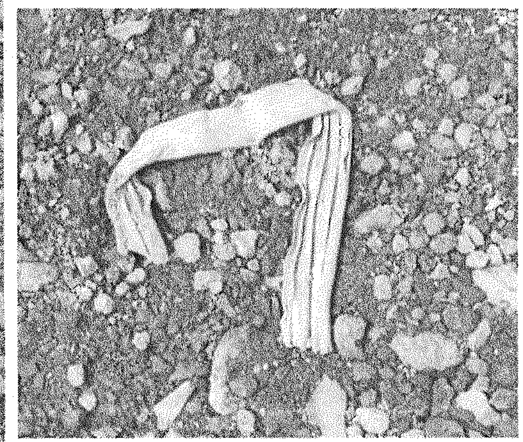
The power plant has been completely demolished and removed from the site along with waste piles previously staged. The site has been regarded and the surface is covered with a layer of C&D debris generated during the building demolition activities. The driller is onsite and is in the process of installing groundwater monitoring wells for the remedial site investigation.



Hollow stem augers are being use to drill borings for monitoring wells. Continuous split spoon samples are being collected and logged by the HDR geologist. A gray clayey silt was encountered below the historical fill layer in the boring for MW-L, at a depth of 12-14 feet. The water table was encountered near the interface between the fill material and native substrate. A PID is being used to screen the samples for volatile organic compounds using the head-space method

A number of monitoring well locations have to be modified because of access problems created by the many power transmission lines which crisscross the site and the setback distance which is required between drilling equipment and the power lines in order to avoid a safety hazard. At the location for monitoring well couplet MW-E, power lines are of the low voltage type and it may be possible to decrease the setback distance by placing a temporary shield over the power lines.

A layer of C&D debris generated by the demolition of the power plant buildings has been used as grading material and spread across the site. Concrete, brick, asphalt pavement and ceramic tile are acceptable for this purpose. (A case-specific BUD was issued for the tile.) As shown in these photos, minor amounts of unacceptable waste types, including metal and wood fragments, wiring and electrical system components, are commingled with the otherwise acceptable fill materials and are dispersed throughout the site.

From the files of CSWAB.org

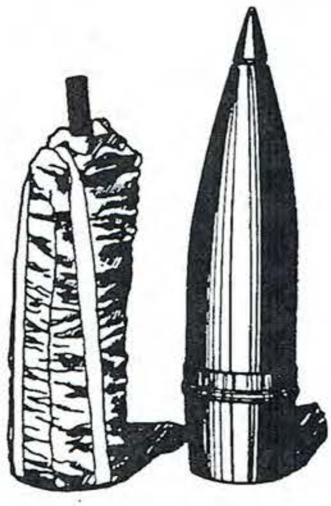
COMMUNITIES IN THE LINE OF FIRE

The Environmental, Cultural, and Human Health Impacts of Military Munitions and Firing Ranges

MILITARY TOXICS PROJECT
June 2002

Communities in the Line of Fire

The Environmental, Cultural, and Human Health Impacts of Military Munitions and Firing Ranges



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"Networking for Environmental Justice"

The mission of the Military Toxics Project is to unite activists, organizations, and communities in the struggle against military pollution, to assure its clean up, to limit the transport of hazardous materials, and to advance the development and implementation of preventative solutions to the toxic and radioactive pollution caused by military activities. The MTP mission is based on mutual respect and justice for all peoples, free from any form of discrimination or bias.

The purpose of the Military Toxics Project is to provide information, education, networking and organizing resources. MTP serves as a bridge and facilitator for organizations concerned with military pollution issues. MTP fosters a relationship of mutual respect and support with its members, networks, and collegiate campaigns around the country. MTP works to assist local communities, not for them but with them. MTP activities focus on both service and organizing efforts. MTP helps member organizations and networks to project their individual voices nationally and internationally.

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of Military Munitions and Firing Ranges

Prepared by the Military Toxics Project

June 2002

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EXECUTIVE SUMMARY

Military munitions pose environmental and human health dangers at each step of their life cycle. Production sites (such as materials processing facilities and ammunition plants), testing sites (such as labs and proving grounds), firing sites (training ranges and areas of conflict), and disposal sites (including burial pits and open burning/open detonation sites) may all contaminate the environment and threaten public health. Dangers at all types of sites exist from both unexploded ordnance (UXO), which poses an immediate safety danger, and toxic munitions chemicals, which pose acute and long-term dangers to human health and the environment.

Up to 25 million acres of land and water at several thousand sites in the U.S. are already contaminated with unexploded ordnance (UXO), toxic explosive compounds and their byproducts, toxic propellants, and heavy metals. More sites are being identified every year. The Department of Defense (DoD) estimates that about 16 million acres of land already transferred to other agencies or the public are potentially contaminated with UXO and munitions constituents. The Army alone has as many as 2,000 sites contaminated with explosives. Federal facilities contain at least thirty large and medium-sized sites containing over one million yards of soil contaminated with the explosive TNT. A 1998 Environmental Protection Agency (EPA) survey of just 266 closed, transferred, and transferring (CTT) and inactive military ranges found that UXO was found on 85% of the ranges. Over 50% of the ranges were known or suspected to contain chemical or biological weapons.

Hundreds of billions of dollars will be required to clean up existing contamination on military training ranges, to say nothing of ongoing pollution from munitions production and use. DoD has estimated its liability for cleanup of UXO at over $100 billion, and the cost to cleanup only closed, transferred, and transferring training ranges at $40-$140 billion. A Navy researcher has estimated that clearing UXO at the Navy's ranges would cost $36 billion, and treating chemical contamination in the soil would cost $33 billion.

Sources of toxic contamination from munitions and their constituents at military testing and training ranges may be divided into several categories. Small arms ammunition contaminated thousands of ranges across the country with lead. UXO poses an immediate safety danger and also corrodes, leaching hazardous munitions constituents into soil and groundwater. Heavy metals and toxic explosive compounds enter the environment when munitions don't detonate completely and when UXO corrode. Propellants contaminate firing positions, impact areas, and neighborhoods downwind. Pyrotechnics release white phosphorus and various other toxins. Unused munitions buried by troops during training or for disposal decay and release their constituents. The open burning/open detonation (OB/OD) of munitions in the open air releases tens of thousands of pounds of metals, explosives, propellants, and other toxins into the air, which travel for miles.

There is abundant and growing evidence of the damage to human health and the environment caused by military munitions and ranges.

- At the Massachusetts Military Reservation on Cape Cod, toxic munitions constituents contaminated the only drinking water supply for 500,000 neighbors of the base, forcing the shutdown of municipal wells. Burning of excess artillery propellant at firing positions was linked to increased lung cancer in people living nearby. Throughout the 1980s, women on the Upper Cape near the base were 64% more likely to be diagnosed with lung cancer than women in the rest of the state.
- Since 1940, the U.S. Navy has used three-quarters of the island of Vieques, Puerto Rico for bombardment, munitions disposal, and other activities. There is strong evidence that heavy metals and other munitions toxins move in the air from the bombing range to the civilian areas. The toxic explosive compound RDX was found in drinking water supplies in civilian areas in the late 1970s. In 2000, excessive levels of mercury were found in the hair and fingernails of 45% of Vieques residents tested. Vegetables and plants growing in civilian areas are highly contaminated with lead, cadmium, and other heavy metals. From 1985-1989, Vieques children aged 0-9 were 117% more likely to contract cancer than children of the same age on the main island of Puerto Rico. Children aged 10-19 were 256% more likely to contract cancer. A 2001 study found that Vieques residents are 73% more likely to suffer from heart disease than residents of the main island, 64% more likely to develop hypertension, 58% more likely to have diabetes, and 18% more likely to be diagnosed with asthma.

- White phosphorus and other toxic munitions constituents contaminated the fragile estuarine salt marsh of Eagle River Flats at Fort Richardson, Alaska. Subsistence fishing grounds have been rendered unusable by the Native Alaskans who have historically used these resources. Munitions contamination killed thousands of waterfowl every year for almost two decades before the Army released even a draft cleanup plan. UXO may exist in, on, and/or under up to 2 million acres of lands and waters outside the current boundaries of the base.
- The Army has trained in Makua Valley on O'ahu, Hawai'i since the 1940s. DoD appropriated the entire valley when World War II broke out, evicting families, but did not return the land after the war as promised. The valley is home to over 40 endangered species, including one found nowhere else on earth. Training and disposal operations at Makua have caused fires that damaged these species; destroyed homes, the local church and Indigenous Hawai'ian temples; and contaminated soil and groundwater. The Army's occupation of Makua denies the community access to these sacred lands.

The regulation (or often the lack thereof) of military munitions and ranges is a complex matter involving federal, state, Tribal, and local laws as well as various governmental and nongovernmental actors. A plethora of agencies and authorities are involved in some aspect of regulation of military munitions and ranges, including: the EPA (both headquarters and the regions); DoD (including the Secretary of Defense, the Defense Environmental Restoration Program, the Department of Defense Explosives Safety Board, and the armed services); other federal agencies; tribes; states; and the public (through various public participation programs, citizen suits, and political action). A variety of laws, regulations, and both formal and informal agreements may be invoked depending on the circumstances.

Regulation of military munitions and ranges occurs primarily under two statutes: the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA, known as the Superfund law); and the Resource Conservation and Recovery Act (RCRA). CERCLA provides for cleanup of contaminated sites and accidents, spills, or other releases of hazardous substances to the environment. RCRA governs solid waste, including hazardous waste, from cradle to grave. Either or both of these laws may be applied to munitions contamination depending on the circumstances. The Safe Drinking Water Act, the Clean Air Act, and the Clean Water Act have also been applied by states and EPA to protect public health and the environment from munitions contamination.

The DoD has historically fought attempts to regulate military munitions and firing ranges, and usually attempts to force any response to munitions contamination to occur under CERCLA, where DoD is the lead agency. DoD oversees CERCLA response actions at its own sites and EPA cannot impose a more protective cleanup process than the DoD chooses. At all CERCLA sites not listed on the National Priority List, EPA cannot act to protect public health and the environment from munitions contamination even in the face of an imminent and substantial endangerment.

In 1992, Congress passed the Federal Facilities Compliance Act, which for the first time subjected federal agencies to RCRA's cradle-to-grave regulation of hazardous wastes. Because RCRA was originally written to govern private sector wastes, Congress directed EPA to write a regulation balancing the need for oversight of military munitions with the DoD's need to train and fight. Instead of balancing these interests, EPA undermined the intent of Congress by completely exempting munitions at active firing ranges from RCRA unless DoD specifically collects them for disposal. EPA's surrender to DoD ensured that millions of pieces of UXO will remain on military ranges leaching their toxic contaminants into the environment. RCRA does allow EPA to issue binding orders to protect public health and the environment from imminent and substantial endangerment by munitions contamination. DoD is seeking a Congressional exemption to remove this authority. Communities near active ranges continue to demand protection from munitions contamination.

Human health, the environment, cultural and historic sites, and subsistence food supplies have already been harmed by munitions contamination. More people are exposed to UXO and chemical contamination from munitions every day because of failures of policy and will by DOD, EPA headquarters, and other federal regulatory agencies. Many states and some EPA regions have taken the lead in addressing the problem, but often without adequate support from Washington. Action is desperately needed to protect communities, ensure cleanups protective of human health and the environment, and prevent additional contamination.

MILITARY MUNITIONS

Military munitions are "all ammunition products and components produced or used by or for DoD or the U.S. Armed Services for national defense and security, including military munitions under the control of the Department of Defense, the U.S. Coast Guard, the U.S. Department of Energy, and National Guard personnel."[1] The term includes a variety of devices used to deliver an explosive, chemical, or pyrotechnic charge to a target. Military munitions include propellants, explosives, pyrotechnics, chemical agents, incendiaries, rockets, missiles, bombs, mortar rounds, artillery shells, small arms ammunition, mines, grenades, cluster munitions, and components of these items. The term does not include nuclear weapons and materials.[2]

Military munitions may be fired or launched (projectiles such as artillery shells and small arms ammunition, mortars, rifle grenades, rockets, and missiles), dropped (bombs, dispensers, and submunitions), placed (mines), or thrown (hand grenades).[3]

This report focuses on conventional munitions (those that are not biological, chemical, or nuclear). However, brief sections on chemical and depleted uranium munitions are included below.

CONVENTIONAL MUNITIONS

Weapons and ammunition have been used for at least two thousand years, but the munitions of today are a far cry from the stones, spears, and other munitions used in past centuries. The weapons of today contain a wide variety of chemical compounds and heavy metals that are hazardous to human health and the environment.

The term conventional munition is used to denote a piece of ordnance which is not nuclear, chemical, or biological. The bulk of a conventional munition is the explosive train, which is composed of a sensitive but not very powerful initiating compound or mixture, which detonates a booster compound, which detonates a less sensitive but more powerful explosive compound such as RDX or TNT (see below). The three parts of the explosive train are generally called the fuze, the booster, and the main charge.

The fuze (as distinguished from "fuse") contains the primer/detonator and is the most critical part of a munition. It must be able to arm and detonate at – and only at – the appropriate time. Fuzes may be designed to initiate the detonation on impact, at a specific time, or in proximity to a target. There are twenty-nine families of fuzes, which are generally classified as either mechanical or electrical.[4] Booster charges, generally consisting of a small amount of moderately sensitive explosive, amplify the force generated by the fuze to ensure complete (or high-order) detonation of the main charge.[5]

The main charge of a munition produces most of the explosive force and requires a large initiating shock to detonate. These explosives are designed to achieve specific effects based on the intended purpose of the mention. Main charges are more stable secondary explosives (see below) such as TNT, RDX, C4, and others.[6]

Primary or initiating explosives – those used to initiate the explosive train and detonate a booster charge or main charge – include lead adzide, lead styphnate, and mercury fulminate.[7]

Secondary explosives – more powerful compounds formed to detonate under specific circumstances and therefore used as boosting explosives or main charges – include TNT (trinitrotoluene); RDX (1,3,5-hexahydro-1,3,5-trinitrotriazine, also known as cyclonite, hexogen, royal demolition explosive, or research development explosive); HMX (octahydro-1,3,5,7-tetranitro-1,3,5,7-tetrazocine, also known as octogen or high melting explosive); and tetryl (2,4,6-trinitro-phenylmethylnitramine).[8] According the the Army Corps of Engineers, "TNT

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[1] National Defense Authorization Act.
[2] U.S. Army Corps of Engineers (USACE) Pamphlet No. 1110-1-18, *Engineering and Design Ordnance and Explosives Response*, April 24, 2000.
[3] Interstate Technology and Regulatory Council (ITRC), *UXO Basic Training Manual*, May 2002, What is a Bomb?, slide 23.
[4] Ibid., slides 11-15.
[5] Ibid., slide 17.
[6] Ibid., slide 18.
[7] USACE, Cold Regions Research and Engineering Laboratory, *Guide for Characterization of Sites Contaminated with Energetic Materials*, Sonia Thiboutot, Guy Ampleman, and Alan D. Hewitt, February 2002, p.2.
[8] Ibid.

and RDX constitute the largest quantity of secondary explosives used in military applications, because they are major ingredients in nearly every munition formulation."[9] These explosive compounds also contain various other substances (such as powdered metals, plasticizing oils, and waxes) used as stabilizers and fillers, and may contain production impurities including various isomers of dinitrotoluene and dinitrobenzene.[10]

The main body of a munition is called the casing, which contains the main explosive charge. Casings are designed to achieve a specific purpose - such as fragmenting to injure or kill personnel, or piercing armor - based on the intended use of the munition.[11]

Propellants are used for propulsion: to move the munition forward. As noted later in this report, propellants are classified as either single-based, double-based, or triple-based (or composite) based on their constituents. All the components of small arms ammunition are usually held together by a cartridge case, while in larger types of ammunition the propellant is packed in separate combustible bags.

INERT, DUMMY, PRACTICE, AND "GREEN" MUNITIONS

Although the terms "inert", "dummy", and "practice" or "training" munition (and more recently "green" ammunition) are often used interchangeably, the munitions themselves and their environmental and human health impacts are distinctly different. These munitions may and often do contain explosive, toxic, or hazardous components.

According to a Department of Defense policy on munitions, "wholly inert" ammunition would mean a munition that has never been employed and has never contained reactive materials. Once an item is employed as a component of a military munition, it is no longer considered "wholly inert."[12]

Dummy munitions are reproductions produced from a variety of materials for many purposes, such as display, instruction, or special tests.[13]

In practice or training munitions, the main charge is replaced by an inert filler. Fillers may be either non-toxic or toxic, and include concrete, wax, sand, plaster, antifreeze (to simulate chemical agents), water, and molasses. Practice munitions do usually contain some type of high-explosive spotting charge, as well as fuzes, primers, igniter charges, and propellants - all of which may contain hazardous or toxic components.[14]

The DoD has recently developed so-called "green" ammunition. The new "green" small arms ammunition is based on tungsten rather than lead. Unfortunately, even this ammunition still requires toxic propellants to be fired. An official at U.S. EPA Region 1 concluded that a 2001 study "indicates that small arms firing over time deposits high levels of those propellants."[15]

CHEMICAL MUNITIONS

In July of 1917, the Bureau of Mines officially started research and development of chemical and biological weaponry at American University in Washington, D.C. Chemical weapons have been manufactured in the form of blistering agents such as mustard gases and lewisite, or nerve agents such as sarin and VX. These agents are disbursable through projectiles or mortar shells, bombs, rockets or missiles, submunitions or bomblets and spray tanks. The military lists twenty-five different types of chemical combinations as some of the agents produced for chemical weapons. Blistering agents cause respiratory tract problems, eye irritation and blistering of the skin. Nerve agents impair breathing, inhibit nerve conduction and can cause nausea and vomiting and ultimately death.[16]

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[9] Ibid., p.4.
[10] Ibid. and Center for Public Environmental Oversight (CPEO), *Toxic Ranges*, April 2002, p.4 and Integrated Publishing, *Gunner's Mate Training: High and Low Explosives*, at http://www.tpub.com/gunners/2.htm.
[11] ITRC, What is Bomb?, slide 22.
[12] Military Toxics Project (MTP), *Toxic Hazards of Practice Ammunition*, p.1.
[13] Integrated Publishing.
[14] MTP, *Toxic Hazards of Practice Ammunition*, and ITRC, What is a Bomb, slides 19 and 20.
[15] Kevin Dennehy, "Hazard found in 'green' bullets," *Cape Cod Time,*, March 29, 2001.
[16] Brigadier General Russ Zajtchuk, M.C. US Army, Editor in Chief, *Textbook of Military Medicine*, Part I, Medical Aspect of Chemical and Biological Warfare, Office of the Surgeon General, , 1997.

Chemical weapons and chemical weapons production facilities are scheduled for destruction under the treaty commonly known as the Chemical Weapons Convention, signed on January 13, 1993 and ratified by Congress on April 25, 1997.

Opposition to incineration of chemical weapons by national groups such as the Chemical Weapons Working group and local community groups has forced the military to explore alternative disposal methods. However, the Army's preferred method of disposal still seems to be incineration because of the emphasis on cost effectiveness.

Stockpile chemical weapons are those stored at arsenals such as Pine Bluff Arsenal. Non-stockpile chemical weapons are any chemical munitions found at sites other than the stockpile sites during cleanup or that have resurfaced where they have been buried and forgotten. Officials at the Department of Defense maintain that records of chemical weapons disposal were not adequately or accurately kept. The military has identified 38 sites where non-stockpile chemical weapons have been discovered, but hundreds of sites are suspected to contain chemical munitions. Sites that have been labeled "no further action" by the military have later been found to contain chemical weapons and related contamination.

The most widely publicized site of chemical weapons contamination is Spring Valley, a neighborhood in Washington, D.C. Chemical weapons contamination was discovered in January 1993, some time after the Army Corps of Engineers had declared the area uncontaminated. Elevated levels of arsenic were found in soil samples in the neighborhood.[17] Low levels of arsenic are known to cause decreased production of red and white blood cells.[18] Two children in Spring Valley died from aplastic anemia, a disease so rare that only about 1,000 deaths per year have been attributed to the condition; yet in one year two were from the same community. Even though scientists have not conclusively linked arsenic and aplastic anemia, the deaths should be cause for precaution. Yet the Agency for Toxic Substances and Disease Registry's (ATSDR) report on contamination in Spring Valley concluded there was **no danger** to community members.

To date, over $50 million has been spent to clean up the contaminants affecting this affluent community where ambassadors and other government officials live. Still, members of the Spring Valley Restoration Advisory Board are critical of some of the methods used for assessment and cleanup. They complain that military contractors took soil samples from sites where landscapers had already replaced contaminated soil. Even though millions of taxpayer dollars have already been spent on this site, community members feel it has not been enough and that the military continues to resist full disclosure and cleanup. This experience demonstrates two critical points. First, that all communities tend to be treated badly by the DoD, regardless of their racial and economic status. Second, that some communities – usually middle-class or wealthy areas with predominantly white populations – usually receive much better responses from DoD and regulatory agencies once problems are brought to light.

Public involvement is crucial for establishing the methods to be developed and used to destroy both stockpile and non-stockpile chemical weapons. The lack of adequate public involvement stems from the fact that the methods used to inform the public are often inadequate and untimely. The Chemical Weapons Working Group, a national oversight, non-governmental organization, provides up-to-date information on their website at www.cwwg.org.

DEPLETED URANIUM

The ultimate recycling horror story of our lifetime is depleted uranium (DU) munitions. Natural uranium is mined and enriched for use in nuclear weapons and nuclear reactor fuel. During the enrichment process, a small amount of the U-235 isotope is extracted. The radioactive waste from this process – composed of 99% U-238 – is called depleted uranium (DU). DU is low-level radioactive waste; it is about 60% as radioactive as natural uranium and has a half-life of 4.5 billion years. It has been accumulating for sixty years and the U.S. stockpile exceeds a billion pounds. The U.S. military began developing DU for use in armor piercing weaponry and tank armor in the 1960s.[19]

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17 *Spring Valley Washington,DC, Project Overview*, U.S. Army Corps of Engineers Baltimore District, http://www.nab.usace.army.mil/projects/WashingtonDC/springvalley/overview.htm.
18 ToxFAQs™for Arsenic, Agency for Toxic Substances and Disease Registry, http://www.atsdr.cdc.gov/tfacts2.html.
19 Dan Fahey, *Case Narrative: Depleted Uranium Exposures*, third edition, Military Toxics Project et al, September 20, 1998.

DU is both toxic and radioactive. As a chemically toxic heavy metal, it can cause kidney and lung damage. As a radioactive substance, it emits alpha radiation, which irradiates powerfully for short distances. Alpha radiation cannot penetrate the skin, but can irradiate lung cells and other cells in the body if inhaled or ingested. U-238 decays into two principal isotopes. These emit beta radiation, which can travel through up to 1,000 cells, and gamma radiation, which can travel through about 200 cells.[20]

The ingestion of minute quantities of uranium in food or drinking water can cause irreparable damage to the kidneys. When uranium weapons burn, corrode or are machined, uranium oxide dust is created. When inhaled, small particles (those less than 5 millionths of a meter, smaller than the eye can detect) can lodge in human lung tissue, exposing the host to a growing dose of alpha or beta radiation. This can cause lung cancer in people of all ages, and is particularly hazardous to children.

When depleted uranium is alloyed with titanium, it becomes extremely dense. It is also "pyrophoric," which means it burns upon impact. Uranium weapons were developed not only because they promised to be effective, but also because the metal itself is very cheap.

The U.S. military has tested DU rounds at various firing ranges around the country and used depleted uranium weaponry in the Gulf War, Bosnia, and Kosovo. The military contends that DU does not damage the environment or cause health problems, but their claims are based on incomplete data. No research by the U.S. military has been conducted in battlefield areas.

A study done by the United Nations Environmental Programme's Depleted Uranium Assessment Team found airborne DU particles at two sites a full two years after the use of DU. The study also pointed out that the danger of contaminated groundwater is high and underlined the importance of monitoring water quality.[21]

Uranium particles were found as far as 26 miles downwind of a production facility in Colonie, New York in 1980. NL Industries Uranium Weapons factory was forced to close. At another manufacturing plant in Concord, Massachusetts radioactive materials have contaminated surface water, ground water and land. Independent testing by a local grassroots organization found DU at 18 times the background level.[22]

Over 200,000 servicemen and women in the Gulf War may have been exposed to toxic and radioactive DU dust by friendly fire, moving through areas contaminated by DU, entering destroyed Iraqi vehicles, and cleaning up battlefields. Military leadership failed to warn, equip, or train cleanup crews to prepare them for DU contamination, despite the existence of DoD training materials. Over 100,000 veterans have diagnosed and undiagnosed illnesses related to the Gulf War.[23]

U.S. Representative Cynthia McKinney of Georgia has sponsored a bill to suspend the use and sale of depleted uranium munitions. The bill would also require studies of the contamination of DU and effects on military personnel and civilians near DU facilities. MTP applauds this type of bill and the efforts of like-minded members of Congress to continue working to protect the health of innocent people in all parts of the world as well as those who serve our country in uniform.

IMPACTS OF MILITARY MUNITIONS ON COMMUNITIES AND THE ENVIRONMENT

Military munitions pose environmental and human health dangers at each step of their life cycle. Production sites (such as materials processing facilities and ammunition plants), testing sites (such as labs and proving grounds), firing sites (training ranges and areas of conflict), and disposal sites (including burial pits and open burning/open detonation sites) may all contaminate the environment and threaten public health. A recent study of techniques to analyze explosives contamination in the environment noted:

> Facilities that may be contaminated with explosives include active and former manufacturing plants, ordnance works, army ammunition plants, naval ordnance plants, army depots, naval ammunition depots,

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[20] Grassroots Action for Peace and MTP, *Health Effects of Depleted Uranium*, Gretel Munroe, p.4.

[21] United Nations Environment Programme, *Depleted Uranium in Serbia and Montenegro: Post-Conflict Environmental Assessment in the federal Republic of Yugoslavia*, UNEP Depleted Uranium Assessment Team, at http://postconflict.unep.ch/

[22] MTP, *Depleted Uranium: Agent Orange of the 90's Another Pentagon Coverup.*

[23] MTP, "The Human Cost of Military Toxics" June 2002.

army and naval proving grounds, burning grounds, artillery impact ranges, explosive ordnance disposal (EOD) sites, bombing ranges, firing ranges, ordnance test and evaluation facilities, etc.[24]

Close to 2,000 Formerly Used Defense Sites (FUDS) are known or suspected to contain unexploded ordnance (UXO).[25] Dangers at all types of sites exist from both unexploded ordnance (UXO), which poses an immediate safety danger, and chemical contamination by munitions constituents, which can pose both acute and long-term dangers to human health.

Up to 25 million acres of land and water at several thousand sites in the U.S. are already contaminated with unexploded ordnance (UXO), toxic explosive compounds and their byproducts, toxic propellants, and heavy metals.[26] More sites are being identified every year. DOD estimates that about 16 million acres of land already transferred to other agencies or the public are potentially contaminated with UXO and munitions constituents.[27] The Army alone has as many as 2,000 sites contaminated with explosive constituents.[28] Federal facilities contain at least thirty large and medium-sized sites containing over one million yards of soil contaminated with the explosive TNT.[29] An EPA survey of just 266 closed, transferred, and transferring (CTT) and inactive military ranges found that UXO was found on 85% of the ranges. Over 50% of the ranges were known or suspected to contain chemical or biological weapons.[30]

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TYPES OF SITES CONTAMINATED WITH MUNITIONS

A great variety of locations have been and continue to be contaminated by the production, testing, use, and disposal of conventional military munitions. Several types of sites contaminated with unexploded ordnance and munitions constituents are described below.

Munitions Production – Because DOD and its contractors operated a variety of munitions production facilities for decades before any environmental regulation existed, these facilities are often highly contaminated with munitions constituents and other substances. The persistence of munitions constituents in soil and groundwater for decades and their mobility over several miles is great cause for precaution at any site where munitions are made, fired, or destroyed.

At the Badger Army Ammunition Plant in Wisconsin, groundwater near the propellant burning ground was contaminated with carbon tetrachloride and trichloroethene, and residential wells were contaminated with carbon tetrachloride at 80 parts per billion (more than 15 times the safe level). Other groundwater contaminants at the facility include tetrachloroethene, 1,1,1-trichloroethane, chloroform, 2,4-DNT, 2.6-DNT,nitrates, cadmium, chromium, lead, sulfates, and benzene. In March, 2001, the munitions constituent

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[24] Bruce A. Tomkins, *Explosives Analysis in the Environment*, Encyclopedia of Analytical Chemistry, 2000, pp. 2402-2441.
[25] U.S. Environmental Protection Agency (USEPA), *Used or Fired Munitions and Unexploded Ordnance at Closed, Transferred, and Transferring Military Ranges*, April 2000, p. 1.
[26] ITRC, Introduction page 4.
[27] U.S. General Accounting Office (GAO), *Environmental Liabilities: DOD Training Range Cleanup Cost Estimates Are Likely Understated*, April 2001, p. 11.
[28] Tomkins.
[29] Ibid.
[30] USEPA, *Used or Fired Munitions...*, April 2000, p. 53.
[31] GAO, p. 5 and p. 13.
[32] MTP, *Smoking Guns: The Environmental, Economic, and Cultural Impacts from Conventional Munitions.*

dinitrotoluene was found in monitoring wells at the fenceline at 3-6 micrograms per liter (ug/l), far above the Public Health Groundwater Quality Standard of 0.5 ug/l.[33]

A variety of other ammunition plants exhibit even worse contamination. Groundwater just west of the city of Grand Island, Nebraska was contaminated with the explosive compound RDX at up to 100 parts per billion (ppb), well above the federal standard of 2 parts per billion for drinking water, by the Cornhusker Army Ammunition Plant, prompting officials to ban residential water wells in the area.[34] In Middletown, Iowa, the Iowa Army Ammunition Plant contaminated groundwater 5-6 miles from the plant with RDX in concentrations 10 to 20 times safe levels.[35] Ground water at the Louisiana Army Ammunition Plant is contaminated with RDX up to 27,000 ppb and TNT up to 25,000 ppb.[36] In 1978 -1979, the Army found the aquifer under the Milan Army Ammunition Plant in Tennessee contaminated with RDX, HMN, 2,4,6-TNT, and 4-DNT. In January 1994, one of three city wells was closed because it was contaminated with RDX at over 4 ppb. The other two wells showed RDX in lower concentrations.[37] There are other examples.

Munitions Testing – Military munitions are tested at various DoD and private labs as well as at military testing ranges, generally called proving grounds. Jefferson Proving Ground (JPG) in Indiana, an Army testing range, was founded in 1940-41 to test bombs, propellants, grenades, and high explosives. The facility – which includes about 55,000 acres – closed in 1995. While various chemical contaminants exist at JPG, the two primary dangers are unexploded ordnance (UXO) and depleted uranium. Over 25 million rounds were tested at JPG from 1941 to 1995. The 51,000-acre Northern Firing Range Area contains an estimated 1.5 million UXO. Cleanup of the firing range will cost from $2 billion to $8 billion and long-term monitoring will be required for at least 25 years. From 1984 to 1992, the Army tested about 220,500 pounds of depleted uranium shells at JPG. There is an estimated 150,000 pound of DU remaining on or in the ground in a 1,250 acre area. The Army proposed to walk away from this contamination, performing no remediation and no monitoring.[38]

Aberdeen Proving Ground (APG) in Maryland, also an Army test range, covers 80,000 acres of land and water on Chesapeake Bay. APG is included on the National Priorities List as one of the most contaminated sites in the U.S. One million UXO litter firing ranges and an additional four million UXO sit in surface waters next to the facility, including Chesapeake Bay.[39] In May 2002, the munitions constituent perchlorate was found in groundwater within 300 feet of the city of Aberdeen's drinking water wells.[40] A recent report concluded that "distinct groundwater plumes consisting of explosive chemicals are more rare [than sporadic detections], but they do exist. In general, they are present at shell washout facilities, munitions disposal areas, munitions cleaning facilities, and bombing fields."[41]

Munitions Firing and Training – Over two million acres of land in the United States are designated as firing range impact areas.[42] The Army and National Guard operate more than fifty firing ranges in thirty-three states. The Navy has more than 21 major ranges covering 2.4 million acres. The Air Force lists 14 munitions-contaminated ranges on over 3.6 million acres.[43] Shells frequently land not only within but outside of range boundaries in public and private lands and waters.

The Massachusetts Military Reservation (MMR) is perhaps the best known example of munitions contamination at a training range. In 1997, EPA Region 1 halted firing at the base because it concluded that contamination of drinking water supplies by munitions constituents posed an immediate and substantial threat to public health. Over 66 billion gallons of the sole drinking water supply for 500,000 people and 70,000

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[33] Citizens for Safe Water Around Badger, *Badger Army Ammunition Plant," 2001; "Army Will Identify and Sample Private Wells Near BAAP for Possible Explosives Contamination*, July 10, 2001.
[34] *Lincoln Journal Star*, "Grand Island forbids water wells west of city," July 26, 2001.
[35] *The Hawk Eye*, "Cleanup officials describe contaminated water plume," May 26, 2001.
[36] USEPA Region 6, *Louisiana Army Ammunition Plant*, updated February 27, 2001, at http://www.epa.gov/earth1r6/6sf/pdffiles/la-army.pdf
[37] Email posed to CPEO Military Environmental Forum newsgroup, "RDX-Milan, TN," July 19, 1998.
[38] See JPG site profile in this report.
[39] MTP, Comments of the Military Toxics Project Conventional Munitions Network before the Environmental Protection Agency regarding the Military Munitions Rulemaking under Section 3004(y) of the Resource Conservation and Recovery Act.
[40] Lane Harvey Brown, "APG, Aberdeen officials devise four ways to keep chemical from wells," *Baltimore Sun*, June 6, 2002.
[41] Cal Baier-Anderson, *Survey of Explosive Chemicals in the Groundwater at Aberdeen Proving Ground*, University of Maryland, Baltimore, for Aberdeen Proving Ground Superfund Citizens Coalition, February 2002.
[42] Military Toxics Project, Comments of the Military Toxics Project Conventional Munitions Network before the Environmental Protection Agency regarding the Military Munitions Rulemaking under Section 3004(y) of the Resource Conservation and Recovery Act.
[43] MTP, *Smoking Guns...*

homes and businesses is contaminated with the explosive RDX and other toxic compounds. Explosives contamination has been found in about half the 200 monitoring wells in the Camp Edwards section of MMR, and the contamination in 53 wells exceeds EPA's health advisory level.[44]

Munitions Disposal - Several decades of negligent disposal of munitions and propellants has littered the U.S. with UXO and explosives chemical contamination. A study of characterization of explosives contamination in the environment noted that "during World War I, World War II, the Korean War, and the Viet Nam War" munitions wastes were often disposed of in "unlined lagoons, which could not contain species such as munition charges."[45] For decades, troops often buried munitions they could not or did not fire in training areas. These caches of munitions stay under the ground until someone stumbles upon them during cleanup, construction, or other activities. Like all other abandoned munitions, they will eventually decay and release components into the soil and groundwater.

It should not be assumed that current techniques for munitions disposal are protective of human health and the environment; in fact, the reverse is true. Current military practices such as open burning/open detonation of surplus munitions and burning of excess propellant create widespread toxic air pollution and, through deposition, soil and groundwater contamination as well.

At the Massachusetts Military Reservation on Cape Cod, troops routinely burned excess propellant in the open air at artillery and mortar firing positions. In a 1991 study, Boston University researchers found an association between these firing positions and lung and breast cancer in people living nearby. The authors recommended and end to the practice of burning propellant.[46]

Various military facilities continue to dispose of munitions by open burning/open detonation (OB/OD), a practice which releases thousands of pounds of toxins into the air at sites across the country. More information on this practice can be found in the open burning/open detonation section of this report.

Formerly Used Defense Sites (FUDS) – FUDS may be any of the types of sites listed above, or other types. Up to 2,000 FUDS are known or suspected to contain unexploded ordnance.[47] Some FUDS – such as the Spring Valley neighborhood in Washington, DC – contain buried chemical weapons and related contamination. Many FUDS doubtless contain soil and groundwater contamination by explosives and propellant compounds, but for the most part no one has looked.

SOURCES OF CONTAMINATION

A full assessment of all sources of environmental contamination, human health impacts, and cultural and economic damage originating from military munitions is well beyond the scope of this report. However, it is possible to identify some major issues. According to a recent report by the Center for Public Environmental Oversight (CPEO), sources of toxic contamination from munitions and their constituents at military testing and training ranges may be divided into several categories.[48]

Small Arms Ammunition – Until very recently, most U.S. small arms ammunition contained lead, which has been found in the environment at various sites. Any of the thousands of small arms ranges scattered across the country is a potential source of lead contamination.

Unexploded Ordnance (UXO) – Five to ten percent of munitions that are fired, launched, or dropped do not explode as designed. These munitions are known as unexploded ordnance or UXO. As noted previously, up to 25 million acres of lands and waters at several thousand sites in the U.S. are contaminated with millions of pieces of UXO. UXO may sit on or just under the surface, several feet underground, on top of boulders, or in trees. UXO poses an extreme safety danger to anyone in the area. Many children and adults both within and

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[44] Letter from Steven Herman, Assistant Administrator, EPA Office of Enforcement and Compliance Assurance, and Timothy Fields, Assistant Administrator, EPA Office of Solid Waste and Emergency Response, to Sherri Goodman, Deputy Under Secretary of Defense (Environmental Security), January 14, 2000 and Melissa Robinson, "Water woes on upper Cape severe," *Associated Press*, May 28, 2002.
[45] Tomkins.
[46] Ann Aschengrau and David Ozonoff, "Upper Cape Cancer Incidence Study Final Report," Boston University School of Public Health, September, 1991.
[47] U.S. Environmental Protection Agency, *Used or Fired Munition...*, p. 1.
[48] CPEO, pp.3-5.

outside the U.S. have been injured or killed by U.S. military UXO. UXO will also corrode and release their constituents - including metals and explosive compounds - into the environment.

Explosives and Metals - Explosive compounds and heavy metals can and often do enter the environment when munitions are produced, when they detonate, and if they fail to detonate as designed. According to the CPEO, "when a conventional high-explosive munition detonates, it releases a large variety of chemical compounds and metals into the environment."[49] Low-order detonations (in which the munition does not explode completely) seem to produce greater amounts of explosives contamination than high-order (complete) detonation, which fully combust the explosive compound.[50] Many explosives and their degradation products are very mobile in groundwater and persist in the environment for long periods of time (at least decades). Even when munitions detonate as designed, heavy metals including lead, cadmium, chromium, nickel, copper, and barium remain and can contaminate air, soil, and water.[51] As noted immediately above and in the UXO section later in this report, munitions that fail to detonate are also major sources of contamination.

Propellants - The chemicals that move explosives forward are called propellants. Military munitions use several types of propellants, including single-based propellants (such as nitrocellulose; these may also contain 2,4-dinitrotoluene), double-based propellants (containing, for example, nitrocellulose and nitroglycerine), and triple-based (nitrocellulose, nitroglycerine, and nitroguanidine).[52] Propellants may be found near and downwind of firing positions and may also be found in impact areas. Open burning/open detonation of propellant may also be a source of contamination. Surplus artillery propellant bags have been routinely burned at artillery firing positions; the propellants may spread some distance through the air. As noted elsewhere, burning of propellant has been associated with health problems in neighbors of the Massachusetts Military Reservation on Cape Cod.

Pyrotechnics - Pyrotechnics are intended to produce light, heat, smoke, or noise, and include tracers, flares, incendiaries, photoflash compounds, and smoke generators. Contaminants originating from pyrotechnics include red and white phosphorus; barium and strontium; perchlorates; polyvinyl chloride; titanium tetrachloride; and hexachloroethane and hexachlorobenzene.[53] The Eagle River Flats range at Fort Richardson, AK - a fragile coastal wetland - is heavily contaminated with white phosphorus from pyrotechnics, which killed thousands of waterfowl each year for over a decade.

Burial Sites - For decades, troops have buried munitions that they can't or don't fire in the field. In some place, large numbers of munitions were consciously buried as a disposal method. These caches of munitions stay under the ground until someone stumbles upon them during cleanup, construction, or other activities. Like all other abandoned munitions, they will eventually decay and release components into the soil and groundwater.

Open Burning/Open Detonation (OB/OD) Sites - Military munitions - as well as large varieties of other military wastes - are routinely disposed of through open burning/open detonation or OB/OD. In open burning/open detonation munitions and other wastes are incinerated at relatively low temperatures with little or no environmental controls. OB/OD sites and the surrounding environment are usually contaminated with munitions constituents, PCBs, dioxins and furans, perchlorate, and other toxic substances. The practice of burning artillery propellant bags noted above is essentially a small-scale and form of OB/OD. The OB/OD section of this report contains more information on these practices.

COMMON CONTAMINANTS AND HEALTH DANGERS

Military munitions contain dozens if not hundreds of toxic and hazardous components that pose extreme dangers to human health and the environment. These substances may be divided into three basic categories: metals; explosive compounds and their degradation products; and propellants. There are a variety of good sources of information on these contaminants, though less is available on the health effects of many. A list of common contaminants and some basic information about each follows.

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[49] Ibid., p.3.
[50] U.S. Army Corps of Engineers, Engineer Research and Development Center, "Characterization of Explosives Contamination at Military Firing Ranges," Thomas F. Jenkins et al, July 2001, p.26.
[51] CPEO, p.4.
[52] USACE, *Guide for Characterization of Sites Contaminated with Energetic Materials*, p.3.
[53] Ibid. and CPEO, p.4.

Common Munitions Constituents[54]

Ammonium Nitrate – May be found in propellant or main charge. Linked to various health effects, including fall in blood pressure, nausea and vomiting, collapse and coma.

Barium Nitrate – An oxidizing compound found in some incendiary mixtures and single-based propellants. Ingestion can result in gastroenteritis, muscular paralysis, decreased pulse rate, and ventricular fibrillation.

Benzene – Used as a starting material for the development of explosives. Linked to aplastic anemia and leukemia.

Cadmium – Heavy metal linked to respiratory tract cancer and kidney damage.

Chromium – Heavy metal linked to respiratory tract cancer.

Composition A, B, and C – Explosive compounds.

DEGDN – A propellant. Causes acute renal failure.

Dinitrotoluene or DNT (including 2,4-DNT and 2,6-DNT) – A component of single-base propellants. A probable human carcinogen. Can cause anemia, liver necrosis, and other effects.

Explosive D – An explosive compound used as a main charge. Causes nausea, vomiting, coma, and seizures, among other effects.

Hexachloroethane – Used in some smoke ammunition. Linked to kidney and liver damage, and is a possible human carcinogen.

HMX – Used as a booster or main charge, and in pyrotechnics and incendiaries. Linked to damage to the nervous system and liver.

Hydrazine – Used in rocket propellants and fuels. A known human carcinogen. May cause liver, kidney, and respiratory damage and harm reproductive organs.

Lead – Lead adzide and lead styphnate are used as primary charges or intiators. Lead damages the central nervous system in children and the peripheral nervous system in adults. Lead adzide can cause anemia, kidney and brain damage, and other effects. Lead styphnate causes widespread organ and systemic effects on the central nervous system, immune system, and kidneys.

Mercury – Mercury fulminate is used as a primary charge or initiator. Linked to renal failure, shock and circulatory collapse, gastrointestinal problems, and other effects.

Nitrocellulose – The primary component of propellants, smokeless powders, rocket fuel, mortar increments, and some explosives. Animal studies suggest it is largely non-toxic to humans, but data is not available regarding human toxicity from drinking water and other exposures.

Nitroglycerine – Component of double- and triple-base propellants, also may be found in main charge. Linked to methemoglobinemia (reduced oxygen content of the blood); impacts circulatory system.

Nitroguanadine – Component of triple-base propellants. Little information is available on its toxicity.

Perchlorates – Common ingredient in propellants; also found in pyrotechnics. Damages the thyroid, which is especially dangerous to fetuses, infants, and children. Also causes acute health effects.

PETN - Booster, main charge, pyrotechnics, incendiary. Irritates the eyes and skin and causes headaches, weakness and drop in blood pressure.

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[54] Sources for this section include. CPEO, pp.7-9. MTP, *Toxic Hazards*.... The Ordnance Shop, at http://www.ordnance.org. U.S. Agency for Toxic Substances and Disease Registry, ToxFAQs. U.S. Army Center for Health Promotion and Preventive Medicine, Military Specific Health Advisories, at http://chppm-www.apgea.army.mil/dwater/standards_and_operations/healthadvisories/index.html. U.S. EPA, "Handbook on the Management of Ordnance and Explosives at Closed, Transferring, and Transferred Ranges and Other Sites," Interim Final, February 2002.

Picric Acid – An explosive used as a main charge. Causes serious problems in the central nervous system, as well as blood cell damage, acute hepatitis, and other effects.

RDX – Very common secondary explosive used as booster or main charge, and in pyrotechnics and incendiaries. Damages the central nervous system and is a possible human carcinogen.

Tetrazene – An initiating explosive. Can cause mucous membrane destruction, pulmonary edema, death.

Tetryl – A common explosive during World Wars I and II as a booster charge; no longer used. Indications of various health effects, including nosebleeds, nausea, and vomiting. Its carcinogenicity has not been studied.

TNT – A very common secondary explosive used as a main charge. Is both mutagenic and carcinogenic; linked to effects on brain, liver, blood, reproductive organs, kidneys, urinary bladder, and eyes.

White Phosphorus - Used in pyrotechnics and incendiaries. Contact with the skin while burning may cause burns and liver, heart, and kidney damage. Causes reproductive effects. Inhalation of vapors may irritate the nose, throat, lungs, skin, eyes, and mucous membranes. Ingestion may cause liver, heart, or kidney damage, as well as vomiting and death.

Health Damage

Obviously a complete exposition of the health dangers posed by all these substances is well beyond the scope of this report. We present some information here to demonstrate the need for precautionary action to prevent additional exposures and clean up existing contamination. It is important to note that the question of cumulative effects from the variety of contamination at military ranges and other munitions contamination sites is never addressed. Scientists have not found a method to determine the effects and document them in an irrefutable manner. Common sense tells us that if we are being exposed to more than one contaminant then the effects of each contaminant must interact with the others in some adverse manner.

A 1998 EPA survey of project managers at 206 closed, transferred, and transferring (CTT) and inactive military ranges found widespread health dangers. The report concluded that:

> contamination resulting from used or fired munitions including UXO is found on almost all ranges.... UXO has been found on 85 percent of the ranges and chemical or biological weapons are known to exist or are suspected at over 50 percent of the ranges. **The risks from contamination resulting from ordnance use are widespread. Ranges in this report potentially pose significant risks to human health and safety because of their proximity to growing surrounding populations....**[55]

There is abundant and growing evidence of the damage to human health and the environment caused by military munitions and ranges. The consequences for communities are very real.

At the Massachusetts Military Reservation on Cape Cod, toxic munitions constituents contaminated the only drinking water supply for 500,000 neighbors of the base, forcing the shutdown of municipal wells. Burning of excess artillery propellant at firing positions was linked to increased lung cancer in people living nearby. Throughout the 1980s, women on the Upper Cape near the base were 64% more likely to be diagnosed with lung cancer than women in the rest of the state.

Since 1940, the U.S. Navy has used three-quarters of the island of Vieques, Puerto Rico for bombardment, munitions disposal, and other activities. There is strong evidence that heavy metals and other munitions toxins move in the air from the bombing range to the civilian areas. The toxic explosive compound RDX was found in drinking water supplies in civilian areas in the late 1970s. In 2000, excessive levels of mercury were found in the hair and fingernails of 45% of Vieques residents tested. Vegetables and plants growing in civilian areas are highly contaminated with lead, cadmium, and other heavy metals. From 1985-1989, Vieques children aged 0-9 were 117% more likely to contract cancer than children of the same age on the main island of Puerto Rico. Children aged 10-19 were 256% more likely to contract cancer. A 2001 study found that Vieques residents are 73% more likely to suffer from heart disease than residents of the main island, 64% more likely to develop hypertension, 58% more likely to have diabetes, and 18% more likely to be diagnosed with asthma.

[55] USEPA, *Used or Fired Munitions...*, p. 53.

The effect of contamination from heavy metals is well documented, but the military continues using them in the munitions that litter every state and many other countries. As noted above, lead is a common component of small arms ammunition, and lead adzide and lead styphnate are used as initiating explosives in various munitions. Lead is known to affect the production of blood cells, reproductive system and behavior. The risks of lead poisoning are greatest for children and pregnant women. Children have a tendency to ingest greater amounts in relation to their body weight. Pregnant women are at risk for miscarriage and damage caused to the developing child. Lead adzide and lead styphnate cause even worse health effects.

The toxic effects of explosive compounds have not been extensively studied. They are becoming more known because of rapidly increasing evidence of widespread environmental contamination by these substances and the exposure of military neighbors to their effects. Even the existing state of the science presents great cause for alarm, especially when combined with the experiences of communities exposed to these toxins.

The range of health problems in humans and animals associated with exposure to these components of munitions include seizures, nausea and vomiting, decreased body weight, liver and kidney damage, anemia, skin irritation, cataracts, and harmful effects on the immune system. Many of these substances are suspected or proven to cause cancer in humans. According to the U.S. Army Corps of Engineers, "secondary explosives [those most prevalent on military sites] are considered carcinogenic and mutagenic."[56]

TNT persists in the environment for at least decades and is very mobile in groundwater. Millions of feet of soil in the U.S. are contaminated with TNT. It is both mutagenic (causes birth defects) and carcinogenic, and may damage the liver, blood, reproductive organs, kidneys, and other organs in humans. It is also thought to cause blood, liver, immune system, and reproductive system damage in wildlife.

RDX can also move long distances in groundwater and may persist there for long periods of time. Groundwater at hundreds of sites across the country is contaminated with RDX. It affects the central nervous system and may cause cancer in humans. In wildlife, RDX may cause neurological damage and harm the liver and reproductive system.

UNEXPLODED ORDNANCE (UXO)

Unexploded ordnance (commonly known as UXO) presents both an immediate safety danger (from explosion) and a long-term health threat (from toxic contamination) to thousands of communities. DoD defines "explosive ordnance" as any munitions, weapon delivery system, or ordnance item that contains explosives, propellants, and chemical agents. UXO consists of these same items after they (1) are armed or otherwise prepared for action, (2) are launched, placed, fired, or released in a way that they cause hazards, and (3) remain unexploded either through malfunction or design.[57]

Up to 25 million acres of land acres on thousands of sites in the U.S. may contain unexploded ordnance, most of which result from weapons testing and troop training activities conducted by the Department of Defense (DoD).[58] This property includes active military bases, formerly used defense sites (FUDS), and base realignment and closure (BRAC) sites. Approximately 16 million acres already transferred from DoD to other federal agencies and the public, and at least 2,000 formerly used defense sites are known or suspected to contain UXO.[59] Fifty-nine military sites listed on the federal Superfund National Priorities List (the most contaminated sites in the country) are contaminated with UXO.

UXO may be found at the ground surface or partially or fully buried as deep as 30 feet or more below the ground surface. Ordnance stabilized by parachute may be completely buried, but the parachute may appear at the surface. UXO may also be found fully intact or in parts or fragments. All UXO, whether intact or in parts, presents a potential hazard and should be treated as such.

UXO may be encountered as isolated munitions or as one of many in a given area. The density and type of UXO in an area depends on the intensity and proximity of troop training and weapons testing activities, the degree of UXO cleanup already conducted, and the types of ordnance used. UXO such as dispensers, missiles, rockets,

[56] USACE, *Guide for Characterization of Sites Contaminated with Energetic Materials*, p.26.
[57] USEPA, Federal Facilities Environmental Management, *Executive Guide to Facility Environmental Management*, November 9, 1995.
[58] ITRC, Introduction page 4.
[59] USEPA, *Used or Fired Munitions...*, p. 1, and GAO, p. 11.

or projectiles may still contain submunitions, or those submunitions may have been scattered across a large area. If any UXO is found, one should assume that other UXO are in the area.[60]

UXO casings may have deteriorated depending on the type of material used, the length of time since deployment, and the elements to which they were exposed. UXO that has deteriorated presents a particular hazard because it may contain chemical agents that could become exposed. Environmental contamination and human health damage caused by corroding UXO are addressed in more detail elsewhere in this report.

In April 1998, the Defense Science Board (DSB) Task Force on Unexploded Ordnance Clearance, Active Range Clearance, and Explosive Ordnance Disposal Programs, composed primarily of retired generals and former Defense appointees, released a report on Unexploded Ordnance Remediation. The Task Force found that:

> Because the suspect sites have not been surveyed, there is great uncertainty about the actual size of the UXO problem. However, even if only 5% of suspect acreage needs cleanup, remediation costs would still be high (possibly exceeding 15 billion dollars) and times would be long (possibly exceeding several decades to complete) using current technologies.[61]

More recently, DoD has estimated the cost to clean up UXO at over $100 billion, and the U.S. General Accounting Office estimates the cost to clean up UXO on just closed, transferred, and transferring ranges at up to $140 billion.[62] The total DoD budget for cleanup for all active facilities and Base Realignment and Closure (BRAC) sites is approximately $1.7 billion per year.

The DSB Task Force found that the Department of Defense, as a whole, lacked specific cleanup goals, objectives, or management plans for UXO, and recommended the establishment of a focal point within the Environmental Security office to address the Department's unmet management responsibilities. The report suggested that the Deputy Undersecretary of Defense for Environmental Security [DUSD(ES)] "take the policy lead for DoD UXO remediation efforts, in coordination with other relevant DoD components." The report also endorsed stable funding. It said the Environmental Security office should:

> Identify the needed Congressional actions that will drive UXO requirements for an improved DoD program, including the likely FUDS/BRAC sites with high Congressional priority and those closed ranges that should be converted to more productive uses.... Therefore, we recommend the establishment of a closed range UXO remediation line item in the Environmental Security budget. This line item will offer the DoD and Congress the opportunity to determine the proper level of effort for UXO response. . . it will make it easier to apply relative risk principles to the allocation of UXO project money without comparison to totally different kinds of risks.[63]

The Task Force actually suggested two UXO line items: one for remediation and one for research, development, test, and evaluation (RDT&E).[64] DoD has recently taken some steps to address safety hazard posed by UXO, if not the chemical contamination issues, and Congress has also become involved.

In late 2001, Congress included several provisions on UXO championed by Representatives Earl Blumenauer (D-Oregon) and Bob Riley (R-Alabama) in the 2002 Defense Authorization Act. Congress mandated that DoD complete by May 31, 2003 and update annually an inventory of unexploded ordnance, discarded military munitions, and munitions constituents at defense sites other than operational ranges. DoD will be required to consult with states and tribes to develop a process to prioritize these sites, and provide an opportunity for public comment on the prioritization protocol. The legislation bars DoD from using its prioritization of sites to avoid regulation. Other provisions established program elements in DoD's budget to track cleanup of UXO, discarded munitions, and munitions constituents, and required DoD to produce both an interim and final report on projected costs for the cleanup of ordnance at both operational ranges and at other defense sites (including former ranges and disposal sites).[65] For the first time, Congress and the public will be able to find out the true

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[60] USEPA, *Executive Guide to Facility Environmental Management*.
[61] U.S. Department of Defense (DoD), Office of the Under Secretary of Defense for Acquisition and Technology, *Report of the Defense Science Board (DSB) Task Force on Unexploded Ordnance Clearance, Active Range Clearance, and Explosive Ordnance Disposal Programs*, April 1998.
[62] GAO, p. 5 and p. 13.
[63] U.S. DoD, *Report of the Defense Science Board....*
[64] The Center for Public Environmental Oversight & the Pacific Studies Center. Citizens' Report on the Military and the Environment, *Landmark Report on Unexploded Ordnance Remediation*, May 1998.
[65] Center for Public Environmental Oversight, "U.S. EPA vs. DoD," Lenny Siegel, October 2001.

scope of UXO and munitions chemical contamination at non-operational DoD ranges and track spending to remedy these dangers.

OPEN BURNING/OPEN DETONATION (OB/OD)

Open burning/open detonation (OB/OD) is a form of uncontrolled incineration. Open detonation and open burn operations are used to destroy excess, obsolete, or unserviceable munitions and energetic (i.e., explosive) materials, including UXO and surplus propellant. In open burning, materials are destroyed by self-sustained combustion after being ignited. In open detonation, a charge is used to detonate and destroy the munitions and energetic materials. In the past, these operations occurred at land surface or in pits. OB/OD is more acceptable when munitions, such as those recovered in a firing range, are unsafe to move. Pressure on DoD and EPA to stop OB/OD activities has been increasing, and the practice may eventually be abandoned, at least for large-scale munitions disposal. Some minimal environmental controls have recently begun to be used for some OB/OD operations. However, EPA has concluded that "harmful emissions may pose human health and environmental risks and are difficult to capture sufficiently for treatment."[66] Safer alternatives for munitions disposal – including the controlled detonation chamber (the "Donovan" chamber) already exist.

During the process of open burning and detonation of munitions, airborne contamination is dispersed into the atmosphere and toxic residues are left in the soil. According to a report by the Weizmann Institute of Science in Israel and the University of Florida, OB/OD causes serious contamination of the surrounding environment.[67] Burning common explosives produces toxic gases such as nitric oxide and carbon monoxide. In an attempt to measure and identify emissions from the burning of propellants, Sandia National Lab conducted the so-called "Bang Box" tests. Emission factors from these tests included toxic and carcinogenic substances such as carbon monoxide, methane, benzene, 2,4 dinitrotoluene (2,4 DNT), 2,6 dinitrotoluene (2,6 DNT) and nitrogen oxides.[68]

At many training and firing ranges, the areas where OB/OD operations took place are the most contaminated sites on the ranges. According to EPA, underlying soil and groundwater may become contaminated with byproducts of incomplete combustion and detonation.[69] These contaminants may include explosive and propellant compounds, heavy metals, dioxins, and other hazardous substances.

There is little doubt that people living near military ranges and disposal sites have been exposed to emissions and particulates from OB/OD operations. Residents miles downwind of OB/OD sites are at risk for increased cancers and respiratory problems. Two communities on opposite sides of the country - but both in proximity to OB/OD activities - exhibit high incidences of lung cancer.

At the Massachusetts Military Reservation (MR) on Cape Cod, a Boston University study found a relationship between residence proximity to the artillery training area, where propellant bags were burned, and the risk of lung and breast cancer. Other OB/OD operations also occurred at the base over the past several decades. Studies of open detonation operations at MMR found that explosive residues are emitted into the air during these actions, and are commonly deposited on the soil in concentrations significantly above action levels.[70] From 1982-1990, women living in census tracts near the base were 64% more likely to be diagnosed with lung cancer than women in the rest of the state.

In Utah, open detonation and burning has been used for years for the disposal of military propellants, explosives and pyrotechnics at Toole Army Depot. While the state of Utah has some of the lowest cancer rates in the country, a comparison of cancer rates shows the incidence of lung cancer in Toole County, home of the Depot, is well above the state average from 1966 to 1990.

In the early 1980's, dozens of military installations were granted "interim" status under RCRA Subpart X (see appendix), which allowed them to conduct open burning and open detonation (OB/OD) of munitions without RCRA permits. Regulations promulgated in 1987 required these facilities to apply for permits in 1988. Any facility that did not apply for a permit by November 8, 1988 automatically lost its interim status on November

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[66] U.S. EPA, *Handbook...*, p.5-7.
[67] *Chemical and Engineering News*, August 15, 1994, p.33.
[68] Citizens for Safe Water Around Badger, Letter to Beth Fiore, Wisconsin Division of Health, November 2, 1995.
[69] U.S. EPA, *Handbook...*, p.5-7.
[70] Ibid.

8, 1992.[71] At least one facility, the Sierra Army Depot (California's largest toxic air polluter, according to recent EPA data), did not apply for a permit in 1988 as required, but continued OB/OD through the 1990's without action by the EPA.[72] Since 1988, seventy installations have submitted Subpart X permit applications for Open Burning/Open Detonation (OB/OD) units. As of mid 1998, EPA was still allowing many OB/OD sites to operate under interim status and treat hazardous waste (munitions) by burning and detonation at low temperatures in the open with little or no pollution controls.[73]

IMPACTS ON CULTURAL AND HISTORIC SITES

Indigenous communities and their cultural, spiritual, and historic sites are often heavily and uniquely impacted by military munitions. Because many Indigenous cultures are inextricably linked to the environment and traditional practices, military contamination of food sources can devastate the way of life of these peoples. Impacts on subsistence food supplies are addressed in the section on contamination of these resources. We present two examples of egregious impacts on cultural, spiritual, and historic sites here.

The Hawai'ian island of Kaho'olawe – 28,000 acres about six miles southwest of Maui – was inhabited for over a thousand years by native Hawai'ians before the U.S. military for target practice at the outset of the Second World War. Kaho'olawe was an important training ground for kahuna (priests) and navigators. Decades of Navy training denied Native Hawai'ians access to the island, which is a wahi pana (sacred place) and a pu'uhonua (place of refuge and spiritual regeneration), destroyed religious and cultural sites, and contaminated the land and water with tens of thousands of pieces of unexploded ordnance. After a decades-long struggle led by the Protect Kaho'olawe 'Ohana, the island was formally returned to the State of Hawai'i in 1994 in anticipation of its eventual transfer to a Native Hawai'ian sovereign entity. The 'Ohana continues to lead the struggle to hold the DoD to its (and Congress') commitments to cleanup UXO on the island and return it to a state "reasonably safe for human habitation."[74]

The Army has trained in Makua Valley on O'ahu, Hawai'i since the 1940s. DoD appropriated the entire valley when World War II broke out, evicting families, but did not return the land after the war as promised. Makua contains Indigenous Hawai'ian sites and artifacts – including *heiau* (temples), burial grounds, and fishermen's shrines – dating back 900 years. Fifty years of training, firing, and munitions disposal have destroyed homes, the local church and Indigenous Hawai'ian temples, and contaminated soil and groundwater. The Army's occupation of Makua continues to deny the community access to these sacred lands.[75]

CONTAMINATION OF FOOD SUPPLIES AND DRINKING WATER

Munitions chemical contamination is insidious. It can work its way into water supplies and into the food chain, poisoning people who eat contaminated plants and animals, drink contaminated water, or even eat plants from gardens watered with contaminated water.

Individuals and communities that eat fish that they catch, game that they hunt, and plants that they gather or grow may also be exposed to munitions toxins. Certain toxins and radioactive isotopes accumulate in plant and animal flesh and move up the food chain until they reach human bodies. These substances tend to persist in animal and human tissue for long periods, accumulating to harmful levels over months and years. Indigenous communities and other populations that eat large amounts of fish and other local plants and wildlife are most exposed. Many Indigenous communities depend on wild fish, plants, and game for subsistence and also for the preservation of traditional ways of life. The end of subsistence fishing, hunting, and gathering means the end of these communities and their culture. They may be wiped out by munitions contamination of their food supply.

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[71] USEPA, Office of Solid Waste and Emergency Response, Directive 9489.00-2, April 22, 1987.
[72] Responses to Freedom of Information Act Request filed by Military Toxics Project, August 1998.
[73] U.S. Army Environmental Center, "Open Burning/Open Detonation Guidance Documents", at http://aec.army.mil/prod/usaec/eq/comp/hazman/openburning.htm, August 3, 2000.
[74] Kaho'olawe Island Reserve Commission, *The Kaho'olawe Island Reserve*, at http://www.state.hi.us/kirc/main/home.htm, and Protect Kaho'olawe 'Ohana, "Comments on Kaho'olawe Cleanup Plan", June 5, 1998, at http://www.kahoolawe.org/cleanup.html.
[75] Kyle Kajahiro, "Resisting the Military Juggernaut in Hawai'i," in Voices Unidas, Southwest Organizing Project, Fall 2001, pp.15-17, and Sally Apgar, "Army, Hawaiian Group Reach Deal on Training," *Washington Post*, October 7, 2001, p.A03.

Unexploded ordnance (UXO) and munitions constituents contaminated – and still contaminate – traditional hunting and fishing grounds on and around Fort Richardson, Alaska. Since 1949, the Army has fired ordnance including high explosives, smokes, and illumination flares into the sensitive estuarine salt marsh of Eagle River Flats, leaving 10,000 pieces of UXO. These munitions continue to leach toxic substances into the environment, and pose a safety danger to hunters and others who enter the area because no physical barriers prevent access. Both UXO and chemical contamination has precluded subsistence hunting and fishing by area tribes. White phosphorus contamination in the Flats killed thousands of waterfowl each year for almost two decades before the Army released a draft cleanup plan. UXO may also exist in, on, and/or under up to 2 million acres **outside** the current boundaries of Fort Richardson. There has never been a systematic attempt by the Army to identify areas likely to contain UXO or to investigate or remedy either the safety or toxic hazards either within or outside the current boundaries of the base.[76]

Mercury released by operations at the Badger Army Ammunition Plant contaminated fish eaten by neighbors of the site. In late 2000, the Wisconsin Bureau of Environmental Health sent letters to at least 21 homes near the plant warning residents who had at some point received permits to fish at the ballistics pond on the plant property that the fish were contaminated with mercury. The letter noted that "the Army recommends to permit holders that they eat no fish from the ballistics pond."[77] Plant neighbors had caught and eaten fish from this pond for years before the mercury contamination was finally discovered. In water, mercury becomes methylmercury, an unusually toxic form that accumulates in fish tissue. Methylmercury can cause nuerological damage and death in humans in small amounts. Mercury crosses the placenta to contaminate fetuses in the womb, and can damage memory and other skills in exposed children. There is no way to remove mercury from fish tissue before consumption.[78]

Munitions constituents have contaminated drinking water wells and supplies in several communities around the country, exposing people to dangerous toxins. Many munitions constituents and their degradation products are mobile and persistent in groundwater and pose severe threats to human health. There is little documentation easily available to the public about the scope and severity of contamination of drinking water supplies near military firing ranges across the country. We do know that the scale of munitions contamination of soil and groundwater is very large; we just don't know how many drinking water supplies have been contaminated, primarily because adequate testing and monitoring has never been done.

At the Massachusetts Military Reservation (MMR) on Cape Cod, decades of munitions firing and disposal contaminated the sole source drinking water aquifer for half a million permanent and seasonal residents of the Upper Cape. A January, 2000 EPA letter to the DoD notes that:

> There is now ample evidence that military munitions used and disposed of during training at Camp Edwards have contaminated parts of the Sagamore Lens with RDX and other toxic compounds – evidence that DoD, in its objection to EPA's April 1997 SDWA order restricting training at Camp Edwards, asserted would not be found. In fact, 10 percent of the monitoring wells installed as part of the groundwater study conducted pursuant to the February 1997 SDWA order show RDX concentrations above EPA's health advisory.[79]

More recently, perchlorate was found in drinking water supply wells in the town of Bourne, forcing the shutdown of several wells. To date, explosives contamination has been found in about half of the 200 monitoring wells installed on Camp Edwards (one part of MMR); contamination in 53 exceeds EPA's health advisory levels. Because of pollution from MMR, the Upper Cape could face a drinking water shortfall of 11 million gallons a day by 2020.[80]

Neighbors of the Trojan Explosives plant in Mapleton, Utah have been exposed to RDX and nitrates in their drinking and irrigation water. Neighbors of the facility used contaminated water to drink, shower, water

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[76] See Fort Richardson site profile later in this report and "Alaska Native and Public Interest Organizations Work to Protect Public Health and the Environment at Fort Richardson, AK," available from the Military Toxics Project.
[77] Letter from Henry Nehls-Lowe, Epidemiologist, Wisconsin Bureau of Environmental Health, to holders of fishing permits for the ballistics pond at the Badger Army Ammunition Plant, October 10, 2000.
[78] National Institutes of Health, *Mercury Health Hazards*, at <http://www.nih.gov/od/ors/ds/nomercury/health.htm>.
[79] Letter from Steven Herman, Assistant Administrator, EPA Office of Enforcement and Compliance Assurance, and Timothy Fields, Assistant Administrator, EPA Office of Solid Waste and Emergency Response, to Sherri Goodman, Deputy Under Secretary of Defense (Environmental Security), January 14, 2000.
[80] Melissa Robinson, "Water woes on upper Cape severe," *Associated Press*, May 28, 2002.

gardens, fill swimming pools, and fill ponds from which they ate fish. Several neighbors have been diagnosed with non-Hodgkins lymphoma. Plaintiffs in several lawsuits allege negligent practices by Trojan and charge that the company knew about the water contamination as early as 1981 but did not warn the neighbors. In 1986, the company found RDX in one of its groundwater monitoring wells at 10.7 parts per million (5,500 times the federal health advisory level).[81] Private wells near Trojan showed concentrations of RDX at 23 parts per billion (ppb), 16 ppb, and 12 ppb. The federal health advisory level is 2 ppb.[82]

IMPACTS ON ENDANGERED SPECIES AND THE ENVIRONMENT

While military munition impacts on human health are often most reported, damage to plants, wildlife, and the environment is also widespread and equally significant. Damage to air, soil, and water may persist for generations, or in the case of radioactive contamination, for thousands or millions of years. Large areas of land and water in the U.S. contaminated with UXO and toxic substances from military munitions is unsafe for housing, schools, and many other uses, and will be for the foreseeable future. Military munitions contamination adds significantly to the heavy load of toxic contamination already borne by the environment that sustains all life on the planet. Endangered and threatened plant and animal species - already pushed to the brink of extinction by human activities - are in some cases danger of being pushed over the edge by military munitions.

Makua Valley in Hawai'i is home to over 40 endangered species, including one found nowhere else on earth. Forty years of Army training and disposal operations at Makua have wreaked havoc on these species. Native forest has been destroyed by over 270 fires caused by military activities.[83]

White phosphorus and other toxic munitions constituents contaminated the fragile estuarine salt marsh of Eagle River Flats at Fort Richardson, Alaska. As noted above, subsistence fishing grounds have been rendered unusable by the Native Alaskans who have historically used these resources. Munitions contamination also killed thousands of waterfowl every year for two decades before the Army released even a draft cleanup plan.[84]

As readers will doubtless gather from the rest of this report, there are countless other examples of damage to natural resources and species caused by military munitions and firing ranges.

REGULATION OF MILITARY MUNITIONS AND RANGES[85]

The regulation (or often the lack thereof) of military munitions and ranges is a complex matter involving federal, state, Tribal, and local laws as well as various governmental and nongovernmental actors. A plethora of agencies and authorities are involved in some aspect of regulation of military munitions and ranges, including: EPA (both headquarters and the regions); DoD (including the Secretary of Defense, the Defense Environmental Restoration Program, the Department of Defense Explosives Safety Board, and the armed services); other federal agencies; tribes; states; and the public (through various public participation programs, citizen suits, and political action). A variety of laws, regulations, and both formal and informal agreements may be invoked depending on the circumstances. The currently shifting landscape of statutory and regulatory authority, as well as the large cast of actors on these issues, makes it difficult to present a straightforward or unequivocal analysis of when, where, how, and why munitions and ranges are - or are not - regulated. The laws applied and the parties involved often differ from site to site. This section attempts to address in a reasonably structured manner the major laws, regulations, and issues involved.

Regulation of military munitions and ranges occurs primarily under two statutes: the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA, known as the Superfund law); and the Resource Conservation and Recovery Act (RCRA). CERCLA provides for cleanup of contaminated sites and

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[81] Brent Israelson, "Explosives Company to Go on Trial as Judge Refuses to Dismiss Lawsuit," *Salt Lake Tribune*, March 3, 2001.
[82] Brent Israelson and Mark Eddington, "Mapleton lawsuits claim contamination caused illnesses," *Salt Lake Tribune*, April 15, 2001.
[83] Kajahiro.
[84] See the Fort Richardson site profile later in this report.
[85] Sources for this section are as follows. Fort Ord Toxics Project, "Fort Ord Toxics Project's Law Suit," January 12, 1998. Joshua E. Latham, "The Military Munitions Rule and Environmental Regulation of Munitions," Boston College Environmental Affairs Law Review, Vol. 27, No.3, 2000. ITRC. MTP, *Defend Our Health*. USEPA, *Handbook*....

accidents, spills, or other releases of hazardous substances to the environment. RCRA governs solid waste, including hazardous waste, from cradle to grave. Either or both of these laws - in addition to others - may be applied to munitions contamination depending on the circumstances.

Two legal issues often prevent application of federal environmental and public health laws such as RCRA and CERCLA to federal agencies, including the Department of Defense. The doctrine of sovereign immunity - literally a remnant of the days when kings and queens were above the law - means that federal laws cannot be applied to the federal government itself unless Congress specifically, completely, and unequivocally waives that immunity. The unitary executive theory - adopted by the U.S. Department of Justice - holds that because the entire Executive Branch of the federal government is one entity headed by the President, federal agencies cannot bring suits against each other. One of these doctrines - sovereign immunity - is universally accepted throughout the U.S. legal system, and the other - unitary executive - is simply a position adopted by the Executive Branch but not universally recognized elsewhere. Taken together, these two legal doctrines prevent any application of either the routine or enforcement provisions in environmental and public health laws to the DoD unless Congress has clearly and unequivocally waived federal immunity **and** explicitly granted EPA or other relevant agencies the authority to issue binding orders or take legal actions against other federal agencies to enforce the law.

DEPARTMENT OF DEFENSE EXPLOSIVES SAFETY BOARD

The Department of Defense Explosives Safety Board (DDESB) was established by Congress in 1928 to provide objective advice to the military on explosives safety and to prevent hazardous conditions arising from the explosive and environmental effects of military munitions.[86] The DDESB establishes and oversees implementation of explosive safety requirements throughout the DoD, and is authorized to resolve issues between explosives safety standards and environmental standards.[87] While DDESB directives are binding on DoD munitions cleanups in addition to any other regulatory requirements, like the policies of the Defense Environmental Restoration Program (DERP - see below) they are not enforceable by other federal agencies, states, tribes, or the public.

COMPREHENSIVE ENVIRONMENTAL RESPONSE, COMPENSATION, AND LIABILITY ACT (CERCLA)

Enacted in 1980 and amended in 1986, CERCLA created broad federal authority for systematic identification and cleanup of highly contaminated sites that threaten human health and the environment. The blueprint for CERCLA implementation is the National Oil and Hazardous Substances Pollution Contingency Plan (generally referred to as the National Contingency Plan or NCP). CERCLA provides for action when there is a release or threat of a release of a hazardous substance into the environment, especially when the release or threat of release poses an imminent and substantial endangerment to public health. CERCLA requires investigation of releases or potential releases, as assessment of risk to the public, and, if there is an unacceptable threat to the environment or human health, consideration of cleanup options, documentation of decisions, and finally actions to address the contamination.

CERCLA authorizes two kinds of actions to respond to contamination (generally known as "response actions"):

- Short-term **removal actions**, taken to address releases or threatened releases requiring prompt response, such as emergency situations.
- Long-term **remedial actions**, taken to permanently and significantly reduce the dangers associated with releases or threats of releases of hazardous substances that are serious, but not immediately life threatening.

Sites addressed under CERCLA may be divided into two categories:

- NPL Sites - Highly contaminated and dangerous sites are placed on the National Priority List (NPL). Both removal (short-term or emergency) and remedial (long-term) actions occur at these sites.

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[86] USEPA, *Used or Fired Munitions...*, p.47.
[87] USEPA, *Handbook...*, p.8.

- Non-NPL Sites – Sites not placed on the NPL are, rather obviously, known as non-NPL sites. Such sites owned by private parties are usually addressed only be removal actions, though in some cases remedial actions occur as well. Both removal and remedial actions occur at federal non-NPL sites.

Although CERCLA is administered and primarily implemented by the federal government, states, tribes, and the public play a significant role in CERCLA implementation. Recently some states – especially Colorado – have been pursuing more active roles in investigation and cleanup at military CERCLA sites in order to protect the health and welfare of their residents. This development has tended to encourage more protective cleanups because states can step in when EPA – often under national pressure from DoD – fails to act.

Congress appeared to waive federal sovereign immunity under CERCLA in Section 120, which clarified that:

- Federal agencies are subject to CERCLA in the same manner as nongovernmental entities.
- Federal agencies are bound by guidelines, regulations, and other criteria applicable to assessments, evaluations, and remedial actions in the same manner as other entities.
- An interagency agreement (IAG, often referred to in this context as a Federal Facility Agreement, or FFA) must be signed between EPA and the federal agency responsible for each site placed on the NPL to ensure prompt cleanup.
- EPA (at NPL sites) or the relevant state (at non-NPL sites) must concur with uncontaminated property determinations made by DoD.

However, the CERCLA sovereign immunity waiver is not clear and unequivocal enough to accomplish its goal. In reality, EPA, states, tribes, and the public cannot hold DoD accountable to CERCLA in the same manner as other entities. Problems in the waiver and various Executive Branch orders and decisions often frustrate the application of CERCLA at military sites.

The CERCLA sovereign immunity waiver passed by Congress is not clear enough to allow full application of the law to federal facilities. Courts have ruled that CERCLA does not waive federal immunity from state laws at sites no longer owned or operated by federal agencies, even when those agencies clearly contaminated the site. At least one court has ruled that the waiver does not allow civil penalties to be awarded for past violations by a federal agency under CERCLA. DoD claims that CERCLA does not waive immunity from state cleanup standards, but merely requires it to comply with the standards it deems appropriate. States may attempt to enforce their standards, but the lack of an unambiguous waiver and federal resistance usually force states to give up or undertake time-consuming and expensive litigation.

The CERCLA statute grants the authority to implement the law to the President of the U.S. President Reagan's Executive Order (EO) 12580 empowered EPA to manage most of the CERCLA program, but delegated to DoD the authority at most military CERCLA sites. At sites listed on the NPL, DoD is the "lead agency," meaning that it has responsibility for CERCLA responses at its own sites. EPA must concur with DoD's decisions about the site (formalized in a ROD, or Record of Decision), but cannot impose a more protective cleanup process as it can at private sector sites. At non-NPL sites (the vast majority of sites that require a response under CERCLA), DoD must simply **consult** with EPA, states, and tribes, but retains complete authority to make decisions on its own in all situations except removal actions that are emergencies. EO 12580 prevents EPA from ordering a cleanup at a military facility without approval by the Department of Justice (charged with representing both agencies).

EPA's authority under CERCLA Section 106 to act to protect public health and the environment in the case of an imminent and substantial endangerment is critical to fulfilling CERCLA's promise that the dangers posed by environmental contamination will be promptly addressed. Unfortunately, EPA cannot utilize even this essential authority at most DoD sites. The terms of a Clinton Executive Order and a 1998 memorandum of understanding between EPA and other federal agencies cripples EPA's ability to protect the public from munitions contamination under CERCLA. At non-NPL sites, the authority to address imminent and substantial endangerment of human health and the environment under Section 106 of CERCLA resides with the DoD itself rather than EPA. The problem with this arrangement is clear: even in the face of an immediate danger to public health, EPA cannot force DoD to act under CERCLA. The health of the environment and DoD's neighbors at these sites depends solely on its voluntary cooperation. The history of DoD's failures to protect the public does not provide great cause for optimism in these cases.

Defense Environmental Restoration Program

Congress amended CERCLA in 1986 by passing the Superfund Amendments and Reauthorization Act (SARA), with the intent of clearing up complex areas of the law that had been encountered in its first six years. SARA required that CERCLA actions take into consideration existing state and federal regulations and laws. It increased state involvement in every phase of the program. Possibly the most important change was the requirement that EPA revise its Hazard Ranking System to more accurately reflect the relative degree of risk to human health and the environment posed by uncontrolled hazardous waste sites placed on the NPL.[88]

SARA Section 211 formally established the Defense Environmental Restoration Program (DERP) by statute (DoD had operated an installation restoration program since the mid-70s). The law requires that the DERP be carried out in consultation with EPA, states, and tribes, and that state, tribal, and local governments be able to comment on response actions (except in emergencies). Three goals were established for the DERP:

- Cleanup of contamination consistent with CERCLA Section 120;
- Correction of environmental damage that poses an imminent and substantial danger to public health and the environment;
- Demolition and removal of unsafe building and structures.

It is critical to note that other agencies, states, tribes, and affected communities have little or no independent recourse if military facilities fail to comply with DERP requirements. This situation renders DERP requirements essentially unenforceable when voluntary compliance fails.

RESOURCE CONSERVATION AND RECOVERY ACT (RCRA)[89]

RCRA (pronounced "rick-rah") created a federal program to control solid waste (including hazardous waste) from generation to disposal (from "cradle to grave"), including generation, transportation, treatment, storage, and disposal. RCRA regulates not only day to day management of wastes but also the cleanup of releases to the environment, whether intentional or accidental. The law focuses only on active and future facilities and does not address abandoned or historical sites (which are regulated under CERCLA). Subtitle C of RCRA created a strict system of permits, transportation manifests, and treatment, handling, and disposal standards. Subtitle G authorizes EPA to issue binding orders or seek court injunctions to protect health or the environment from "imminent and substantial endangerment" by hazardous waste, and empowers citizens to sue for injunctions if EPA fails to act.

The definition of solid waste in the RCRA statute passed by Congress in 1978 is broader than the definition adopted by EPA in the regulations implementing the law. Materials can only be regulated as hazardous waste under the strict Subtitle C system if they meet the narrower EPA definition. However, citizen suits and EPA orders and actions taken under the Subtitle G "imminent and substantial" endangerment provisions occur under the broader statutory definition.[90] These distinctions become critically important in the regulation of military munitions and firing ranges.

Because the DoD resisted compliance with RCRA, in 1992 Congress passed the Federal Facilities Compliance Act, or FFCA (Section 6001, as amended by PL 102-386). The FFCA clarified that federal facilities are subject to all provisions of federal, state, interstate, and local hazardous waste laws and regulations. The law also specifically empowers EPA to issue binding orders to other federal agencies under RCRA, and empowers both EPA and the states to impose fines and penalties against federal facilities that violate RCRA.[91] Unfortunately, much of the FFCA's promise to protect communities endangered by DoD waste - including military munitions and related contamination - has not been met.

Because RCRA was originally written to govern private sector wastes, Congress directed EPA to write a regulation (known as the "Munitions Rule") balancing the need for oversight of military munitions with the DoD's need to train and fight. Section 107 of the FFCA mandated that the Munitions Rule (1) determine when

[88] SARA Overview, Superfund Program, USEPA, at http://www.epa.gov/superfund/action/law/sara.htm.
[89] 42 USC s/s 6901 et seq. (1976)
[90] Latham.
[91] Ibid.

military munitions become hazardous waste and therefore subject to regulation under RCRA Subtitle C; and (2) ensure that when munitions are considered hazardous waste they are safely stored and transported.[92]

When EPA failed to write the rule in a timely manner, MTP sued to force the rulemaking to proceed. In a court-negotiated settlement, EPA agreed to produce the regulations by October 31, 1996. Because Congress failed to approve the federal budget that year, the deadline was extended to December 2, 1996. EPA ultimately issued the final Munitions Rule in February 1997.

When the Munitions Rule was finally issued, EPA undermined the intent of Congress by completely exempting all munitions at operational firing ranges from RCRA's definition of hazardous waste unless and until DoD specifically collects them for disposal. EPA's surrender to DoD ensured that millions of pieces of UXO will remain on military ranges leaching toxic contaminants into the environment. EPA also surrendered on transportation standards, ruling that munitions that would otherwise be regulated as hazardous waste under Subtitle C are exempt from those provisions as long as they are transported in accordance with DoD standards.

MTP challenged the Munitions Rule in Court, arguing that EPA acted contrary to law when it determined that munitions deposited on or off operational ranges are not a RCRA solid waste and therefore will never be considered a hazardous waste. Although the court ruled in favor of EPA, the challenge to the Rule's obvious surrender to the Pentagon's political and cultural clout galvanized affected communities to organize and seek out other means to protect themselves from munitions contamination (see below).

The Munitions Rule issued in 1997 postponed final action on the statutory status of used or fired munitions at non-operational (closed, transferring, and transferred, or CTT) military ranges, and the regulatory status of used or fired munitions that are recovered and then treated on-range at a CTT range. EPA deferred to DoD to write this section of the regulation, known as the "Range Rule." DoD produced a draft Range Rule in 1997, and a final draft Range Rule in 1999. EPA, states, tribes, and communities forced DoD to withdraw its proposed rule in November 2001 because of its many problems, including the failure to follow either RCRA or CERCLA processes and its failure to protect public health and the environment. In March 2000, DoD and EPA agreed to management principles for ordnance at CTT ranges, which serve as interim guidance until a Range Rule is adopted. As of this writing, no new draft Range Rule has been proposed by either agency.

Despite EPA's surrender to DoD on the Munitions Rule, the FFCA does provide at least one tool to protect communities from munitions contamination at active ranges. The FFCA specifically authorized EPA to issue binding orders under RCRA to protect public health and the environment from imminent and substantial endangerment by federal agencies' hazardous wastes. EPA Region 1 used this authority at the Massachusetts Military Reservation on Cape Cod because of munitions contamination of the only available drinking water supply for 500,000 people. DoD is currently seeking a Congressional exemption to remove this authority.

Historically, the DoD has argued that because of its mission (national defense) it should not be held to the same laws and standards as other agencies, private companies, and individuals. Military munitions and firing ranges have been no exception to this pattern. As noted above, the DoD argued successfully that it is capable of regulating its own munitions and operational ranges, despite over a hundred years of evidence to the contrary. Perhaps more importantly, the DoD attempts to avoid accountability to EPA, states, tribes, and the public even when laws and regulations apply. The DoD consistently seeks to address munitions contamination (including unexploded ordnance) under CERCLA rather than RCRA, and even seeks to use the CERCLA provisions that allow EPA and others the least input and recourse.

At the former Ford Ord in California, the Army argued for a decade that cleanup of UXO was not covered by the Federal Facilities Agreement for the base, that UXO is not a hazardous substance, that UXO is not subject to regulation under CERCLA, and that EPA had no authority over UXO cleanups. It took a suit by the Fort Ord Toxics Project to force the Army to submit to CERCLA and EPA regulations on UXO cleanup and perform a CERCLA remedial investigation/feasibility study for cleanup of UXO on the base.[93] More recently, the Army attempted to prevent regulatory and public involvement in its cleanup of ordnance and explosives by claiming

[92] Ibid. This article provides a much more detailed discussion of the legal and regulatory rationale behind the Munitions Rule.
[93] Fort Ord Toxics Project.

that its cleanup was a "removal" action, despite the fact that the effort had been underway for six years and was clearly part of a plan to implement a permanent solution to the contamination.[94]

At the Massachusetts Military Reservation (MMR) on Cape Cod, EPA Region 1 was forced to issue binding orders under the imminent and substantial endangerment provisions of the Safe Drinking Water Act and RCRA to protect public health from immediate dangers posed by munitions contamination. Region 1 resorted to the orders only after years of refusal by the military to investigate and correct existing contamination and ongoing practices that clearly endangered the facility's neighbors. The DoD contested the third of these orders – which required cleanup of the contamination – arguing that investigation and cleanup at MMR should occur only under the CERCLA Federal Facility Agreement (where EPA conveniently has no authority to issue binding orders to DoD). Fortunately for the community around MMR, EPA rejected this challenge.

Not surprisingly, states, EPA regions, tribes, and the public often prefer to use RCRA rather than CERCLA as an enforcement tool at military ranges and other sites with munitions contamination. RCRA provides much clearer and stronger means to protect public health and the environment and compel compliance with the law.

OTHER STATUTES

EPA Headquarters' surrender to the DoD on the Munitions Rule did not solve the problem of dangers posed by thousands of military sites contaminated with UXO and munitions constituents. The public, tribes, states, and EPA regions have been forced to seek out means other than regulation of munitions under RCRA Subtitle C to protect human health and the environment. Fortunately, other tools do exist and are beginning to be used.

EPA Region 1's use of the Safe Drinking Water Act (SDWA) to protect human health from immediate and substantial endangerment caused by munitions contamination of the sole source drinking water aquifer for 500,000 people on Cape Cod is probably the best known example of the application of a law other than RCRA and CERCLA to military munitions and firing ranges. Contamination at the Massachusetts Military Reservation (MMR) and EPA's orders under the SDWA are covered more fully in the MMR site profile later in this report.

The Clean Air Act (CAA) has also been invoked to prevent damage to human health and the environment by military munitions contamination. Because the CAA can be enforced by U.S. EPA, local officials, and the public, it provides an unusual avenue for community action to prevent exposure to munitions toxins. The Sierra Army Depot site profile later in this report provides more information on the successful struggle of the Pyramid Lake Paiute Tribe and others downwind of Sierra to use the Clean Air Act to protect their health.

The Clean Water Act (CWA) may also apply to military munitions, but problems with the law's waiver of federal sovereign immunity limit its ability to fully address munitions contamination. Under the CWA, the DoD must generally apply for and receive a permit to discharge munitions into waters. Unfortunately, an incomplete waiver prevents EPA from fining the military or issuing binding orders to enforce compliance with the law.

Over the past decade, affected communities, states, and tribes have sought out available tools to protect their health and environment. Pressure on the DoD to clean up toxic contamination and prevent ongoing pollution has increased at the local, regional, and national levels. The application of the SDWA, CAA, CWA, and other statutes to military munitions and firing ranges – despite EPA headquarters' surrender to the Pentagon on the Munitions Rule – reflects the reality that people being poisoned will always find means to protect themselves.

The DoD's response to these challenges has usually been to seek statutory exemptions that forestall any independent oversight of its activities. In May 2002, the DoD presented to Congress a package requesting blanket nationwide exemptions for all military munitions, training ranges and activities, and related operations from critical provisions of the Clean Air Act, RCRA, CERCLA, the Endangered Species Act, the Marine Mammal Protection Act, and the Migratory Bird Treaty Act. Despite the agency's repeated promises to work with states, tribes, and affected communities, the exemption proposals were developed without any consultation with those groups. DoD's proposals were presented to the House Armed Services Committee less than a week before markup of the 2003 Defense Authorization Act, to which the Pentagon wanted its proposals attached. At the time of this writing, Congress is still considering DoD's proposals.

[94] U.S. District Court for Nothern California, Ruling C-99-20485-RMW, Judge Ronald M. Whyte, March 12, 2001.

CONCLUSION

While defending our country, the Department of Defense has consistently poisoned communities through its production, testing, use, and disposal of munitions. Military firing ranges – as centers for the constant use of munitions – are often extremely contaminated and pose grave threats to public health and the environment. Residents of communities all over the world suffer chronic and acute health effects caused by the constituents of military munitions, and are endangered by unexploded ordnance. DoD has resisted efforts to investigate and remedy munitions contamination, and has sought special status above the law.

Community organizations that publicly question the impacts of military munitions and firing ranges, and challenge the DoD to protect their communities from its own practices, are often branded unpatriotic fanatics or even accused of intentionally undermining national security. The Department of Defense is under the mistaken impression that anyone who challenges its indiscriminate use and disposal of munitions is unpatriotic or treasonous. On the contrary, because we believe that the democratic process is what makes the United States strong and that our military exists to defend us from harm, even at its own hands, we have a duty to express our beliefs and question decisions that will affect our lives and the lives of our descendents.

How unpatriotic is it to want to protect the health of your community? How fanatical is it to want clean water to drink and uncontaminated food to eat? Does it make sense to poison communities in order to defend them?

The importance of holding our military responsible for its actions is apparent in light of the chronic disease and death associated with chemical contamination from munitions and the acute safety danger posed by unexploded ordnance.

Federal and state laws can only protect communities when those affected by military contamination and pollution are involved in the decision-making process and have unrestricted access to information about existing contamination, ongoing pollution, and cleanup options. Affected communities, tribes, and states must be full partners, not bit players, in the oversight of military munitions and firing ranges.

As should be clear by now, inadequate waivers of sovereign immunity, failures of policy and will by EPA headquarters, and cultural deference to the military have combined to allow expended or discarded military munitions and their byproducts to contaminate air, soil, water, and subsistence food supplies and damage human health. Despite a variety of statements by Congress that federal agencies and facilities should be treated the same as everyone else, communities poisoned by munitions contamination and endangered by UXO still receive less protection than communities affected by private facilities.

Human health, the environment, cultural and historic sites, and subsistence food supplies have already been harmed by munitions contamination. Military munitions and firing ranges and the poisons they release often remain largely unregulated until a catastrophe – such as contamination of the sole drinking water supply for half a million people – has already occurred. Many states and some EPA regions have taken the lead in addressing the problem, but often without adequate support from Washington. Action is desperately needed to protect communities, ensure cleanups protective of human health and the environment, and prevent additional contamination. Communities, tribes, and states will continue to seek out the means to protect themselves and organize to demand military accountability to our laws.

CASE STUDIES

Badger Army Ammunition Plant

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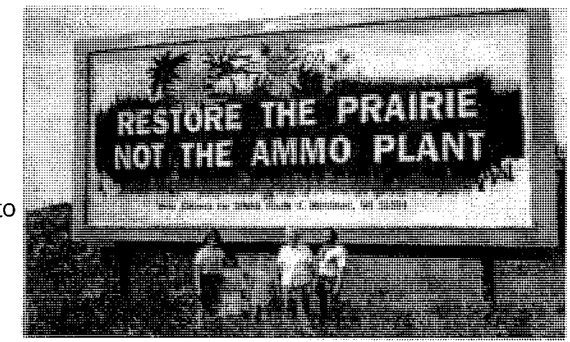

Members of Citizens for Safe Water Around Badger (Photo courtesy of CSWAB)

Badger Army Ammunition Plant (BAAP) was constructed in 1942 in Sauk County, Wisconsin, near the city of Baraboo. The plant was operated intermittently over a 33-year period to produce single- and double-base propellant for cannon, rocket and small arms ammunition. Plant operation was terminated in March 1975 and all production facilities and many support functions were placed on standby status. In November 1997, the Army announced there is no longer a national strategic need for Badger and the facility was closed.

Through previous studies at BAAP, it was discovered that during plant operation, various types of chemicals were processed and were disposed through open ditches, landfills, ponds, or through open burning. These materials, which include dinitrotoluenes (DNT), organic solvents, and acids, have percolated into the sand and gravel aquifer that lies directly beneath BAAP. In an effort to curb the migration of these contaminants, an interim groundwater extraction, treatment and discharge system was constructed. Construction began in October 1989 and operations began in May 1990.

Various investigations at BAAP have identified contamination of groundwater, sediments, and surface soils at the facility. Groundwater is the sole supply of drinking water for residents within a 4-mile radius of BAAP. Community water supply wells within the 4-mile radius of the facility serve the towns of Prairie du Sac (pop. 2,640) and Sauk City (pop. 2,700). A plume of carbon tetrachloride and trichloroethene-contaminated groundwater extends to the south of the Propellant Burning Ground Area. Although an Interim Remedial Measure (IRM) has been implemented to treat dinitrotoluene (DNT) and volatile organic contaminants in groundwater in the vicinity of the Propellant Burning Ground, a plume of trichloroethene and carbon tetrachloride contaminated groundwater extends downgradient of the capture zone of the system. Residential wells located in the glacial till aquifer were contamianted with carbon tetrachloride above the maximum contaminant level (MCL), and replacement wells in the bedrock aquifer have been installed.

Additional contaminants identified in the groundwater in the vicinity of the Propellant Burning Ground include trichoroethene, chloroform, 2,4-DNT, 2,6-DNT, and nitrates. Groundwater in the vicinity of the Deterrent Burning Ground and Existing Landfill is contaminated with chloroform, trichloroethene, 1,1,1-trichloroethane, 2,4-DNT, 2,6-DNT, cadmium chromium lead, sulfates, and nitrates. The groundwater in the Oleum Plant and v Pond Area contains elevated sulfate concentrations. Contaminants associated with the Rocket Paste Area groundwater include nitrates, sulfates, lead, cadmium, 1,1,1-trichloroethane, trichloroethene, tetrachloroethene, and 2,4-DNT. Nitrate and sulfate concentrations are elevated in samples collected from monitoring wells in the vicinity of the Nitroglycerine Pond Area, the Old Acid Area, and New Acid Area. Cadmium was detected at concentrations above background levels in the Old Acid Area. Benzene is a contaminant of groundwater in the Old Fuel Tank Area.

Inorganic contaminants identified in sediments associated with drainage-ways and ponds at the facility include aluminum, lead, mercury, and sulfate. Organic contaminants in on-site sediments include nitroglycerine, 2,4-DNT, and diethyl phthalate. Off-site sediments in Gruber's Grove Bay contained nitrocellulose, diethylphthalate, di-n-butyl phthalate, and lead. Adjacent to and downgradient of the facility are

Lake Wisconsin and the Wisconsin River. Extensive recreational uses include swimming, boating, and fishing. Sensitive areas identified include the bluffs adjacent to the eastern shore of the Wisconsin River near Prairie du Sac and Sauk City, which serve as wintering grounds for the American bald eagle (Haliaeetus leucocephalus), which is a federally listed threatened species in Wisconsin.

Soil contamination in the Propellant Burning Ground with carbon tetrachloride, trichloroethylene, 2,4-DNT, 2,6-DNT, n-nitrosodiphenylamine, phthalates, arsenic, copper, lead, and mercury has been identified. Soils in the vicinity of the Deterrent Burning Ground contain the same contaminants with the exception of arsenic and mercury. Elevated concentrations of nitrate and sulfate are present in Oleum Plant and Old Acid Area soils.

The RI/FS process has been completed. The RCRA permit modification of January 6, 1996 has been approved and finalized, but significant portions are being challenged by the Army.

ENVIRONMENTAL HEALTH AND CULTURAL PROBLEMS GENERATED BY THE MUNITIONS AT THE SITE

Occupational Exposures

The active years at Wisconsin's Badger Army Ammunition Plant have exacted a toll on the environment. In recent years, the U.S. Army has spent tens of millions of dollars on environmental studies documenting soil and groundwater contamination at dozens of sites at BAAP, however, virtually no resources have been devoted to identifying occupational exposures to former workers and the potential health effects these people may suffer. According to the Wisconsin Division of Health, very little information is available regarding potential occupational exposures at the plant. Occupational exposures were never monitored and consequently, no records exist.

Elevated cancer rates

In 1990, in response to community concerns about these exposures, the Wisconsin Division of Health conducted a health survey. The study confirmed that communities near the Badger plant have a significantly higher incidence of cancer deaths; the incidence of non-Hodgkins lymphoma and kidney/ureter cancer deaths are 50% higher than the balance of the state.

Hazardous Waste Incineration

On the west side of the plant is a series of "Contaminated Waste Area Pits". It has been estimated that as much as 500 gallons per week of 2,6-DNT (a component of explosives), benzene, diphenylamine, and chlorinated solvents were dumped in these pits from 1966-1970. These contaminants have, over the years, moved through the soil column and reached groundwater more than 100 feet below the surface. The U.S. Army has proposed excavation and incineration of contaminated soils as a possible remediation technology. This proposal, however, places nearby rural residents - including residents of the Bluffview Retirement Community - at unnecessary and excessive risk. The Army's own reports describe the potential impacts to public health and the environment: "Air quality impacts from incinerators can pose a potential risk to the community during remedial action if highly concentrated wastes are burned. Potential adverse effects as a result of implementing incineration include (1) releases of low levels of products of incomplete combustion during process upsets, and (2) releases or particulate matter containing heavy metals." Moreover, burning of chlorinated solvents is known to produce dioxins, one of the most toxic substances known to mankind.

COMMUNITY RESPONSE

Citizens for Safe Water Around Badger (CSWAB) was organized in 1990 when the community learned private drinking water wells near the Badger Army Ammunition Plant were polluted with high levels of cancer-causing chemicals. Although the Army documented extensive soil and groundwater contamination, this information never reached nearby residents and as a result three families living near the plant drank poisoned water for over 15 years. Levels of carbon tetrachloride were found in drinking water at 80 parts per billion; the safe standard is only 5. The founders, Alison Jones Pena and Laura Olah, believed community involvement could have prevented this tragedy and consequently organized CSWAB both to empower and to protect nearby residents and plant workers from further harm.

Of the 40 contaminated military sites in Wisconsin, the Defense Environmental Restoration Account has cited Badger as the most contaminated; 32 areas within the plant are polluted with solvents, toxic metals and explosive wastes. Environmental cleanup efforts, however, remain at a virtual standstill - held hostage to a politicized process, regulatory apathy and a lack of federal funds. Environmental cleanup costs have been estimated as high as $250 million - the legacy of production that spanned three wars and over fifty years.

The land for the Badger Ordnance Works was procured by the government on March 1, 1942 and construction was started mid-year in 1942. The powder plant displaced about 60 farmers. Many of the farms had been in the families for several generations; the neighborhood was closely knit and many families were related. This upheaval shattered the lives of virtually everyone living at that time in eastern Sumpter.

Active production of smokeless powder commenced at Badger Army Ammunition Plant in January 1943, and continued until September 1945, when the plant was placed on standby status. During this time, Badger employed about 7,500 people and manufactured 271 million pounds of single- and double-base propellant. The facility was reactivated during the Korean conflict during which approximately 286 million pounds of propellants were manufactured. Badger was reactivated in late 1965 and produced about 890 million pounds of propellants; the base returned to inactive status in 1975 and remains on standby to this day.

During active production years, facilities like Badger's Old Acid Area were used for production of nitric acid from ammonia. During this process, poisonous nitrogen oxides were released from rooftop vents. These gases had a red color and were irritating, and during temperature inversions, the Old Acid Area was enveloped in a brown haze. Open burning of waste propellants dispersed metal-contaminated ash to the environment.

Contamination occurred in areas such as the Propellant Burning Grounds, located on the west side of the plant near Highway 12. It has been estimated that as much as 500 gallons per week of 2,5-dinitrotoluene, benzene, diphenylamine, and other cancer-causing chemicals used in the manufacture of propellants were dumped in a series of waste pits at the Propellant Burning Grounds from 1966-1970. Over the years, these chemicals soaked into and affected the soils deep underground. Groundwater is rainwater and melted snow that has seeped into the soils and collected underground. We now know the waste pits are the principle source of a three-mile long plume of contaminated groundwater that has moved offsite and has reached the Wisconsin River, polluting private drinking water wells in its path.

Both carbon tetrachloride and chloroform, the chemicals discovered in nearby private wells in May 1990, can cause cancer and other illnesses in humans, depending on the amount and duration of the exposure. Liver tumors are most likely, but tumors also could form in lungs and kidneys, state health officials say.

In June 1990, nearby residents organized a meeting at the Sauk Prairie Community Center and CSWAB was formed. Within a month, the group called for a meeting with Army officials and successfully lobbied for off-site testing of 17 private wells near the Northeast corner of the plant, another area threatened by groundwater pollution. Months later, CSWAB uncovered a deliberate effort by Olin Corporation, the operating contractor at the plant, to edit test results from private drinking water wells resulting in the deletion of all data that exceeded health standards. The EPA now requires Badger to report all test results directly to homeowners.

In August of 1990, the Army announced that MetaTRACE, the company responsible for a facility-wide environmental study at Badger, had plead guilty to fraud. Another $6 million study was initiated to re-do much of this work. This same year, the Army began drilling two replacement private wells. CSWAB successfully lobbied Congress for DoD cleanup funds for Badger. Only one other site was selected for such funds.

In January 1991, Congressman Klug and Senator Kasten lobbied for the controversial $425 million Superconducting Magnetic Energy Storage (SMES) test model to be built at Badger. The technology was originally developed to store the tremendous amounts of energy needed to power directed energy weapons for use in ballistic missile defense or for use as an anti-satellite weapon. CSWAB, supported by previous efforts to block the proposal, successfully organized local residents in opposition to the experimental project. The electromagnetic effects of SMES - known to affect reproduction, cause embryotic changes, and cause leukemia and related cancers - were projected to extend 3,300 feet into the air. The project was not funded and consequently never built. This same year, Badger applied to the Wisconsin Department of Natural Resources for a permit to open burn up to 2,500 pounds per day of waste propellants. CSWAB organized a campaign to block the permit, including full page ads in the Shopper Stopper which were donated by the publication's owners Bart and Char Olson, and led to the Army's November 30, 1993 announcement that they would "discontinue the open burning and drop the application process." If the permit had been awarded, the facility could have released as much as 13,688 pounds per year of lead, making Badger the second highest emitter of lead in the State of Wisconsin.

In 1994, CSWAB successful petitioned the Department of Defense to establish a citizen advisory board - only one of five such boards nationwide. Soon afterward, the group revealed and then publicly criticized the Wisconsin Department of Natural Resources and the U. S. Environmental Protection Agency for failing to communicate persistent problems with the credibility of results from Olin's water testing laboratory, including samples from private drinking water wells. CSWAB's 1995 campaign successfully reopened the Wisconsin Division of Health's cancer cluster investigation of communities near the Badger plant, conducted five years earlier, which found a higher than expected number of male kidney/ureter cancer and female non-Hodgkin's lymphoma deaths. Documentation presented by CSWAB convinced the Division to expand the study to include breast and lung cancers, and to include data from the Wisconsin Tumor Registry - information that was missing from the original investigation. The following year, CSWAB received funding to conduct its own health survey of 200 families living near the Badger plant. The results were submitted to the Wisconsin Division of Health. In November 1997 the Division announced an updated health assessment would be done.

CSWAB successfully blocked a permit to use a diffuser structure on the bed of Lake Wisconsin for the direct discharge of treated industrial and sanitary wastewater. A subsequent proposal by Olin Corporation to convert the mothballed ammunition plant into a commercial industrial park and to target industries including pulp & paper mills, varnish, paper, and lacquer manufacturers, and ethanol and fertilizer plants was announced on September 18, 1996. This outrageous proposal met community resistance from all levels, including the county and local boards. Olin's proposal failed.

Afterward, the General Services Administration (GSA), the federal agency responsible for final disposition of the Badger lands, indicated their intent to market significant portions of the land for industrial operations. From their inception, one of CSWAB's goals was to build a sustainable future on the prairie now occupied by the Badger plant. The group believed the closure of Badger Army Ammunition Plant as a military facility provided a remarkable and unique opportunity to heal the land and the community. In 1941, local author August Derleth wrote to Franklin D. Roosevelt about the proposed new munitions plant to be built near Merrimac; "You can understand with what apprehension we look forward to the influx of elements which may work to bring about such change here that not even the inexorability of time can ever again rectify the errors that may have been done." The community now faced the challenges foretold so many years ago.

With the help of two other federal agencies, the US Department of Agriculture and the US Department of the Interior on behalf of the Ho-Chunk Nation, expressed interest in gaining ownership of the Badger property. In 1999, the Saux County Board of Supervisors asked the GSA to delay any final decisions for one year; the agency agreed. The following month, the County appointed a 21-person reuse committee that included delegates of state, local, federal, and tribal governments, and representatives of historic, cultural, conservation, and environmental interests, including CSWAB. An intergovernmental group followed, and today a conceptual land use plan divides the entire Badger property between the Ho-Chunk Nation, Dairy Forage Research Center, and the Wisconsin Department of Natural Resources, ensuring no lands are available for industrial development. Instead, proposed land use will be limited to conservation, sustainable agriculture, recreation, and education.

FORT RICHARDSON, ALASKA - EAGLE RIVER FLATS IMPACT AREA

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(U.S. Army Corps of Engineers photo)

From its origin at the Eagle Glacier within the Chugach Mountains in Alaska, the Eagle River flows swiftly in a northwesterly direction, traveling the relatively short distance of about 30 miles in its journey to tidewater. In its lower reaches, the river fans out into an intricate network of channels and ponds as it enters Knik Arm of Cook Inlet, forming the rich salt marsh of Eagle River Flats.

The lower Eagle River lies within the boundaries of Fort Richardson, a 56,000-acre active Army base located immediately north of Anchorage, Alaska. The Eagle River Flats, encompassing about 2,200 acres, have been used as an impact range since 1949. Now pocked with thousands of craters, the wetlands and associated mud flats contain an estimated 10,000 pieces of unexploded ordnance from years of mortar and artillery training. According to the Army, three types of munitions have been fired into Eagle River Flats: high explosives, smokes (white phosphorus or hexachoroethane-zinc mixture (HC)), and illumination flares.

The military continues to use the delta as an active range for high explosives and illumination rounds during winter months. An 8-acre site on the eastern edge of the Eagle River Flats was used for open burning and open detonation (OB/OD) of expired ordnance for more than 30 years, with the OB/OD ceasing in 1988. Demolition of propellants on the pad contaminated the adjacent salt marsh.

Thousands of waterfowl and other birds are known to die each year on the Eagle River Flats-estimates range from 2,000-5,000 deaths per year. In the spring of 1989 alone, 2,500-3,000 ducks were killed in a four-week period. Green-winged teal, northern pintails, and mallards are the most susceptible of the waterfowl, but the white phosphorus also poisons tundra and trumpeter swans, bald eagles, ravens, shorebirds, and herring gulls. It was not until 1990 that biologists determined that the birds are dying from the ingestion of white phosphorus, used by the military in their incendiary and smoke screening projectiles.

It took the Army nearly 20 years to begin to address the cause of the massive bird die-off first discovered in the early 1980s. Only in February, 1998 did the Army release a draft cleanup plan to address the white phosphorus poisoning that continues to kill untold numbers of shorebirds, waterfowl, and other wildlife each year. Trapped in saturated marsh sediments, the white phosphorus remains a poison indefinitely. The Army is detonating explosions and pumping to drain the areas most intensely contaminated with white phosphorus. When exposed to air and warm temperatures the white phosphorus oxidizes or volatilizes, rendering it less harmful to wildlife.

The military steadfastly refuses to take action to investigate or remedy the toxics and safety hazards posed by extensive contamination from unexploded ordnance on the Eagle River Flats. Contamination from the OB/OD area has not been adequately sampled to determine effects on wildlife and subsistence resources used by the Eklutna Tribe in the immediate area. The Eklutna Tribe uses a nearby site as a subsistence fishing camp. The Army continues to devastate and contaminate this ecologically fragile and productive salt marsh with high explosives testing.

At a meeting in early June 1998, Alaska Community Action on Toxics staff and local tribal leaders met to discuss action strategies to address military contamination at Eagle River Flats and throughout the area. The meeting included Janet Daniels and Pamela Miller, staff of Alaska Community Action on Toxics (ACAT), ACAT board member Alan Larson, Sr., Eklutna tribal leader Lee Stephan, and Upper Cook Inlet Environmental Protection Consortium (a consortium of area tribes) coordinator David Harrison. The group agreed to work together to ensure meaningful tribal involvement in cleanup decisions regarding Fort Richardson.

Jefferson Proving Ground

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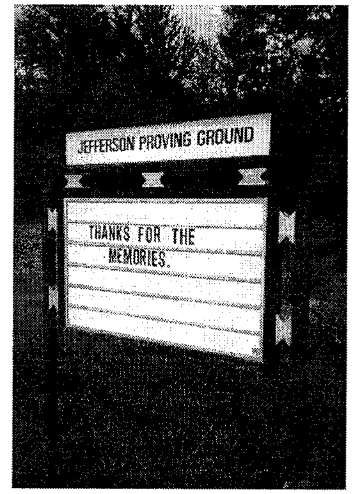

Memories left behind at JPG include 150,000 pounds of depleted uranium

The U.S. Army founded Jefferson Proving Ground in 1940-41 as an active munitions facility to test bombs, propellants, grenades, and high explosives. JPG closed in 1995 under the Base Closure and Realignment Act. The Proving Ground is divided into the Northern Firing Range Area, approximately 51,000 acres, and the Cantonment Area south of the firing line, approximately 4,000 acres. The area surrounding JPG is mostly rural farmland. The Proving Ground itself is an important natural habitat within the farmlands and urban areas bordering it. In addition to grasslands, the Proving Ground has 11,000 acres of forest within its boundaries, the largest continuous forest block in southeastern Indiana, and one of the largest in the Lower Midwest region of the U.S.

In December 1988 when the BRAC Commission recommended the closure of the Proving Ground, environmental studies identified contaminated sites including landfill and disposal areas, hazardous waste storage areas, fire training areas, underground storage tanks (USTs), and asbestos in buildings. Contaminants present at the base included depleted uranium, solvents, PCBs, heavy metals, VOCs, and petroleum hydrocarbons. Actions recommended included landfill closure, removal of USTs, and contaminated soil excavation.

The problem areas identified focus largely on the overall issue of unexploded ordnance that dominates the Proving Ground. Over 25 million rounds were tested at JPG between 1941 and 1995. The Ground may still contain up to 1.5 million pieces of UXO. An Archives Search Report produced by the Army Corps of Engineers in 1995 concluded:

> No areas north of the firing line were identified in this study that would not warrant further action prior to release from the government for civilian use. Though lower potential of OEW [ordnance and explosive waste] than the designated impact areas in the center, there is still an OEW potential. Any of the areas north of the firing line, proposed for release should be further characterized by statistical UXO sampling or cleared of OEW to a level (i.e. depth) in keeping with the land use. Subsurface intrusions of any sort at JPG should be approached with a degree of caution and respect for the possibility of OEW.

Cleanup of the Northern Firing Range Area will not be completed until at least 2003, and long-term monitoring will be required for at least 25 years. Cost estimates for study and cleanup range from $2 billion to $8 billion.

The Cantonment Area south of the firing line has been leased to private companies and public agencies for reuse. After the lease for reuse of the area was signed, Army surveys discovered previously unknown environmental contamination, which has cost much more to clean up than the Army received for the lease.

Between 1994 and 1999, the Army Environmental Center conducted two controlled technology demonstrations at the Jefferson Proving Ground. The tests were held to assess the capabilities of technologies that detect, identify, and remove unexploded ordnance. The Naval Explosive Ordnance Disposal Technology Division documented the demonstration results to aid in selecting effective and efficient systems to support its defense-related environmental cleanup effort. During Phases I, II, and III of the project, various ground-based technologies achieved an average probability of UXO detection as high as 77%, with a false alarm rate ranging from 60-149%. Analysis of Phase IV results was still underway at the time the report documenting results of the project was released. The report - entitled Jefferson Proving Ground Technology Demonstration Program Summary (May, 1999) - can be found on line at http://aec-www.apgea.army.mil/prod/files/jpgsummary.pdf.

In early 1998, the American Bird Conservancy named the Proving Ground a Globally Important Bird Area. The designation was announced by the Army and the U.S. Fish and Wildlife Service, which assists the Army in managing fish and wildlife resources at the 51,000 acre area. The Proving Ground is situated within one of the Midwest's finest forest and grassland habitats. This environment is important to the Henslow's sparrow, a grassland dependent bird which is suffering a population decline due to the land encroachment of humans and toxins in groundwater and soil. Loss of its grassland-nesting habitat has reduced Henslow numbers by an estimated 8 percent a year over the past three decades. The Proving Ground provides the breeding habitat for one of the five largest remaining Henslow's sparrow populations in the world, about 751 breeding pairs, according to the conservancy.

After eight years of advocacy by JPG neighbors and the Hoosier Environmental Council, the Proving Ground was converted to Big Oaks National Wildlife Refuge in 2000 through a 25-year lease to the U.S. Fish and Wildlife Service. The Army maintains responsibility for cleanup of all JPG lands. Limited access to the refuge is allowed for hunting, fishing, and escorted tours. At 50,000 acres, Big Oaks is the largest refuge in Indiana and the lower Midwest. Its residents include rare and endangered species such as bobcats, Indiana Bats and bald eagles.

Between 1984 and 1992, the Army tested approximately 220,500 pounds of depleted uranium penetrators (anti-tank ammunition) at JPG under a license from the Nuclear Regulatory Commission. Since these weapons are no longer being tested, the Army is requesting permission from the NRC to terminate that license. One part of this license termination process is the development of a Decommissioning Plan (DP) by the Army.

As of January 2001, the Army was attempting to walk away from 150,000 pounds of DU lying on and in the soil at JPG and proposing to do no remediation, no monitoring, and to rely on institutional controls to ensure no future human use of the area. It is estimated that approximately 150,000 pounds of DU remain in a 1,250 acre area of JPG called the Delta Impact Area. Big Creek, one of the major creeks in Jefferson County, runs from east to west directly through the Impact Area. DU penetrators were tested at JPG beginning in 1983 and continued for about ten years. Many of the DU rounds have been fragmented by collision with objects in the soil (other DU rounds, conventional UXO, etc.). Other DU rounds are buried in the soil.

PANAMA

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This section is excerpted from "Collateral Damage" by Carmelo Ruiz-Marrero, published by Resource Center of the Americas at http://www.americas.org, March 2001.

The United States ended its 96-year occupation of Panama in December 1999, withdrawing from a 10-mile-wide strip straddling the Panama Canal as required under the 1977 treaty. The occupation zone, totaling 360,240 acres, included three major military firing ranges.

U.S. officials estimate that the United States cleaned up about 80 percent of the range land, removing more than 8,500 pieces of unexploded ordnance and 2.1 million pounds of scrap metal. The Pentagon admits that at least 110,000 pieces of undetonated mines, mortar shells and bombs are scattered along the ground or buried under forest cover. U.S. officials say dense jungle and steep slopes made the task of clearing the ordnance too difficult and dangerous and that further work could have damaged the canal's watershed. Before leaving, the military recommended that "high-impact" areas, totaling more than 8,000 acres, remain permanently closed to humans.

To assess the cleanup, Panama hired the Washington law firm Arnold and Porter. The firm's report, including more than 60 photographs, contested the U.S. position that most of the land was ready for civilian use. The photos showed unexploded mortar shells and rocket warheads on the firing ranges. And they showed grenades and mounds believed to contain undetonated munitions outside the high-impact areas.

Even some U.S. officials say the cleanup was superficial. Members of cleanup crews told the Washington Post they were often instructed to look for unexploded munitions only at ground level and that they were not equipped with metal detectors. Rick Stauber, a former Army bomb disposal expert sent to assess the problem, told USA Today that the Pentagon wasn't interested in removing deadly debris and that his superiors hid information from him and barred him from inspecting sites.

The administration of Panamanian President Mireya Moscoso claims the cleanup fell short of a provision in the treaty requiring the United States "to take all measures to ensure insofar as may be practicable that every hazard to human life, health and safety is removed." Environmentalists say clearing the forests without destroying them is only a matter of spending time and money.

With unemployment exceeding 50 percent in parts of the country, Panamanians see a thorough cleanup as vital to their economy as well as for public safety. The firing ranges sit near real estate that Panama wants to use for housing, industry and ecotourism and for widening the canal to allow more shipping. In February, a backhoe dug up a live anti-aircraft round at a construction site in Pedro Miguel near the canal's east bank. The 40-millimeter projectile was the eighth military explosive found in Panama outside the ranges since 1997.

Many of the 60,000 people living in shantytowns near the ranges scavenge for scrap metal. The size of this population is expected to double in coming years as Panamanians move closer to the capital in search of opportunities. U.S. military explosives on or near the ranges already have killed at least 21 Panamanians in the last two decades. Last year, Moscoso issued a decree making it illegal to enter the ranges, but Panama can't afford to fence off the land or guard it.

The United States left behind more than unexploded munitions. The United States conducted secret tests of Agent Orange and other toxic herbicides in Panama during the 1960s and 1970s, according to a Dallas Morning News report that cited nine witnesses. The defoliant, used by the United States in Vietnam, contains deadly carcinogens known as dioxins.

The U.S. chemical weapons program in Panama ran for more than 40 years, according to a 1998 report by the Fellowship of Reconciliation, the Earthjustice Legal Defense Fund and the Chemical Weapons Working Group. The tests began in the 1920s and included live munitions training, according to participants and military documents obtained through the Freedom of Information Act. The United States sent three tons of a lethal nerve agent called VX for testing in Panama in 1964.

The United States didn't report the tests to Panama, even after ratifying the Chemical Weapons Convention (CWC) in 1997. (The convention requires a member state that has used chemical weapons on foreign territory to declare whether it has abandoned the weapons within 30 days of ratification.) At contaminated chemical weapons sites in the United States, the U.S. government has been prepared to clean up those sites. Clean-up is not technically impossible and the United States is obligated by the CWC to make sure that chemical-weapon test sites in Panama are cleaned up.

Stauber told the Christian Science Monitor that when he helped prepare the Defense Department report on leftover ordnance in Panama he saw documentation that 120-millimeter projectiles made with depleted uranium had been fired. At first, U.S. Ambassador William Hughes denied Stauber's claim. When the Fellowship of Reconciliation alerted Panamanian newspapers, U.S. officials admitted that the military had stored the uranium shells in Panama to test their deterioration in tropical climates. Stauber said the alleged test-firing would have been necessary to assure that the shells would function in battle.

Panamanian officials say a serious long-term threat is that toxic chemicals and heavy metals will seep into groundwater. Water samples last year near buildings at a former Army base, Fort Kobbe, and the former Howard Air Force Base showed high levels of petroleum distillates, according to the Panama News.

Pushing for a thorough cleanup, Panama has turned to the United Nations. In September, Panama's U.N. ambassador, Ramón Morales Quijano, said he had asked the U.N. General Assembly to take up the dispute in debates on the environment and on land mines, chemical weapons and biological weapons.

MAKUA VALLEY

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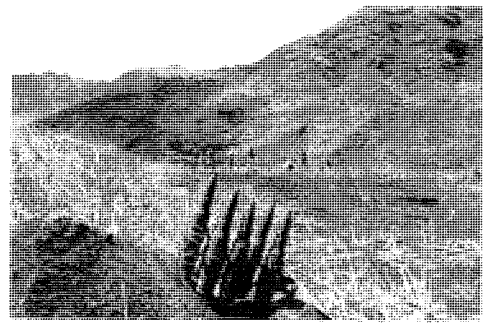

Abandoned shells at Makua (Photo by Ed Greevy)

1869-1941 ....Most of the Valley leased to cattle ranchers
1942-1943 ........U.S. Army confiscates 6,600 acres
1964....................U.S. Army returns 2,400 acres to the state
Present...............4,190 acres leased to the Army until 2029

The United States Army has occupied Makua Valley since World War II. DoD appropriated the entire valley when World War II broke out, evicting families, but did not return the land after the war as promised. Makua contains Indigenous Hawai'ian sites and artifacts – including *heiau* (temples), burial grounds, and fishermen's shrines – dating back 900 years. Fifty years of training, firing, and munitions disposal have destroyed homes, the local church and Indigenous Hawai'ian temples, and contaminated soil and groundwater. There are concerns that the valley is the most heavily contaminated area of unexploded ordnance after Kaho'olawe, Hawai'i. The Army's 1990 site assessment revealed that Makua is contaminated with metal fragments and dust, as well as over80 tons of toxic and hazardous chemicals and materials that were burned each year.

Environmentalists are also concerned with the loss of species relating to the Army's firing of ordnance in the valley and the increased risk of fire. The Army allows fires caused by training to go unchecked and notification of the state Department of Land and Natural Resources has been seriously deficient. The Army has always claimed that the setting of fires was necessary to clear vegetation needed to expose unexploded munitions. This would also allow the construction of what the Army claimed to be a fuel break road, which entails the expansion of the impact area where training exercises are conducted. The Fish and Wildlife Service (FWS) reprimanded the Army in July of 1992 in a letter stating that the intentional burning "did not conform to our previous agreements and understandings regarding prescribed burns."

Makua Valley is renowned for its botanical and biological life. The Achatinella genus of snails, of which there were believed to be over 40 species, is down to about 16 species. This is due to the continual destruction of the snails' habitat, and in 1981 the FWS put the entire genus on the endangered list. The Army has paid no attention to any need to preserve the native forest that houses the snails, openly burning tracks of forest to detonate unexploded ordnance and using this land as an impact area for sea launched rockets and aerial bombs. This takes a toll on the rich botanical diversity in the area. It is estimated that in some places of the valley 42 different species of plants can be found within a radius of 150 yards. A plant survey in 1977 identified 340 species of plants, 163 of which are found only in Hawaii. Forty-eight species of plants are currently on the federal endangered species list. There has been no communication between the Army and the FWS on measures to protect these plants even though under Hawaii state law endangered plants are subject to the same federal laws for endangered animal species. Any action that directly or indirectly leads to the death of a member of a protected species is prohibited. Fire threatens at least 16 of the species covered in the listing. Within the period 1990-1991, 10 fires resulted from the military's firing activities.

The Army has always been annoyed at people living on the beach side of Farmington Highway, which runs in front of the base. This area is owned by the state and on lease to the Army. This is rarely used by the Army, which confines most of its activities to the area of Makua off the road. The residents of this beach area, who the Army considers "squatters", are targeted for removal. Under the pretense of "safety", the Army has approached both the Board of Land and Natural Resources and the Department of Human Services to "determine the 'homeless' status to these people and initiate appropriate measures to relocate them from Makua beach." The state has not responded to this request.

Public outcry surfaced in November 1992 when the Army applied for a permit from the EPA to openly burn and detonate unexploded ordnance in Makua Valley. In September 1994, the Army withdrew this request. The danger lies in the fact that this permit is necessary for only 5% of the open burn/open detonation at Makua, the other 95% is classified as a training ground and is exempt from EPA regulation.

The emphasis is on a grassroots approach to forcing the military to take action and stop these practices, which are detrimental to the cultural, spiritual, and biological composition of Makua. The movement holds the military responsible for the pollution of the environment and looks to the community for input as to how these practices are affecting their lives. These are problems reflecting the ethnicity of people in the Valley, and the cultural upheaval that results from these practices. It further relates to economic disparity and the military's insistence on ignoring the needs of these people reflects a racist and classist bias in their activities.

The Army contends that the arid rocky floor of Makua Valley plays a crucial training role for soldiers, and has been utilized during World War II, Korea, and Vietnam. Due to current budgetary constraints and the pressure for maximum utilization of defense dollars, the Wai'Anae military training facility has broadened its activities. Eighty percent of the live-fire training on Oahu by Army soldiers and on Kaneohe by Marines takes place on 4,190 acres the Army leases from the state. The Army predicts that the need for combat ready troops in Hawaii will not decrease in the next 50 years as the country's commitment to the Pacific continues to rise. Makua is used for both firing on targets at fixed distances and tactical skill development. Under pressure from the community, the Army decided to modify its training activities in 1989 no longer allowing rockets or missiles to be used on the range. To combat fires, two 300,000-gallon helicopter dip ponds have been constructed and a firefighting crew is maintained. There was also a repositioning of targets on five firing points to prevent tracer bullets from landing outside the firebreak. These measures have not halted the damage to endangered species or the continuing contamination of the environment. The Army's continuing occupation of Makua still denies the community unrestricted access to these sacred lands.

The Army halted its live-fire training in 1998 after a series of fires and Malama Makua's indication that it intended to sue under the federal Endangered Species Act. The parties reached a settlement in 1999 in which the Army agreed to prepare an Environmental Impact Statement (EIS) for live-fire training at MMR. In December 2000, the Army produced an environmental assessment instead of the more comprehensive EIS, leading to another lawsuit that halted operations.

After September 11, both groups agreed to a settlement. The Army is now allowed to train in the valley for a limited number of days each year as long as it completes an EIS within three years. In return, Malama Makua was given monthly visitation privileges to the grounds and its members can observe the Army's activities in the valley. Community involvement will be an essential component to the Army's study this time around, with community members determining the scope and focus of the EIS.

MASSACHUSETTS MILITARY RESERVATION

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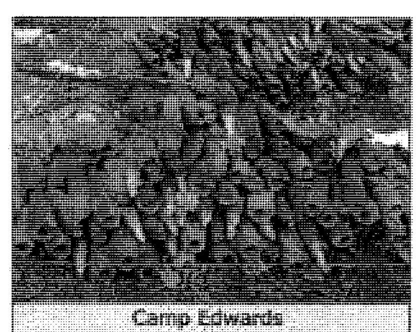

EPA photo of UXO at MMR

The Massachusetts Military Reservation (MMR) on Cape Cod includes Otis Air National Guard Base, Coast Guard Air Station Cape Cod, and Army National Guard Camp Edwards. MMR covers 22,000 acres on the Upper Cape near the towns of Falmouth, Mashpee, Bourne, Yarmouth, Barnstable, and Sandwich. Camp Edwards – which includes an artillery impact area and weapons firing ranges - occupies about 15,000 acres in the northern part of the base. Otis occupies most of the rest. MMR has been used to provide training for Air Force, Army, and National Guard units since 1911. The heaviest activity occurred from 1940 and 1946 (Army) and from 1955 to 1972 (Air Force). The Army Reserve and National Guard have used most of MMR for training including weapons firing for the past few decades.

Ninety years of operations at MMR have wreaked havoc on the environment and human health in the Upper Cape. There are at least 11 major plumes of groundwater contamination both on and off the base, emanating from over 75 contaminated sites including munitions impact, firing and disposal areas as well as fuel and chemical spill areas, landfills, and others. Hazardous contaminants include munitions constituents such as lead, propellants, and the explosive compounds RDX, HMX, and TNT, as well as volatile organic compounds (such as trichloroethane, tetrachlorethylene, and ethylene dibromide), jet fuel, benzene, and detergents. MMR was placed on the federal National Priorities List – the list of the most contaminated and dangerous sites in the country, often known as the Superfund list – in 1989.

MMR sits atop the Sagamore Lens, the recharge area for the Upper Cape's sole source drinking water aquifer - the only source of drinking water for 500,000 permanent and seasonal residents. The aquifer provides drinking water to over 70,000 homes and businesses. Decades of munitions firing and disposal, along with other activities, have contaminated over 66 billion gallons of the aquifer. A January, 2000 EPA letter to the DoD notes that:

> There is now ample evidence that military munitions used and disposed of during training at Camp Edwards have contaminated parts of the Sagamore Lens with RDX and other toxic compounds – evidence that DoD, in its objection to EPA's April 1997 SDWA order restricting training at Camp Edwards, asserted would not be found. In fact, 10 percent of the monitoring wells installed as part of the groundwater study conducted pursuant to the February 1997 SDWA order show RDX concentrations above EPA's health advisory.

In 1997, EPA Region 1 concluded that munitions constituents and other contaminants in the drinking water aquifer posed an "immediate and substantial endangerment" to human health in the area, and has ordered the DoD to take actions to investigate and remedy the contamination. Without the unequivocal waiver of federal sovereign immunity from the Safe Drinking Water Act that Congress adopted in 1996, and without EPA's ability to issue binding orders to force DoD action, the people of the Upper Cape would not know the extent of the danger to their health and cleanup would not be underway.

To date, EPA Region 1 has issued four administrative orders regarding investigation and cleanup of contamination both on and off MMR property. These orders were necessary because DoD refused to investigate possible contamination and take measures to protect the public from its pollution. Administrative Order (AO) 1, issued in February 1997 under the Safe Drinking Water Act (SDWA), required the National Guard bureau to undertake a comprehensive study of the groundwater under and emanating from the training range and impact area. AO 2, issued in April 1997 under the SDWA, suspended the firing of high explosive and inert artillery and mortars and the use of propellants and pyrotechnics until the groundwater study was completed and EPA indicated that the use of these munitions could resume; suspended planned demolition of ordnance and explosives, except as part of range clearance activities; and required a sweep of the impact area and training ranges for unexploded ordnance (UXO). The provision for a UXO sweep was removed by EPA headquarters based on a statement by the Department of Defense Explosives Safety Board that UXO does not leak or corrode and that the potential for contamination occurring from UXO was "virtually zero." DoD's statement was proven false by the results of the groundwater study.

AO 3, issued in January 2000 under the SDWA, required the National Guard Bureau to conduct rapid response actions to remove soil at six highly contaminated source areas; evaluate groundwater and soil cleanup technologies at five areas requiring long-term remedies; and implement remedies once they were selected by EPA. This order found that, in spite of DoD's assertion to the contrary, UXO at MMR and other bases is often corroded and leaks explosives into the soil, where they often move into groundwater. AO 4, issued in January 2001 under the Resource Conservation and Recovery Act, required the use of the controlled detonation chamber (also known as the Donovan Chamber) to dispose of munitions and UXO.

More recently, the munitions constituent perchlorate was found in drinking water supply wells in the town of Bourne, forcing the shutdown of several wells. Perchlorate contamination at up to 311 parts per billion has been detected in the southeast corner of the base near the most productive part of the aquifer. Region 1 recently established a groundwater cleanup level that the agency "believes protective of individuals potentially exposed to perchlorate in drinking water at Camp Edwards" of 1.5 parts per billion.

To date, explosives contamination has been found in about half of the 200 monitoring wells installed on Camp Edwards (one part of MMR); contamination in 53 exceeds EPA's health advisory levels. The toxic explosive compound RDX has been detected in groundwater at up to 370 parts per billion (EPA's standard is 2 parts per billion). Region 1's Betsy Higgins has stated "they're finding a lot more contamination than anyone ever thought we would find." Because of pollution from MMR, the Upper Cape could face a drinking water shortfall of 11 million gallons a day by 2020.

The true scope of MMR's impact on the health of its neighbors will not be known for some time, if ever. There is already evidence that the impact may be severe and that women in particular may be in danger. In 1991, A Boston University study found as association between artillery and mortar firing positions and lung and breast cancer in people living nearby. The authors recommended an end to the practice of burning excess propellant in the open air at firing positions. Between 1969 and 1993, women in the Upper Cape died of lung cancer at a rate 33% higher than the state average. From 1982-1990, women in the Upper Cape were 47% more likely to be diagnosed with lung cancer than women in the rest of the state. Women in the Lower Cape were only 14% more likely to be diagnosed with lung cancer. The rate was even higher in census tracts near MMR, 64% greater than the rest of the state. These findings suggest exposure to airborne contamination, which MMR produced in abundance through burning of propellant, open burning/open detonation of munitions, and live fire training. Little research has been done to date on the health effects of the drinking water contamination.

OKINAWA

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Okinawa consists of a group of islands that make up the southern-most prefecture in Japan. It is situated midway between Tokyo and Manila, and called the "keystone of the Pacific" by military planners because of its strategic location.

During World War II, Okinawa was a strategic front-line defense base for the Japanese Forces. It became the site of the only ground battle fought on Japanese soil, and was also one of the fiercest battles in the Pacific Campaign, in which about 220,000 civilians and soldiers died. After the surrender of Japan, Okinawa became a U.S. controlled territory until 1972.

After reversion to Japan, most of the U.S. bases remained in Okinawa. Seventy-five per cent of U.S. military bases in Japan are located in Okinawa, although Okinawa is only 0.6 % of the land area of Japan. The U.S. military bases occupy about 11% of prefectural land. On Okinawa's main island, U.S. military bases take up roughly 19 % of the land. The high concentration of U.S. military facilities and areas has had various impacts on the Okinawan people's lives and the environment.

Okinawan people have suffered from health and environmental problems from U.S. military base activities. These have included noise pollution, destruction of forest, forest fires from military exercises, soil erosion from bombing ranges and training areas, and chemical pollution. There have also been disturbances to the marine ecosystem, including destruction of coral reefs smothered by eroded soils, destruction of cultural assets, and the possibility of contamination from depleted uranium. Furthermore, military planes and helicopters crash every year in Okinawa. Other accidents have involved splinters of shells and stray bullets striking local residents.

The situation in Okinawa is particularly problematic because neither Japanese law nor U.S. laws apply to U.S. Forces overseas. The military is self-regulated. The U.S. Forces Northern Training Area is a treasury of rare plats and animals, such as the Okinawa rail and Pryer's Woodpecker. Since the U.S. military is the only body that has authority to inspect the areas it uses, there is no way for local governments to determine if natural resources are being protected.

Serious damage from U.S. military operations has been documented outside of the bases, however. In Camp Hansen, for example, live-fire exercises have denuded the surrounding mountains and large quantities of red clay have flowed out of the mountains into the Kin Bay, with the resulting mud polluting the coral reef. Aircraft noise measured near the Kadena Air Base has exceeded the standards set by Japan's Environmental Agency at over half of the noise measuring spots. This has negatively affected residents as well as interrupted classes at schools in the vicinity. When the U.S. returned a former communications site to Japan in 1995, PCBs and mercury exceeding the environmental limit were detected in the dirt left inside purification tanks. Because U.S. law does not require the military clean up its overseas bases, the Japanese government had to spend two years cleaning the site, seriously delaying reutilization plans for the facility. Additionally, in the past five years, the military has caused 69 bush fires and 21 oil spills in Okinawa.

In addition to facing environmental problems, Okinawans have had to deal with hit-and-run accidents involving drunken soldiers, murders, rapes, and thefts. There have been more than 5,070 criminal charges brought against U.S. military personnel since Okinawa reverted to Japan in 1972. Among the worst cases have been 531 cases of brutal crimes and 955 assaults. Okinawan people have demanded the tightening of discipline for U.S. troops stationed in Okinawa, and an expedited consolidation of U.S. bases in Okinawa since the abduction and rape of a 12-year old Okinawan schoolgirl by three U.S. military service members in 1995.

The public outcry over the rape led to the U.S. agreeing to return the bases to Japan. This is not an exit strategy, however, as the U.S. will build new bases at other Okinawa locations. One site being discussed is the coastal area of Naga City. The coastal area boasts a unique subtropical ecosystem and biologically diverse marine life. The surrounding reef is home to the dugong, an endangered species declared a National Treasury of Japan.

The Marine Mammals Committee (MMC), a U.S. governmental organization, has sent a recommendation to the U.S. Defense and State departments to conduct an environmental assessment at the proposed site. The MMC expressed their concerns that the habitat of the rare sea mammals could be devastated. Should the findings of the assessment indicate a major environmental impact, the standards to be used in any reevaluation of the relocation site must be made clear in advance.

Helen Marsh, a recognized authority on dugongs, criticized Japanese government officials conducting preliminary research at the site for their delaying tactics and requested that the State Minister in Charge of Okinawan Affairs order a prompt scientific assessment. Local people and environmental organizations in Okinawa have monitored the actions of both governments and will continue to push for conservation and cleanup for the dugong and the people in the surrounding communities.

San Diego, California

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Environmental Health Coalition (EHC) is a 22-year old grassroots environmental justice organization dedicated to the prevention and cleanup of toxic pollution threatening our health, our community, and the environment. EHC has a variety of campaigns all centered around environmental justice, pollution prevention, and right-to-know. We work in close coalition with community residents, community interest groups, labor, and other environmental and health organizations.

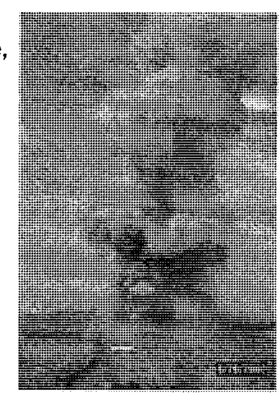
San Diego County is one of the largest military installations in the world. It hosts seven major bases, 120 commands, and the Navy controls 181,000 acres of San Diego County. One third of the US Pacific fleet is home ported here and San Diego Bay now hosts the newest Nuclear Megaport for Navy nuclear vessels. Ultimately, three nuclear-powered aircraft carriers and up to 14 nuclear submarines will be located in close proximity to densely populated areas of downtown San Diego and Coronado.

UXO plague San Diego both in land and sea. The UXO crisis was initially brought to the forefront in 1983 when two young boys were killed when abandoned UXO they found in a residential neighborhood exploded. It took nearly five years and a visit from TV's 20/20 crew to motivate the City to post warning signs in contaminated areas. In the late 90's, more ordnance was found near a local high school. Warning signs about UXO are still posted in public use areas like Mission Trails Park because clearance of UXO cannot be assured. Closure of the Navy's Carrizo Impact Area due to UXO contamination in the Anza Borrego Desert State Park has forever removed from public use some of the most scenic and historic lands in the Park. Live bombs are still found there, on an average, several times a year by patrols.

There are also a variety of problems with historical ordnance dumping in San Diego Bay. The presence of UXO in sediments dredged from San Diego Bay stopped a major beach replenishment project when two 80 mm shells were dumped on local beaches during the project. San Diego has a recurring problem with explosive devices, such as Navy flares, washing up on public beaches along the coast and in the Bay.

Since 1994, EHC's primary military organizing effort has been to clean up the five Navy bases around San Diego Bay and to fight the location of the Naval Nuclear Megaport in San Diego Bay. We are now attempting to effect policies and legislation to bring the Navy up to the same standard of regulation for environmental and public health and safety laws as the private sector. We are working with our local Congressman, Bob Filner, to pass H.R. 2154: the Military Environmental Responsibility Act (MERA) and with local elected officials to secure emergency planning for communities living near naval nuclear reactors.

SIERRA ARMY DEPOT



An OB/OD event at Sierra

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The open-air destruction of obsolete and surplus hazardous waste munitions began at Sierra Army Depot (SIAD) in the 1950s. Since that time, thousands of tons of munitions have been destroyed every year. This issue was brought to the forefront of discussion due to the lack of consideration to Nevada by California regulators.

The burning grounds used by Sierra Army Depot are approximately three miles west of the Nevada State line and 13 miles from the Pyramid Lake Indian Reservation (PLIR). The reservation encompasses 477,000 acres, of which approximately 115,000 are included in Pyramid Lake, a terminal, alkali lake. Unique to Pyramid Lake are two species of fish, the Cui-ui and the Lahontan cutthroat trout. The Cui-ui is an endangered prehistoric suckerfish while the cutthroat is a threatened species. Pyramid Lake also supports a colony of American white pelicans that live on Anaho Island, a small isle off the lake's eastern shore.

The Tribe began the fight against SIAD back in 1995. The Tribe's protests against OB/OD spanned four Tribal administrations. Along with the Tribe, the protest against OB/OD included Senator Harry Reid, Nevada Department of Environmental Protection, Washoe County, Rural Alliance for Military Accountability (RAMA), Residents Against Munitions (RAM), and residents from California and Nevada.

The Pyramid Lake Paiute Tribe (PLPT) has long been concerned about the long-term health of its citizens and the environment from the smoke plume produced by OB/OD. The munitions treated contain heavy metals such as aluminum, beryllium, chromium, copper, lead, nickel, and zinc, as well as dozens of organic and inorganic constituents, many of which are know carcinogens.

In 1993 the California Air Resources Board was involved in an inspection with California EPA. Violations were noted and recommendations made. Even with local oversight though, SIAD was virtually unregulated until 1998, when the Depot was issued its first Title V permit.

A Title V permit includes every "federally-enforceable" air pollution requirement that applies to a particular facility. The Title V is a landmark program because it stems from the Clean Air Act and can be enforced by U. S. EPA and the public. It is the enforcement of the regulations in Title V that brought about the eventual end of OB/OD at SIAD.

The Title V permit issued to SIAD was supposed to regulate all air emission units (AEUs) on site. Unfortunately, along with several errors and omissions, the permit had no regulations listed for either the open burning or the open detonation areas even though both areas were designated as AEUs.

A coalition of environmentalists and the Tribe attended Lassen County Air Pollution Control Board (LCAPCB) meetings for a year trying to persuade the Board to reopen the permit based on the evident flaws. EPA, Region IX, as involved in the process of educating the Board on the appropriate steps to take. Finally, after going through all the imposed hoops that the LCAPCB placed before the public and EPA, the Tribe appealed to EPA in October 2000 to reopen the permit. EPA then made a determination that the LCAPCB must reopen SIADs Title V permit to correct inaccuracies in the permit on November 2000. LCAPCB reopened the Title V permit on December 2000. A final Title V permit was issued on May 25, 2001.

One key provision of SIADs permit is the prohibition of OB/OD unless granted a 4:8 exemption. Regulation 4:7 prohibits open burning in Lassen County unless an exemption, 4:8, can be obtained by showing material in need of disposal poses a fire hazard and that no safe alternative treatment methods are available. The Depot had the burden of proving that the munitions to be treated qualified for the exemption. SIAD officials repeatedly insisted that the Depot's own public officer had the authority to obtain the exemption and that Lassen County had no authority to restrict the Depot's OB/OD operation. This above the law attitude characterized how Depot representatives conducted themselves at meetings throughout the dispute.

In a landmark decision, an Air Pollution Control Officer (APCO), denied a 4.8 Exemption to SIAD on August 2001. Deposition testimony by the head of the demilitarization wing of the Army was crucial to the decision made by the APCO. That testimony stated that alternative technologies existed to treat those munitions and that the munitions could be safely stored.

SIAD officials appealed the ruling to the local Hearing Board for a variance, but a final order issued by Army headquarters on September 26, 2001, ended the matter. The order, issued by Operational Support Command (OSC), instructed SIAD to withdrawal its appeal and discontinue large-scale OB/OD at SIAD rather than challenge the ruling from Lassen County. OSC cited the reason as one of economics.

This brings us to the second permit required by SIAD, a Hazardous Waste Permit issued by the California Department of Toxic Substances Control (DTSC) also known as a RCRA Permit (Resource Conservation Recovery Act). The RCRA permit has been on interim status since 1980. Depot staff frequently stated they were regulated by RCRA, therefore; Title V was unnecessary. The Depot preferred being regulated by the RCRA permit because the permit's interim status meant there would be no regulation.

The winds in this region are generally westerly. This makes Pyramid Lake Indian Reservation, Nevada, and Washoe County down winders. To alienate Nevada further, DTSC produced an Ecological Risk Assessment (ERA) and a Health Risk Assessment (HRA) without consulting anyone in Nevada. Needless to say, the communities down wind of Depot activities felt these documents had very little merit when not a single health, environmental, or state official was contacted in the production of documents that alleged impacts to Nevada were not significant.

A draft permit and Environmental Impact Report (EIR) was released in October 1999 for public comment until

September 2000 with public hearings held at the conclusion of the comment period. A second draft was to be released in 2002.

Very serious questions as to the length of time DTSC took to prepare the permit were brought up by PLPT, the Nevada Department of Environmental Protection, and Washoe County-Nevada. There were concerns about the inadequacy of previous scoping documents to address the impacts of OB/OD as early as 1995. Many of the concerns previously raised were never fully addressed in the 1999 EIR.

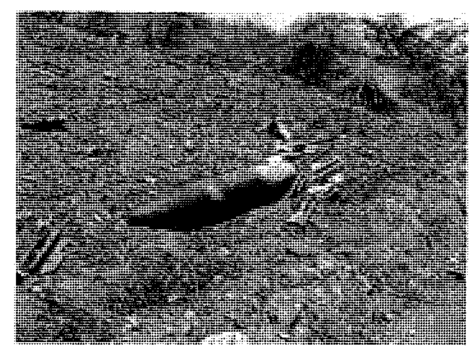
The Tribe was instrumental in bringing attention to the SIADs method of OB/OD. Pyramid Lake Paiute Tribe is a federally recognized Tribe in which the federal government has trust responsibility over its tribal lands. This brought EPA, Region IX to the table. EPA coordinated between all the involved parties to make sure all regulations were being followed and all voices heard. Tribal Chairman, Alan Mandell, also connected with Len Richeson, Tribal Liaison for the Office of the Secretary of Defense (OSD). This office spoke to the higher levels of Army command on our behalf. It was important to OSD that the process of consultation was taking place between all the parties.

Senator Harry Reid brought this issue to the higher levels of the Department of Defense and requested involvement by the Agency for Toxic Substances Disease Registry (ATSDR). After a preliminary investigation, ATSDR accepted Senator Reid's petition to investigate and have a report due spring 2002.

The successful conclusion to the ending of OB/OD at SIAD was a joint endeavor spanning several years. PLPT, RAMA, RAM and concerned citizens met monthly to discuss recent actions and strategize where to apply pressure next. The coalition also kept county, state, and legislative officials informed on a regular basis. The filing of a joint lawsuit against the United States Department of the Army was critical. The implications from this outcome may be helpful to other communities facing similar problems. The key to victory was maintaining friendly, positive contact with personnel from every agency connected to the problem including the "opposition".

VIEQUES, PUERTO RICO

Contact: Committee for the Rescue and Development of Vieques, (787) 741-1717 or oficina@prorescatevieques.org



Navy bombing has littered much of Vieques with thousands of pieces of ordnance

Since 1940, the U.S. Navy has used three-quarters of the island of Vieques, Puerto Rico for bombardment, munitions storage and disposal, weapons testing, and other training. The island's 9,300 residents, sandwiched between Navy activities on the western and eastern sides, have long resisted the Navy's bombing, largely because of the environmental and health devastation the Navy has wreaked on the island.

Environmental alarms have been sounding in Vieques for more than 20 years. A Water Quality Survey conducted by the Navy in 1978 found high levels of zinc and lead in surface water in eastern Vieques. The same study showed the presence of RDX – a toxic component of military explosives – in several drinking water supply sources on civilian land.

The Navy's own reports to the Environmental Protection Agency (EPA) from 1985 to 1999 showed that discharges of lead, barium, cadmium, arsenic, boron, cyanide, hexavalent chromium, and 13 other substances repeatedly violated the Clean Water Act and Puerto Rico Water Quality Standards. The EPA declared in August 1999 that the Navy had committed 102 violations of the Clean Water Act by releasing pollutants into the waters of the Vieques training area. In addition, by refusing to conduct a biological assessment of the effects of naval training on endangered species, the Navy is in violation of the Endangered Species Act. The Navy also operated an open burning/open detonation disposal site for explosives for 15 years, in open violation of the Resource Conservation and Recovery Act. In February 1999, two Marine Corps jets fired 263 rounds, each containing 148 grams of depleted uranium (DU), onto the Vieques range, another clear violation of U.S. law. Only 57 of the rounds were recovered in March 1999. A study conducted later in the year found significantly higher than background radiation levels a mile from where DU was fired.

These violations not only show a disregard for the law, but they are also indicative of a lack of concern for the health of Vieques residents. Prevailing trade winds on the island blow from east to west 80% of the time. During the 1980s, the former departmental chief for the Puerto Rico Environmental Quality Board demonstrated through mathematical studies and field trials that particulate matter raised by munitions impacts in the bombing range reaches Vieques' populated areas. This could explain the high rates of cancer and other diseases believed to be caused by heavy metals and organic compounds that leach out of unexploded ordnance into the food chain and air people breathe. Of 49 Vieques residents who participated in a study in June 2000, 45% had excessive levels of mercury in their hair or fingernails. Six subjects also had high levels of lead. According to Dr. Carmen Ortiz, the epidemiologist and pediatrician who conducted the study, naval training is the only known source of mercury contamination in Vieques. According to the EPA, mercury contamination can cause nervous system damage including tremors, mood and personality alterations and kidney damage. One of the leading routes of exposure to mercury is through ingestion. Mercury bioaccumulates in fish which is a main staple food for residents of Vieques. Pregnant women can pass on mercury contamination to a fetus causing neurological developmental abnormalities. Lead ingestion or inhalation causes problems to the central nervous system, blood, lungs, liver, and reproductive system.

The Navy maintains that the heavy metal contamination in Vieques is a result of natural background concentrations of the metals in seawater. Yet the measurements on which the Navy bases its estimates of background concentrations were taken from a single well in eastern Vieques, close to an area known as the "reef of bombs," used by the military for many years to blow up waste munitions for disposal.

The sound of explosions has also had negative effects on Vieques residents. Not only have explosions cracked roofs and walls, but they have also contributed to severe emotional disturbance among school children. Noise from ships firing and active sonar has also been shown to affect marine animals, including whales beached near Vieques.

The physical environment fares no better when pitted against the Navy. A study conducted in 2000 by biologist Arturo Massol and radiochemist Elba Diaz showed that vegetables and plants growing in the civilian area of Vieques are highly contaminated with lead, cadmium, copper, and other heavy metals. Edible crops had metals substantially above the maximum set by the European Union Council, and also far exceeded plants tested in the Puerto Rican town of Guánica. The plants most affected were those with shallow roots, such as chili, squash, and grasses, while trees were less contaminated. This is consistent with the thesis that heavy metals are deposited in the civilian area through air dispersion by wind from the bombing zone.

Coral reefs, which are declining worldwide, have also been damaged by Navy bombing in Vieques. James Porter, a respected expert in coral reefs, concluded that bombs were causing irreparable harm to the reefs. The Navy told Puerto Rican government officials that holes in the reefs were the result of hurricane damage, but the existence of shrapnel in the holes indicates otherwise.

In addition to the harm that has already been done to the island of Vieques, the potential for even more extensive damage is great. For example, between 1990 and 1992, the Navy sank two vessels loaded with hundreds of barrels of unidentified toxic waste below the surface of the coral reefs. The two sites have a total of approximately 1,100 barrels, containing both solid and liquid materials. During training, both sites are blanketed by live artillery shells and bombs, which fall up to 400 yards offshore.

While the Navy has announced that it will cease operations in Vieques by May 2003, no meaningful cleanup is planned for the area. Current law only requires a surface sweeping of the area. This lack of a cleanup standard has already presented itself on the western end of the island, where the Navy has simply erected fences around contaminated areas and proclaimed them "conservation zones."

APPENDIX A - ABBREVIATIONS AND ACRONYMS

AFB	Air Force Base
AR	Army Regulation
ATSDR	Agency for Toxic Substances and Disease Registry
BLM	Bureau of Land Management
BRAC	Base Realignment and Closure
BRAC ERP	Base Realignment and Closure Environmental Restoration Program
CEQ	Council on Environmental Quality
CERCLA	Comprehensive Environmental Response, Compensation and Liability Act of 1980
CITES	Convention for International Trade and Endangered Species
CFR	Code of Federal Regulations
CSWAB	Citizens for Safe Water Around Badger
CWA	Clean Water Act
DENIX	Defense Environmental Network and Information Exchange
DERP	Defense Environmental Restoration Program
DOD	Department of Defense
DOE	Department of Energy
DU	Depleted Uranium
DUSD(ES)	Deputy Undersecretary of Defense for Environmental Security
EA	Environmental Assessment
EHC	Environmental Health Coalition
EIS	Environmental Impact Statement
EJ	Environmental Justice
EPA	U.S. Environmental Protection Agency
FONSI	Finding of Not Significant Impact
FOTP	Fort Ord Toxics Project
FUDS	Formerly Used Defense Sites
FWS	Fish and Wildlife Service
FY	Fiscal Year
GAO	General Accounting Office
HMX	High Melting Explosive
HRS	Revised Hazard Ranking System
HSWA	Hazardous and Solid Waste Amendments
IRM	Interim Remedial Measure
MACOM	Major Army Command
MCL	Maximum Contamination Level
MERA	Military Environmental Responsibility Act
MTP	Military Toxics Project

NAS	Naval Air Station
NCP	National Oil and Hazardous Substances Pollution Contingency Plan
NEJAC	National Environmental Justice Advisory Committee
NEPA	National Environmental Policy Act
NPL	National Priorities List
NTS	Nevada Test Site
OB/OD	Open Burning/Open Detonation
OEW	Ordnance and Explosive Waste
PRP	Potentially Responsible Party
RAB	Restoration Advisory Board
RCRA	Resource Conservation and Recovery Act
RDT&E	Research, Development, Test and Evaluation
RDX	Royal Demolition Explosive
RI/FS	Remedial Investigation/Feasibility Study
ROD	Record of Decision
ROTHR	Relocatable Over-the-Horizon Radar
SARA	Superfund Amendments and Reauthorization Act
SWDA	Solid Waste Disposal Act
TSD	Treatment, Storage or Disposal
USACE	U.S. Army Corps of Engineers
USAEC	U.S. Army Environmental Center
UST	Underground Storage Tank
UXO	Unexploded Ordnance

APPENDIX B - GLOSSARY

Baseline Risk Assessment – An evaluation of the potential threat to human health and the environment in the absence of any remedial action at a site.

BRAC – the environmental restoration portions of the Base Realignment and Closure Program (BRAC) was established to help identify, investigate, and cleanup contamination on installations identified for transfer or sale under the auspices of the Base Closure and Realignment Commission Report of December 1988. The process consists of the same three phases as the IRP:

> Environmental Baseline Survey or PA/SI – to identify potential sites with hazardous waste contamination;
>
> RI/FS – to determine the nature and extent of contamination at a site and to identify alternatives/recommend the best strategy for remediation or cleanup; and
>
> RD/RA – to implement any remediation necessary prior to sale.

However, the BRAC environmental restoration program differs from the IRP since it also evaluates additional environmental issues such as asbestos, radon, transformers and underground storage tanks which must be addressed prior to transfer of property.

BRAC Cleanup Plan – A comprehensive and consolidated status and strategy for expedited environmental cleanup at BRAC installation where a BRAC Cleanup Team has been established. The BCP is intended to be the BCT's roadmap for execution. The BRAC Cleanup Plan is a living document which should be shared with the Restorations Advisory Board and community.

CERCLA – Comprehensive Environmental Response, Compensation and Liability Act of 1980, also know as "Superfund." Amended in 1986 by the Superfund Amendments and Reauthorization Act.

Community Relations Plan – Document based on community interviews that specified the community relations activities that the Army expects to undertake during a response action.

Contaminant – See Pollutant

Contracting Officer – Individual with the authority to enter into, administer and/or terminate contracts and make related determinations and findings.

Defense Environmental Restoration Program (DERP) – Provides centralized program management for the cleanup of DoD hazardous waste sites consistent with the provisions of CERCLA. The goals of the program are: (1) the identification, investigation, research and development and cleanup of contamination from hazardous substances, pollutants and contaminants; (2) correction of other environmental damage which creates an imminent and substantial endangerment to the public health, welfare or to the environment, and (3) demolition and removal of unsafe buildings and structures.

Defense State Memorandum of Agreement – The program developed to involve States and Territories in the cleanup of DoD installations in compliance with CERCLA. States are reimbursed for their actions in support of the DERP process.

Ecological Risk Assessment – The process to evaluate the likelihood that adverse ecological effects are occurring of may occur in site plant and animals, as a result of chemical, physical, or biological influences.

Facility (as stated in CERCLA) – Any building, structure, installation, equipment, pipe or pipeline (including any pipe into a sewer or publicly owned treatment works), well, pit, pond, lagoon, impoundment, ditch, landfill, storage container, motor vehicle, rolling stock, aircraft or any site or area where a hazardous substance has been deposited, stored, disposed of, placed or otherwise come to be located; but does not include any consumer product in consumer use or any vessel.

Feasibility Study – A study undertaken to develop and evaluate alternatives for remedial action.

Federal Agency Hazardous Waste Compliance Docket – A list, maintained by the U.S. Environmental Protection Agency of Federal hazardous waste treatment, storage, disposal and spill sites. The Docket includes information submitted by Army installations under Sections 3005, 3010, and 3016 of the Solid Waste Disposal Act and Sections 103 and 120 of CERCLA.

Field Sampling Plan – Document that provides guidance for all field work by defining in detail the sampling and data-gathering methods to be used on a project. Part of the Sampling and Analysis Plan that is prepared prior to any non-emergency site sampling activities.

Hazard Ranking System – Method used by the U.S. Environmental Protection Agency to identify sites for inclusion on the National Priorities List, and to prioritize National Priorities List sites for funding by Superfund. The original HRS was revised in December 1991 and is now referred to as HRS2.

Hazardous Substance (as stated in CERCLA) – Any substance designated pursuant to Section 311(b)(2)(A) of the Clean Water Act; any element, compound, mixture, solution or substance designated pursuant to Section 102 of CERCLA; any hazardous wastes having the characteristics identified under or listed pursuant to Section 3001 of the Solid Waste Disposal Act (but not including any waste the regulation of which under the Solid Waste Disposal Act has been suspended by Act of Congress); any toxic pollutant listed under Section 307(a) of the Clean Water Act; any hazardous air pollutant listed under Section 112 of the Clean Air Act; and any imminently hazardous chemical substance or mixture with respect to which the EPA Administrator has taken action pursuant to Section 7 of the Toxic Substances Control Act. The term does not include petroleum, including crude oil or any fraction thereof which is not otherwise specifically listed or designated as a hazardous substance in the first sentence of this paragraph, and the term does not include natural gas liquids, liquefied natural gas or synthetic gas usable for fuel (or mixtures of natural gas and such synthetic gas.)

Health Assessment – Assessment of existing risk to human health posed by National Priorities List sites, prepared by the Agency for Toxic Substances and Disease Registry.

Human Health Risk Assessment – Characterizes the nature and extent of potential adverse impacts form contaminants found in the air, soils, and/or water at the site. The risk assessment process synthesizes available data on exposure of specified receptors and the toxicity of contaminants to estimate the associated risk to human health.

Institutional Controls – Those actions that an Installation Commander can take to limit access to areas of contamination.

National Contingency Plan – Plan established by CERCLA that provides for efficient, coordinated and effective response to discharges of oil and releases of hazardous substances, pollutants and contaminants in accordance with CERCLA and the Clean Water Act. Its full title is "National Oil and Hazardous Substance Pollution Control Plan" and is found at 40CFR 300.

National Priorities List – A list, compiled by the U.S. Environmental Protection Agency, of high priority sites, identified primarily by Hazard Ranking System score, for remediation under CERCLA.

Natural Attenuation – The naturally occurring processes in soil and groundwater environments that act without human intervention to reduce the mass, toxicity, mobility, volume, or concentration of contaminants in those media.

Operable Unit (as stated in the National Contingency Plan) – A discrete portion of a remedial response that by itself eliminates or mitigates a release, threat of a release or pathway of exposure and that requires no additional action to accomplish its objective. The cleanup of a site can be divided into a number of operable units, depending on the complexity of the problems associated with the site. Operable unites may consist of any set of actions performed over time or any actions that are concurrent but located in difference parts of a site.

Pollutant and Contaminant (as stated in the National Contingency Plan) – Any element, substance, compound or mixture, including disease-causing agents, which after release into the environment and upon exposure, ingestion, inhalations or assimilation into any organism, either directly from the environment or indirectly by ingestion through food chains, will or may reasonably be anticipated to cause death, disease, behavioral abnormalities, cancer, genetic mutation, physiological malfunctions (including malfunctions in reproduction) or physical deformations, in such organisms or their offspring. The term does not include petroleum, including crude oil or any fraction thereof which is not otherwise specifically listed or designated as a hazardous substance under Section 101(14, will or may reasonably be anticipated to cause death, disease, behavioral abnormalities, cancer, genetic mutation, physiological malfunctions (including malfunctions in reproduction) or physical deformations, in such organisms or their offspring. The term does not include petroleum, including crude oil or any fraction thereof, which is not otherwise specifically listed or designated as a hazardous substance under Section 101(14)(a) through (f) of CERCLA, not does it include natural gas, liquefied natural gas or synthetic gas of pipeline quality (or mixtures of natural gas and such synthetic gas). For purposes of Subpart E (Hazardous Substance Response) of the National Contingency Plan, the term pollutant or contaminant means any pollutant or contaminant that may present an imminent and substantial danger to public health or welfare.

Potentially Responsible Party – Current and former owners or operators and persons who may be accountable for having generated hazardous substances or were involved in transport, treatment or disposal of hazardous substances at a site under litigation.

Preliminary Assessment – Initial analysis of existing information to determine if a release may require additional investigation or action.

Presumptive Remedies – Preferred cleanup technologies for common categories of sites having similar characteristics, and are based on past experience in site remediation, as well as the EPA's scientific and engineering evaluation of performance data on technology implementation.

Quality Assurance Project Plan (as stated in the National Contingency Plan) – A written document associated with remedial site sampling activities, which presents in specific terms the organization (where applicable), objectives, functional activities, and specific quality assurance and quality control activities designed to achieve the data quality goals of specific project or continuing operation. The quality assurance project plan is prepared for each specific project or continuing operation (or group of similar projects or continuing operations). Part of the Sampling and Analysis Plan that is prepared prior to any non-emergency site sampling activities.

Record of Decision – Documentation of a final remedial response action decision at a National Priorities List site.

Release (as stated in CERCLA) – Any spilling, leaking, pumping, pouring, emitting, emptying, discharging, injecting, escaping, leaching, dumping or disposing into the environment (including the abandonment or discarding of barrels, containers and other closed receptacles containing any hazardous substance or pollutant or contaminant), but excludes (A) any release which results in exposure to persons solely within a workplace, with respect to a claim which such persons may assert against the employer of such persons, (B) emissions from the engine exhaust of a motor vehicle, rolling stock, aircraft, vessel, or pipeline pumping station engine, (C) release of source, byproduct, or special nuclear material from a nuclear incident, as those terms are defined in the Atomic Energy Act of 1954, if such release is subject to requirements with respect to financial protection established by the Nuclear Regulatory Commission under Section 170 of such Act or, for the purposes of Section 104 of this title or any other response action, any release of source byproduct, or special nuclear material from any processing site designated under Section 102(a)(1) or 302(a) of the Uranium Mill Tailings Radiation Control Act of 1978, and (D) the normal application of fertilizer.

Remedial Action or Remedy (as stated in CERCLA) – Actions consistent with permanent remedy taken instead of or in addition to removal actions in the event of a release or threatened release of a hazardous substance into the environment, to prevent or minimize the release of hazardous substances to that they do not migrate to cause substantial danger to present or future public health or welfare or the environment. The term includes, but is not limited to, such actions at the location of the release as storage, confinement, perimeter protection using dikes, trenches, or ditches, clay cover, neutralization, cleanup of released hazardous substances and associated contaminated materials, recycling or reuse, diversion, destruction, segregation of reactive wastes, dredging or excavations, repair or replacement of leaking containers, collection of leachate and runoff, on-site treatment or incineration, provision of alternative water supplies and any monitoring reasonable required to assure that such actions protect the public health and welfare and the environment. The term includes the costs of permanent relocation of resident and businesses and community facilities where the President determines that, alone or in combination with other measures, such relocation is more cost-effective than and environmentally preferable to the transportation, storage, treatment, destruction or secure disposition off site of hazardous substances, or may otherwise be necessary to protect the public health or welfare; the term includes off site transport and off site storage, treatment, destruction, or secure disposition of hazardous substances and associated contaminated materials.

Remedial Action Process – Identification, evaluation, decision-making and design and construction steps required to implement control measures. The remedial action process may lead to remedial actions, removals or decisions to take no further action.

Remedial Investigation – Process undertaken to determine the nature and extent of the problem presented by a release which emphasizes data collection and site characterization. The remedial investigation is generally performed concurrently and in an interdependent fashion with the feasibility study.

Removal (as stated in CERCLA) – The cleanup or removal of released hazardous substances from the environment, such actions as may be necessary taken in the event of the threat of release of hazardous substances into the environment, such actions may be necessary to monitor, assess and evaluate the release or threat of release of hazardous substances, the disposal of removal material, or the taking of such other actions as may be necessary to prevent, minimize or mitigate damage to the public health or welfare or to the environment, which may otherwise result from a release or threat of release. The term includes, in addition, without being limited to, security fencing or other measure to limit access, provision of alternative water supplies, temporary evacuation and housing of threatened individuals not otherwise provided for, action taken under Section 104(b) of this Act and any emergency assistance which may be provided under the Disaster Relief Act or 1974.

Response Action – An action involving either a short-term removal action or a long-term removal response that may include, but is not limited to, removing hazardous materials from a site for treatment or containment; containing the waste safely on-site; treating the waste safely on-site; treating the waste on-site; and identifying and removing the source of contamination and halting further migration of contaminants.

Restoration Advisory Board – The Restoration Advisory Boards are replacing Technical Review Committees. The Restoration Advisory Board is a forum of representatives of the DoD, USEPA, State and local governments, and public representatives of the potentially affected community. Members can provide input to the DoD's environmental restoration program at operating or Base Realignment and Closure installations.

Sampling and Analysis Plan – Document composed of a Quality Assurance Project Plan and Field Sampling Plan that is prepared prior to site sampling activities.

Site – A location on an installation where hazardous wastes have been stored, disposed, spilled or otherwise released into the environment. A site includes land and water resources where they are contaminated by the release, and it includes any structures, earthworks or equipment that are clearly associated with the release. Where multiple sites may contribute to contamination of an aquifer or a common land area, the contaminated resource may be identified as a site that is distinguished from the sites where the release occurred. A site is the basic unit for planning and implementing response actions.

Site Close-Out – When all phases of the remedial activities at a site have been completed and no further action is warranted, the site will enter the Site Close-Out Phase.

Site Health and Safety Plan – Document that specifies policies and procedures for ensuring the health and safety of personnel working at a site.

Site Inspection – On-site inspection to determine whether there is a release or potential release and the nature of the associated threats. The purpose is to augment the data collected in the preliminary assessment and to generate, if necessary, sampling and other field data to determine if further action or investigation is appropriate.

Subpart X Permit Application – Subpart X regulations under RCRA were issued in 1987 to address issues relating to EPA's authority to issue permits in both new and existing facilities. Prior to the issuance of Subpart X, many existing miscellaneous units operated under 40 CFR 265, Subparts P and Q. Interim status units are subject to EPA permit authority to apply Subpart X standards until states receive authorization to permit these types of units.

Technical Assistance for Public Participation – If members of a Technical Review Committee or Restoration Advisory Board become dissatisfied with the Army's ability to provide technical support, the members may seek independent technical assistance to contribute to the public's ability to participate in the restoration program. The Army provides funding for Technical Assistance for Public Participants.

Technical Review Committee – Committee composed of Army and EPA officials, State and local authorities and a public representative of the potentially affected community that reviews and comments on response actions and proposed actions at Army sites on or proposed for the National Priorities List or other major sites (sites that present a significant threat ot human health, welfare or the environment or cause public controversy).

Third Party Site – Privately or municipally owned storage, treatment and disposal sites that received hazardous wastes either from disposal contractors hired by the Army or directly from the Army. The Army, as a potentially responsible party, is designated as the third party in cases where enforcement actions to recover costs of cleanup is initiated. EPA, as the first party, cannot sue the Army to recover such costs, but non-Federal potentially responsible parties, as the second party, can.

APPENDIX C - SUPERFUND SITES WITH UXO CONTAMINATION

Compiled by the Military Toxics Project from DoD and EPA Documents

Aberdeen Proving Ground .... Edgewood and Aberdeen, MD
Adak Naval Facility* .... Adak, Alaska
Alabama Army Ammunition Plant* .... Childersburg, AL
Allegany Ballistics Laboratory .... Mineral County, WV
Andersen Air Force Base .... Yigo, GU
Army RD&E Command (Picatinny Arsenal) .... Rockaway Township, NJ
Bangor Naval Submarine Base .... Silverdale, WA
Cornhusker Army Ammunition Plant .... Hall County, NB
Dahlgren Naval Surface Warfare Center .... Dahlgren, VA
F. E. Warren Air Force Base .... Cheyenne, WY
Fort Devens* .... Fort Devens, MA
Fort George G. Meade* .... Fort Meade, MD
Fort McClellan* .... Anniston, AL
Fort Ord* .... Marina, CA
Fort Pickett* .... Blackstone, VA
Fort Richardson .... Anchorage, AK
Fort Ritchie* .... Fort Ritchie, MD
Fort Sheridan* .... Fort Sheridan, IL
Fort Totten* .... Bayside, NY
Fort Wainwright .... Fairbanks, AK
Fort Wingate* .... Gallup, NM
Grissom Air Force Base* .... Peru, IN
Guam Naval Activities* .... Apra, GU
Hastings Groundwater Contamination Site
(Blaine Naval Ammunition Depot) .... Hastings, NE
Indian Head Naval Surface Warfare Center .... Indian Head, MD
Iowa Army Ammunition Plant .... Middletown, IA
Jefferson Proving Ground* .... Madison, IN
Joliet Army Ammunition Plant .... Wilmington, IL
Lake City Ammunition Plant .... Independence, MO
Letterkenny Army Depot* .... Franklin County, PA
Lone Star Army Ammunition Plant .... Texarkana, TX
Longhorn Army Ammunition Plant .... Karnack, TX
Louisiana Army Ammunition Plant .... Doyline, LA
MacDill Air Force Base* .... Tampa, FL
Malmstrom Air Force Base* .... Great Falls, MT
Mare Island Naval Shipyard* .... Vallejo, CA
Milan Army Ammunition Plant .... Milan, TN
Nebraska Ordnance Plant .... Mead, NB
Pantex Plant .... Pantex Village, TX
Parris Island Marine Corps Recruit Depot .... Parris Island, SC
Patuxent River Naval Ordnance Center .... Lexington Park, MD
Port Hadlock Naval Ordnance Center .... Port Hadlock, WA
Pueblo Chemical Depot* .... Pueblo, CO
Rocky Mountain Arsenal .... Adams County, CO
Sangamo Electric Dump/Crab Orchard National ....
Wildlife Refuge .... Caterville, IL
Savanna Depot Activity* .... Savanna, IL
Sierra Army Depot* .... Herlong, CA
Tobyhanna Army Depot .... Tobyhanna, PA

Tooele Army Depot* .... Tooele, UT
Umatilla Chemical Depot* .... Hermiston, OR
U.S. Disciplinary Barracks .... Lompoc, CA
Warminster Naval Air Warfare Center Aircraft Division .... Warminster Township, PA
Weldon Spring Ordnance Works .... Weldon Spring, MO
West Virginia Ordnance Works .... Pt. Pleasant, WV
White Oak Naval Surface Warfare Center* .... Silver Spring, MD
Wurtsmith Air Force Base* .... Oscods, MI
Yorktown Naval Weapons Station .... Yorktown, VA

*closed bases

APPENDIX D - SITES WITH INTERIM PERMITS FOR OB/OD

Compiled by Military Toxics Project through Freedom on Information Act Requests to EPA Regions

EPA Region	Facility
II	Naval Weapons Station Earle, Wall Township, NJ U.S. Army Armament R & D, Picatinny Arsenal, NJ Fort Drum Department of the Army, NJ U.S. Military Academy, West Point, NJ U.S. Navy Atlantic Fleet Weapons Training, Vieques, PR
III	U.S. Navy Indian Head, MD U.S. Army Aberdeen Proving Ground, MD U.S. Army Letterkenny Army Depot, Chambersburg, PA National Guard Ft. Indiantown Gap, Annville, PA U.S. Navy, Dalgren, VA U.S. Navy, Yorktown, VA U.S. Marines, Quantico, VA U.S. Army, Radford, VA Allegany Ballistics, Rocket Center, WV
IV	U.S. Army Anniston Army Depot, AL U.S. Army Redstone Arsenal, AL Killgore Corporation, TN U.S. Holston Army Ammunition Plant, TN U.S. Milan Army Ammunition Plant, TN Eastman (Corp.), TN AEDC, TN Ensign Bickford Co., KY KYD 985 115 591 USMC Camp Lejeune, NC U.S. Army Airborne, Fort Bragg, NC MCAS Cherry Point, NC MCAS Beaufort, SC Naval Weapons Station, Charleston, SC Shaw AFB, Poinsett Range, Sumter, SC McDonnel Douglas, Titusville, FL Olin Ordnance, St. Marks, FL U.S. AFB Cape Canaveral, FL U.S. DOE Pinellas, Largo, FL U.S.NAS Demolition Key, FL U.S. Navy Submarine Support Base U.S. AFB Moody, Warner Robins, GA
V	U.S. Air Force K.I. Sawyer AFB, Gwinn, MI Austin Powder Co., Red Diamond Plant, MacArther, OH
VI	Pine Bluff Arsenal, Pine Bluff, AR U.S. Army Join Reserve Training Center, Ft. Polk, LA U.S. Army Red River Depot, Texarkana, TX U.S. Army Camp Bullis, San Antonio, TX Lone Star Army Ammunition Plant, Texarkana, TX Lackland AFB, San Antonio, TX U.S Army Fort Hood, TX Naval Weapons Industrial Reserve Plant, Texarkana, TX

EPA Region	Facility
VI	Holloman AFB, Alamogordo, NM Kirtland AFB, Albuquerque, NM U.S. Army Fort Polk, LA White Sands, Missile Range, NM U.S. Army Fort Bliss, Los Cruces, NM U.S. Air Force Melrose Range, NM McAlester Army Ammunition Plant, OK Barksdale AFB, LA
VII	Kansas Army Ammunition Plant, Parsons, KS Iowa Army Ammunition Plant, Middletown, IA
VIII	Fort Carson, CO Pueblo Chemical Depot, CO Malmstrom AFB, MT Minot AFB, ND
IX	Anderson AFB, Guam Beale AFB, CA Castle AFB, CA Davis Monthan AFB, AZ Edwards AFB, CA George AFT, CA Luke AFB, AZ Nellis AFB, NV Vandenberg AFB, CA U.S. Army Fort Ord, CA U.S. Army Fort Irwin, CA U.S. Army Hawthorne Army Depot, NV (2 units) U.S. Army Makua Military Reserve, HI U.S. Army Sierra Depot, CA U.S. Army Yuma Proving Ground, AZ U.S. DOE Nevada Test Site, NV U.S. DOE Sandia National Laboratory, CA USMC Air Station El Toro, CA (closed) USMC Camp Pendleton, CA U.S. NASA Jet Propulsion, Edwards, CA U.S. Navy China Lake Naval Air Weapons Station (2 units)
X	Yakima Fire Center, WA

APPENDIX E - TYPES OF UNEXPLODED ORDNANCE (UXO)

From "Unexploded Ordnance (UXO): An Overview," Federal Advisory Committee for the Development of Innovative Technologies, October 1996.

In the past century, all shapes, sizes, and types of explosive ordnance have been used in the U.S. for weapons system testing and troop training activities. The following types of UXO are those most likely to be encountered on active DoD sites and FUD and BRAC sites:

- Small arms munitions;
- Hand grenades;
- Rockets;
- Guided missiles;
- Projectiles;
- Mortars;
- Projected grenades;
- Rifle grenades;
- Submunitions;
- Bombs.

Ordnance is color-coded during manufacturing for identification purposes. However, color markings cannot be relied upon to identify UXO; markings can be altered or removed by weather or exposure to the environment. Instead, other features should be used to identify UXO. The following sections describe the basic features and characteristics associated with each general type of UXO. Additional information can be found in Unexploded Ordnance (UXO) Procedures (U.S. Army 1994).

Small Arms Munitions

Small arms munitions contain projectiles that are 0.5 inches or less in caliber and no longer than approximately 4 inches. They are fired from various sizes of weapons, such as pistols, carbines, rifles, automatic rifles, shotguns, and machine guns. Generally, the shell casings of small arms munitions are made from brass or steel. Although the hazards associated with these UXO are much less than for other munitions, unexploded small arms munitions may explode if thrown into a fire or struck with a sharp object such as a nail.

Hand Grenades

Hand grenades are small explosive- or chemical-type munitions that are designed to be thrown at short range. Various classes of grenades may be encountered as UXO, including fragmentation, smoke, and illumination grenades. All grenades have three main parts: a body, a fuze with a pull ring and safety clip assembly, and a filler. Fragmentation grenades are the most common type of grenade used. They have a metal or plastic body filled with an explosive material. When the filler explodes, the body of the grenade or a metal fragmentation sleeve breaks into small, lethal, high velocity fragments. These grenades use a burning delay fuze that functions 3 to 5 seconds after the safety lever is released. Other grenades may be made of metal, plastic, cardboard, or rubber and may contain explosives, white phosphorus, chemical agents, or illumination flares, depending on their intended use. Most use a burning delay fuze that functions 3 to 5 seconds after the safety lever is released, but some are activated instantly when the lever is released (smoke grenades).

Rockets

A rocket uses gas pressure from rapidly burning material (propellant) to transport a payload (warhead) to a desired location. Rockets can range from 11/2 to more than 15 inches in diameter, and they can vary from 1 foot to over 9 feet in length. All rockets consist of a warhead section, a motor section, and a fuze. They are unguided after launch and are stabilized during flight by canted nozzles at the base of the motor or fins attached to the motor. The warhead section of the rocket is the portion that produces the intended effect; it can be filled with explosives, toxic chemicals, white phosphorus, submunitions, riot-control agent, or illumination flares. Fuzes may be located in the nose of the rocket or internally between the warhead and motor. The fuzing on rockets can be impact, time-delay, or proximity fuzing. Impact fuzes function when they hit the target. Delay fuzes contain an element that delays explosion for a fixed time after impact. Proximity fuzes are intended to function when the rockets reach a predetermined distance from the target.

Guided Missiles

Guided missiles are similar to rockets however they are guided to their target by various systems. Some are wired-guided, and others are guided by internal or external radar or video. Guided missiles are usually stabilized by fins controlled by internal electronics. Internal proximity fuzes are used in guided missiles, which makes approaching them extremely dangerous. Also, fired guided missiles may still contain residual propellant that could ignite and burn violently.

Projectiles

Projectiles can range from approximately1 inch to 16 inches in diameter and from 2 inches to 4 feet in length. Projectile fuzes can be located in the nose or in the base. Like rockets, projectiles may be stabilized during flight by fins or bands fixed around the circumference of the projectile.

Mortars

Mortars range from approximately 1 inch to 11 inches in diameter and can be filled with explosives, toxic chemicals, white phosphorus, or illumination flares. Mortars generally have thinner metal casing than projectiles, but use the same types of fuzing and stabilization.

Projected Grenades

The most commonly used projected grenade is the 40 millimeter grenade. This grenade is also among the most commonly found UXO items. The 40mm grenade is about the same size and shape as a chicken egg. It contains high explosives and uses a variety of fuzes, including some of the most sensitive internal impact fuzing systems. Because of their relatively small size, 40mm grenades are easily concealed by vegetation. They are extremely dangerous and can explode if moved or handled.

Rifle Grenades

Rifle grenades look like mortars and range from about 9 to 17 inches in length. They may be filled with high explosives, white phosphorus, riot-control agent, illumination flares, or chemicals that produce colored screening smoke. Rifle grenades are fired from standard infantry rifles. They have an opening at the far end of a tube near the fin assembly that allows the rifle grenade to be placed on the barrel of a rifle. Rifle grenades rely on impact fuzing, which is located on the nose or internally behind the warhead.

Submunitions

Submunitions include bomblets, grenades, and mines filled with explosives or chemical agents. They may be antipersonnel, antimateriel, antitank, dual-purpose, incendiary, or chemical submunitions. Submunitions are typically spread over a large area by dispensers, missiles, rockets, or projectiles. Each of these delivery systems disperses the submunitions while still in flight, scattering the submunitions over an area. Submunitions are activated in a variety of ways, depending on their intended use. Some are activated by pressure, impact, or movement or disturbance. Others are activated in flight or when they come near metallic objects. Some submunitions contain a self-destruct fuze as a backup. The self-destruct time can vary from a couple of hours to several days. Submunitions are extremely hazardous because even very slight disturbances can cause them to explode. Some types of submunitions require stabilization to hit the target straight on. Stabilization can be provided through an arming ribbon, parachute, or fin assembly.

Bombs

Bombs range in weight from 1 to 3,000 pounds and in length from 3 to 10 feet. Generally, all bombs have the same components–a metal container, a fuze, and a stabilizing device. The metal container, or bomb body, holds the explosive or chemical filler and may consist of one piece or multiple pieces. Bombs use either mechanical or electrical fuzes, typically located in the nose or tail section, either internally or externally. Mechanical fuzes are generally armed by some type of arming vane. The arming vane operates like a propeller to line up all the fuze parts and thus arm the fuze. The fuzes may be configured as impact, proximity, or delay fuzes. Bombs are stabilized during flight by fin or parachute assemblies attached to the rear section of the bomb. These assemblies often detach from the bomb after impact.

Date: January 26, 2022

To: White House Environmental Justice Advisory Council (WHEJAC)

From: Tina Cordova
Tularosa Basin Downwinders Consortium

Re: Recommendation that WHEJAC requests that the Centers for Disease Control (CDC) as an addition to the Los Alamos Historical Document Retrieval and Assessment (LAHDRA) study the recently discovered spike in infant mortality in New Mexico after the Trinity test.

Good afternoon members of the Council and thank you for this opportunity to provide these comments. My name is Tina Cordova and I'm a native New Mexican, a Downwinder and a cancer survivor. I'm the fourth generation in my family to suffer with cancer since the Trinity bomb was detonated in south central New Mexico July 16, 1945. I grew up in a community about 45 miles the way the crows fly from the test site although we've identified families that lived as close as 12 miles. The US government has always controlled the messaging as it relates to the test and has said and continues to say that the area was remote and uninhabited and that no one lived here and no one was harmed. The truth is according to the 1940 census data there were about 15 thousand men, women and children living within a 50-mile radius to the Trinity Site and close to 500 thousand people living within a 150-mile radius. It is obvious from these numbers that it was neither remote nor uninhabited.

We've been dealing with the devastating health consequences for 77 years now without aid or assistance from the government. The people of New Mexico are the forgotten collateral damage of the first nuclear test. Most of us are indigenous people of color Natives or Mexicanos. We bury our loved ones on a regular basis and then another one of us is diagnosed with cancer. It's time to recognize that mistakes were made and correct the injustices perpetuated on American Citizens. To remain complacent in the face of knowing about the injustice of the nuclear testing and subsequent damage to the health of human beings renders a person complicit in that injustice. There is a moral and ethical imperative to right this wrong. There are bills before Congress now to Amend the Radiation Exposure Compensation Act that will expire in July of this year that would add us to the fund. The bills are House bill 5338 and Senate bill 2798.

While we bring these bills to the attention of the Council our recommendation centers around a different but related subject matter. In 2019 a paper was published in the Bulletin of Atomic Scientists entitled Trinity: "The most significant hazard of the entire Manhattan Project". The article focused on the high rate of infant mortality after the Trinity test. Since the article was published, we've been doing research and the record keeping of infant births and deaths in New Mexico in 1945 is quite incomplete. We have reason to believe that babies born and babies that died during this time frame were not well documented and the lack of accurate records may have understated the number of infant deaths. The spike may have been much worse than reported. It is time for the US government to thoroughly assess this issue and report on the findings. Did we have casualties from the Trinity test and were they our babies? If so, who will be the voice for these forgotten children potentially sacrificed in the testing of the first nuclear device.

We're here to appeal to the Council to reach out to the Centers for Disease Control who conducted a ten-year study called the Los Alamos Historical Document Retrieval and Assessment that addressed the radiation releases from the Manhattan Project and ask that they reopen the study to further research the infant mortality in New Mexico subsequent to the Trinity test. The information about infant mortality was not known to the CDC at the time they conducted their study. Without addressing the infant mortality, the LAHDRA study and report is incomplete at best. If babies died while the US Government looked the other way it is the most egregious offense imaginable and the people of New Mexico deserve to know the truth.

APPALACHIAN CITIZENS' Law Center

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February 4, 2022

Dear White House Environmental Justice Advisory Council,

My name is Courtney Rhoades and I am the Black Lung Organizer at Appalachian Citizens' Law Center based in Whitesburg, KY. ACLC is a non-profit law firm and political advocacy group focused on addressing the ongoing and legacy costs of coal mining in Central Appalachia. I appreciate the opportunity to submit a comment on behalf of the communities we work in and thank you for your consideration as you prepare recommendations.

For over a century, the mountains we call home have been valued by those outside of the region because of the mineral which lies below the surface. Though coal has provided good-paying jobs to the individuals in our communities, the boom in this industry has long passed. Today, the majority of coal has been extracted from Appalachia with no consideration for the impact of this industry on the mountains, its people, or their health. Black Lung Epidemic

Black lung was noticed in coal miners as early as the late 1880s but was not acknowledged fully as a disease until the mid-twentieth century.[1] Miners and widows spent decades fighting for recognition of what was happening to them, as miners coughed up black sputum, struggled to breathe the older they became, and even as some died at younger than expected ages. Though the Black Lung Disability Trust Fund was established in the 1970s, receiving benefits remains a drawn-out process for insufficient monthly payments and the funding mechanism for this fund remains unsustainable as the excise tax that funds the trust fund was once again slashed in half at the start of the new year.

Furthermore, the prevalence of black lung, an entirely preventable disease, has doubled nationwide since 2000.[2] Miners in Central Appalachia have been most affected with 1 in 5 veteran coal miners having black lung disease and 1 in 20 have the most severe and disabling form of this disease - Progressive Massive Fibrosis (PMF).[3] Though our region has been most impacted by this epidemic, other areas with coal could see the same numbers in the next few decades as coal resources decline due to continuously being mined out and if the silica standard for coal miners, who have the highest exposure for workers in the U.S, is not changed.

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[1] A. Derickson. *Black Lung: Anatomy of a Public Health Disaster*.
[2] American Journal of Public Health, 2018. "Continued Increase in the Presence of Coal Workers' Pneumoconiosis in the United States, 1970 - 2017." Blackley et al, AJPH, September 2018 <https://ajph.aphapublications.org/doi/abs/10.2105/AJPH.2018.304517>
[3] Ibid.

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Though black lung is not a traditional environmental issue, it is another example of the glaringly negative impact the coal industry has had on our region and nation for over a century and the ramifications of what happens when issues are continually ignored because of the power an industry has. An industry so powerful and important that when miners strike due to working conditions in the early 1900s, the national guard was sent in. An industry that has ravaged the communities that provided jobs and where it is now normal for folks to not consume the water that flows into their homes. Retired coal miners and widows now fight through local organizations called the Black Lung Associations to showcase the need for funding to the Trust Fund that covers benefits for 18,000 families. These groups also push for an improved silica standard in hopes of decreasing the number of cases of black lung in current and future miners and increased work opportunities not related to extractive industries in our communities.

I urge the White House Environmental Justice Advisory Council to recommend to the Whitehouse Council on Environmental Quality to remember those in the communities that have previously and continue to be negatively impacted by the coal industry as recommendations on future energy policy are prepared. We need this administration to prioritize the reinstatement and extension of the black lung excise tax that funds the Trust Fund since the Build Back Better Act failed to be passed in 2021. Passing S. 2810 and H.B. 6462 quickly would reinstate the black lung excise tax and prevent the fund from losing over $ 2.5 million a week.[4] There also needs to be confirmation of a director at MSHA who will create a silica standard that will decrease exposure, thus preventing future black lung cases in current and future miners.

Thank you for inviting me to submit a comment on behalf of my community and I look forward to working with you to improve the health and safety of residents in Appalachia.

Sincerely,

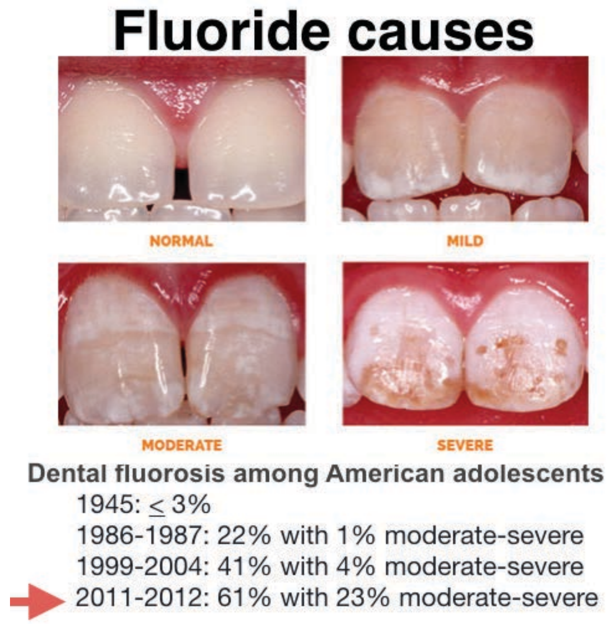
Courtney Rhoades
Black Lung Organizer

[4] Calculated based on 2021 data.

Dental Fluorosis - *a dental disaster*

When fluoridation first began, the proponents promised the American public that the only and worst risk from the program was that something less than 10% of children might have a few faint white spots on their 'cavity resistant' teeth which many would find adds an attractive sparkle.

Also of note, the researchers in the early fluoridation trials treated African American children differently from the outset, writing that it was common knowledge that "Negros" have stronger teeth more resistant to decay.



It didn't take long for those involved to realize that something was very wrong, but their reaction per 1962 memo that noted high rates of fluorosis that was doubled in the African American children emphasized protecting the fluoridation program.

As both the numbers of Americans and percentage of the population drinking fluoridated water swelled, dental fluorosis also grew, disproportionately and with worse severity in Black and Latino populations.

Dental fluorosis is a defect in the tooth due to cell death during the formative stages. Those with dental fluorosis have higher bone fractures as well as higher rates of learning disabilities. Dental fluorosis is the visible evidence of similar defects due to cytotoxic effects that occur in bones and brains during critical periods of development, i.e. prenatal, infancy and early childhood.

Dental fluorosis is also a leading indicator of **higher dental costs** as these unattractive and brittle teeth will likely result in costly veneers and crowns in young adulthood. Per 2011-2012 NHANES figures released in 2017, one in five (23%) American teens have extensive mottling and perhaps pitting on at least two fluorosed teeth due to childhood exposure.

RESOURCES

- 1962 Memo: http://fluoridealert.org/wp-content/uploads/1962_01_10_Blacks_Fluorosis.pdf
    - "Negros in Grand Rapids had twice as much fluorosis - indices 0.15 v. 0.35"
- 2005 CDC MMWR: https://www.cdc.gov/mmwr/preview/mmwrhtml/ss5403a1.htm
    - "Prevalence of enamel fluorosis has increased in cohorts born since 1980."
- 2010 CDC Report: https://www.cdc.gov/nchs/data/databriefs/db53.pdf
    - "(All levels of) dental fluorosis were higher among adolescents aged 12–15 in 1999–2004 than in 1986–1987."
- 2015 "Agua Potable o Veneno" (part 2 of 3): https://www.youtube.com/watch?v=RGswvGZPL-M
    - Ethnic Breakdown: http://www.cdc.gov/mmwr/preview/mmwrhtml/ss5403a1.htm#tab23
- 2017 Dental fluorosis is result of apoptosis: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5770627/
- 2018 Increase: http://jdh.adha.org/content/92/1/23
    - **Conclusion**: "There was a difference of 31.6% in dental fluorosis prevalence between 2012-2011 when compared to data from 2002-2001 in adolescents aged 16 and 17 years. The continued increase in fluorosis rates in the U.S. indicates that additional measures need to be implemented to reduce its prevalence."

May 23, 2018

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White House Environmental Justice Advisory Council
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Memorandum

TO: White House Environmental Justice Advisory Council (WHEJAC)

FROM: Emma Kurnat-Thoma, PhD, MS, RN, FAAN; EPA-HQ-AO-2021-0683-013

DATE: January 26, 2022

SUBJECT: *Laudato Si,* Climate Resilient Health Systems and Environmental Justice

SUMMARY

This memorandum provides summary examples of relevant environmental and social justice scientific literature and recommendation action items that are grounded on the Catholic Church teachings of *Laudato Si* (Laudato Si, 2022; Pope Francis, 2015). The LS response centers on 'integral ecology' which are the bonds between humans and the natural world that we all share, for 'everything is interconnected'.

This summary highlights the following:

- Importance of LS*,* integral ecology ethics, the presence and participation of faith and religious communities in developing and implementing local environmental justice strategic plans;
- The critical need for faith community leadership and participation in the generation, review, and strategic planning of strong science, which helps inform sound policy development for the common good (i.e. Georgetown University's Jesuit Philosophy, *LS* integral ecology principles);
- Summary of *LS* integral ecology-informed environmental justice topics, including: heat-related mortality; ecosystems alterations; human food and agricultural systems considerations; extreme weather events (EWE); air quality; mental health and violence.
- Summary of the 10 elements comprising the World Health Organization's *Operational Framework for Building Climate Resilient Health Systems* and exemplar *LS* healthcare system featuring climate sensitive strategic action plans informed by Catholic social teaching principles.
- Presentation of 3 climate resilience Recommendations to strengthen vulnerable and marginalized population inclusion via clinician-nursing workforce preparedness in U.S. healthcare systems.

ANALYSIS

Introduction

As we are all aware, climate change from global warming and its escalating impacts of widescale natural disasters and weather cycle alterations are placing human civilization at grave risk. Deadly heat waves, rising seas, drastically over-consumptive deforestation and farming practices, exploiting local populations and their lands, increased droughts, wildfires, floods, mass extinctions, widespread oceanic coastal and inland watershed destruction, collectively impact our ecosystems, healthcare institutions and food supply chains. For example, the integrity of delineated boundary habitats becomes altered and facilitates transmission of zoonotic diseases to humans, as demonstrated by the SARS-Co-V2 outbreak and COVID-19 pandemic. Most heartbreaking of all, the poor, vulnerable and the marginalized members of our human family shoulder the greatest burden. Example sequelae include: increased morbidity/mortality from toxic exposures, infectious diseases and environmental crises, forced community displacements, secondary violence and trauma due to migration, long-term inability to thrive in stable employment and agricultural opportunities for which to support their families, and removal from decision-making processes impacting their collective humanity (Sorondo & Ramanathan, 2016).

Background—Laudato Si

Pope Francis authored the encyclical document *Laudato Si* in 2015 which serves as a powerful contribution by the Catholic Church into the worldwide conversation about global environmental health and mitigating the current climate crisis (Pope Francis, 2015). *Laudato Si* response centers on "integral ecology", which defines the nature of shared bonds between humans and the natural world, for 'everything is interconnected'. One of Georgetown University's Jesuit Philosophy core mission priorities is "Care for our Common Home" which is based on LS integral ecology. Integral ecology combines environmental justice and social justice and recognizes the moral imperative to stabilize the science of climate change by supporting the most vulnerable and weakest members of our human family (Sorondo & Ramanathan, 2016). Specifically, by working to address profound wealth inequalities driving health disparities experienced by the poor, leaders can help address and counteract environmental and climate crisis challenges (Sorondo, Frumkin, & Ramanathan, 2018).

For example, healthcare leaders can statistically measure and model the scale of a climate-related disaster. But in the absence of strategic cooperation frameworks based on genuine dialogue and financial support that empowers vulnerable communities with needed tools, knowledge, and resources, it will be difficult to achieve true justice (supporting the weak). Our plans to meet the challenge posed by environmental justice must include healthy politics in the form of a genuine and mutual fraternity in local communities progressing up to international governance frameworks, for which the inclusion of faith communities is critical (Ramanathan, 2022). The spiritual message of LS consists of two prongs—hearing and responding to the cry of the earth and to the cry of the poor (*Laudato Si*, 2022). This means that the resources and gifts of our Earth must be preserved and protected, in addition to reducing poverty and lifting up individuals and communities in the societal peripheries.

Discussion

Healthcare system and policy leaders can use LS' integral ecology to complement leading strategic planning models to address the climate crisis by linking COVID-19 responses in the design of stronger, more equitable, and climate resilient healthcare institutions and workplaces. During crises, integral ecology assists policy leaders to place the strongest science at the service of those who are the most profoundly vulnerable.

This briefing memo uses the World Health Organization's (WHO) *Operational Framework for Building Climate Resilient Health Systems* to select relevant scientific exemplars of populations, interventions, and outcomes that align with LS integral ecology priorities (WHO, 2015). Examples of LS priority areas when planning combined environmental and social justice to achieve climate resilient healthcare systems in the U.S. include, but are not limited to: elderly adults and nursing homes, nursing mothers, young children and adolescents, those with pre-existing medical conditions, individuals belonging to ethnic, racial, gender, tribal minority groups and families, and those under the Federal Poverty Thresholds.

- ***Heat-Related Mortality:*** Adverse effects in response to excessive heat waves including excessive deaths and emergency room admissions for those who are most vulnerable. A recent example are the record-high temperatures of 116ºF (46.7ºC) in the northwestern US in 2021 which were 42ºF (23.3ºC) hotter than average daily temperatures and disproportionately impacted elderly adults (Schramm et al, 2021). Fallout from this event recommended that state Health Departments develop and implement climate health (i.e. heat response) plans for at-risk neighborhoods and populations, especially through near-real time monitoring of health conditions by use of consistent data-sharing practices that accurately guide policy and allocation of available resources to where they are most needed (CDC, 2022).
    - Sample interventions: establish climate change occupational health exposure standards, public education, heat-health action plans that protect highest-risk populations.

- ***Ecosystems Alterations:***
    - ***Food and Water Born Diseases:*** Higher temperatures, variable precipitation, higher sea and freshwater temperatures carry increased risks of food-and water borne diseases (i.e. diarrheal illness) through accelerated microbial growth, survival, persistence, transmission and pathogen virulence (Jones et al, 2020). Under-investments in U.S. water quality and infrastructure and higher rates of water quality enforcement non-compliance disproportionately impact minority communities, largely poor communities, and Tribal communities (National Academies of Sciences, Engineering, and Medicine, 2021a).
        - Sample interventions: ensure adequacy of and/or strengthen food and water quality control standards combined with compliance capability resources; develop national-regional data monitoring and models that better account for environmental, human, and pathogen genetic interaction variations to rapid and dramatic climate changes.
    - ***Vector Born Diseases:*** Accelerated parasite replication and prolonged transmission seasons result in increased risks of vector-borne diseases (Carter et al, 2018). The Southeast is particularly at risk for increased vector borne diseases from *Aedes aegypti* mosquitoes (i.e. dengue, chikungunya, Zika, etc.) due to higher population densities and human and environmental factors such as standing pools of water in man-made structures.
        - Sample interventions: develop enhanced disease surveillance systems, warning systems during high-risk conditions and seasons, particularly at geographic margins, and ensure adequate provision, compliance with vaccination standards.

- ***Human Food and Agricultural Systems:*** Increased temperatures and precipitation changes result in a higher risk of lower food production and lower access to food in poverty stricken areas contributing to undernutrition, malnutrition and correlative sequelae (Lipper et al, 2020). When combined with infectious disease threats, it results in extended exposures to harsh outdoor conditions in physiologically unsafe conditions for workers, decreases labor productivity and exacerbates food shortages. Excessive chronic health impacts of malnutrition in poverty regions experiencing decreased food production and access severely impacts child development (Tirado et al, 2013). It can also mean loss of traditional sources of food for rural and tribal communities depending on the local produce of the land or sea.

- Sample interventions: ensure adequate seasonal nutrition screening for high-risk and poverty-afflicted communities; strengthen integrated food security, nutrition and health interventions in environmentally fragile areas (i.e. drought conditions and high heat seasons, poverty, decreased health-care access/rural health areas); ensure communities have sustainable development plans in place that are driven by the needs of local communities that are culturally appropriate. Example: recent U.S. food system challenges presented by the COVID-19 pandemic and strategic policy responses (Leone et al, 2020).

- ***Extreme Weather Events (EWE):*** International scientific consensus identifies risks of increased injury, disease, and death will occur as a result of increased severity and frequency of storms and EWE including but not limited to: hurricanes, dust, tornadoes, coastal oceanic and river flooding during excessive precipitation events, extreme cold and snow amounts, extreme heat, drought, wildfires, and loss of fresh water reservoirs (Bikomeye et al, 2021). These events will impact both urban and rural healthcare facilities, trigger increased morbidity and mortality and result in healthcare services shortages, economic hits through lost revenue and jobs, delayed and cancelled procedures, increased operation expenses, supply chain disruptions, evaluations, and increase harms to staff and patients (Bikomeye et al, 2021, p. 14).
    - Sample interventions: ensure health care facilities have sufficient climate risk infrastructure planning in place for resilience during EWE; ensure early warning and early action systems are fully functioning, particularly for vulnerable small isolated towns, cities, communities, populations that are at higher risk due to decreased socio-demographic status and increased compositions of racial and ethnic minority groups; ensure integrated chronic disease management plans for vulnerable populations are included in a facility's disaster plans especially in areas experiencing drastic physician shortages and needing the support of advanced practice nurses—such as communities disproportionately impacted by decreased socio-demographic resources and primarily comprised of racial and ethnic minority groups. Example: The Joint Commission-Centers for Medicare & Medicaid Services Emergency Management Standards (TJC Standards, 2022; National Academies of Science, Medicine, & Engineering, 2021b).

- ***Air Quality:*** Climate changes (increased temperature variations, cloud cover, precipitation, wind, humidity) trigger increased risks of wildfires and dust impacting air quality. Air pollutant exposures to ground-level ozone (O_3) and fine particles (<2.5 µm, $PM_{2.5}$), can have dramatic effects on human health (symptom exacerbations of and premature morbidity/mortality from myocardial infarctions, strokes, respiratory diseases, chronic obstructive pulmonary disease, acute lower respiratory tract infections, asthma, lung cancer), (Fann et al, 2021). Appropriate dust control planning and management measures for protection of ambient air quality standards require multisector coordination from a variety of local to national stakeholders at multiple stages of development and hybrid approaches may be necessary to successfully meet regional and national target goals (National Academies of Science, Medicine & Engineering, 2020).
    - Sample interventions: working with key collaborating sectors to ensure adequate air quality standards can be met by the most vulnerable and resource-poor communities; facilitate community resources and foster hybrid capabilities for meeting those standards, especially during high-risk exposure forecasts; plan for increased healthcare utilization demand for high-risk segments of the population during adverse weather conditions and/or high-risk seasons favoring wildfires, dust, wind-storm exacerbations (i.e. allergic diseases including asthma, vulnerable populations with higher prevalence of key respiratory and cardiopulmonary health comorbidity diagnoses).

- ***Mental Health and Violence:*** Preliminary research on the effects of EWEs and climate disasters present profound threats to personal safety and safety of loved ones. For example, wildfires wreak havoc on individual victims, families, and communities, including: acute injuries and mortality (healthcare access); displacement of victims and their families; loss of workplaces, jobs, and livelihoods; chaos on the surrounding communities imposed by response teams to ameliorate the crisis; development and exacerbation of medical conditions such as asthma, diabetes, gastrointestinal disorders, and psychiatric disorders and symptoms (anxiety, depression, Post-Traumatic Stress Disorder, mood, concentration, sleep and academic disturbances) in individuals and families and can impact child and adolescent development trajectories over 2-years post-wildfire (Silveira et al, 2021; To et al, 2021).
    - Sample interventions: ensure disaster preparedness and response plans sufficiently take into account the needs of mental health/disability/access to care for affected patients for a prolonged recovery trajectory; ensuring adequate healthcare access needs of disaster and trauma-exposed individuals, children, and families especially in highly forecasted disaster-prone areas; establish community monitoring mechanisms for impacted individuals, families and surrounding communities during EWEs.

- ***WHO Framework Elements and Exemplar Healthcare System Using LS:***
    - The WHO *Operational Framework for Building Climate Resilient Health Systems* stipulates 10 elements to facilitate evidence-based decision making and implementation standards when designing policies to protect nation-level population health and promote climate change resilience, including:
        - 1) health workforce preparedness;
        - 2) leadership and governance;
        - 3) vulnerability, capacity and adaptation assessment;
        - 4) integrated risk monitoring and early warning capacity;
        - 5) health and climate research;
        - 6) climate resilient and sustainable technologies and infrastructure;
        - 7) management of environmental/social determinants of health;
        - 8) climate informed health programming;
        - 9) emergency preparedness and management; and
        - 10) climate and health financing models.

    - The Catholic Health Association designed and implemented a climate-friendly hospital system operational plan consistent with LS integral ecology principles, including: energy efficiency; building designs responsive to local climate conditions and optimized for reduced energy and resource use; alternative fuel and energy sources; alternative transportation options; sustainably grown local foods for staff and patients; safe alternatives to bottled water; and robust recycling options and reduced waste incineration (Catholic Health Association, 2022).

RECOMMENDATIONS

In accordance with the WHO *Operational Framework for Building Climate Resilient Health Systems* and the moral imperative outlined by *Laudato Si,* more comprehensive responses to supporting U.S. population health of vulnerable and marginalized individuals, families, communities and healthcare systems before, during, and after climate emergencies is needed to achieve environmental justice and advance health equity (*Laudato Si*, 2022; WHO, 2015).

1. Re-review the recently approved The Joint Commission-Centers for Medicare & Medicaid Services Emergency Management Standards (TJC Standards, 2022; National Academies of Science, Medicine, & Engineering, 2021b) to ensure adequate nurse workforce preparedness.

This includes development of healthcare access plans for inclusion of vulnerable and marginalized populations in critical access hospitals and hospitals in very high-risk climate forecast areas for EWEs (such as wildfires, tornadoes, hurricanes, floods, etc.).

2. Ensure adequate healthcare and nurse workforce planning for environmental justice and integration of social determinants of health factors to better support vulnerable and marginalized individuals in climate-crisis impacted communities for which there are documented physician shortages. The Future of Nursing 2020-30 Consensus Report provides detailed plans for community and population health nurse models of care for a wide range of health equity applications (National Academies of Science, Medicine, & Engineering, 2021b).
3. Ensure the HHS Climate Action Plan for climate resilience and readiness incorporates clinician healthcare workforce planning in collaboration with multisector stakeholders for healthcare system preparedness including advocacy organizations such as: American Medical Association, American Nurses Association, and the American Hospital Association.

CLOSING

We are all deeply connected and interrelated, and should recognize our profound connections to the poor and vulnerable. Scientific strategies for COVID-19 recovery will only work to our collective advancement if we actualize integral ecology in our local communities, states, countries, and international governance structures to build resilient staffs, organizations, and multisector collaborations to build a just world that serves the weakest among us.

Thank you for the thoughtful consideration of these comments. I applaud WHEJAC and the Biden/Harris Administration’s commitment to these important matters in the service of all humanity.

Respectfully Yours in the Service of the Common Good,

Emma Kurnat-Thoma, PhD, MS, RN, FAAN

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U.S. FEDERAL COURT

ACTION NO. 17-CV-02162

FOOD AND WATER WATCH, *et al.* v. U.S. EPA

EXPERT DECLARATION OF PHILIPPE GRANDJEAN, MD, DMSc

PREPARED ON BEHALF OF PLAINTIFFS

20 May 2020

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I, Philippe Grandjean, MD, DMSc, declare that:

1. I am a physician and environmental epidemiologist and serve as both an Adjunct Professor at the Harvard T.H. Chan School of Public Health, and Professor and Chair of Environmental Medicine at the University of Southern Denmark.

2. I was asked by Plaintiffs' counsel to provide an evaluation of the neurological health risks associated with the exposure to fluoride in drinking water.

I. SUMMARY OF QUALIFICATIONS

3. A complete summary of my qualifications and publications can be found in my Curriculum Vitae, which has been marked as Plaintiffs' Exhibit 3 and attached herein.

4. Over the past 25 years, my research has focused on developmental exposures to environmental chemicals and the association with adverse health effects in children, as described in my book "Only One Chance" (2013) published by Oxford University Press.

5. My research has been entirely funded by public sources, mainly the National Institutes of Health (NIH). In 2003-2007, my study of children's vulnerability to environmental immunotoxicants was supported by the U.S. Environmental Protection Agency (EPA). My current funding as principal investigator includes grants from the Superfund Research Program at the National Institute of Environmental Health Sciences and the U.S. Agency for Toxic Substances and Disease Registry (ATSDR).

6. I have published about 500 scientific papers, of which most are research articles in international scientific journals with peer review. My h-index in the Web of Science data base is 70, and my work is cited in scientific journals well over a thousand times every year. Seven of my articles published in the last 10 years have earned the attribute "Highly Cited Paper," i.e., they received enough citations to place them in the top 1% of published papers in the field.

7. My study on the neurodevelopmental effects of prenatal mercury exposure in a birth cohort from Faroe Islands was relied upon by the EPA as the critical study for the Agency's derivation of a

Reference Dose for methylmercury (EPA 2001).

8. I have served as a technical advisor to the World Health Organization on environmental health issues, including five occasions where I was elected Rapporteur. I have also served on, sometimes chaired, or acted as rapporteur for, expert committees under the auspices of the EPA, ATSDR, Food & Drug Administration (FDA); NIH; White House Office of Science and Technology Policy; International Agency for Research on Cancer (IARC), European Commission, European Environmental Agency, European Food Safety Authority, and other organizations. I have also served for over 30 years as Consultant in Toxicology for the Danish Ministry of Health.

9. I am (Founding) Editor-in-Chief of the journal *Environmental Health* (since 2002), which ranks among the most frequently cited journals in the field. I also serve or have served on editorial boards of about a dozen journals within medicine, environmental science, and toxicology. As editor and as reviewer for other major journals, I frequently evaluate manuscripts on environmental epidemiology and toxicology.

10. I have received various awards and honors for my scientific work, including the John R. Goldsmith Award from the International Society for Environmental Epidemiology, which is given to investigators for "sustained and outstanding contributions to the knowledge and practice of environmental epidemiology."

11. I have been retained as an expert on the impact of environmental chemicals on human health by government bodies, including the U.S. Department of Justice (on behalf of the EPA) and the State of Minnesota.

12. I first began studying fluoride in 1980 at the suggestion of Dr. Irving J. Selikoff, who was my mentor at the Mt. Sinai School of Medicine during my two-year Senior Fulbright Scholarship. Upon returning to Denmark, I initiated a series of studies on a cohort of workers who had been occupationally exposed to fluoride. I have remained involved in fluoride research since that time and have published 16 peer-reviewed reports on fluoride exposure and toxicity in humans.

13. In 1984, I drafted the Environmental Criteria Document on fluoride for the World Health

Organization (WHO). Ten years later, I drafted the Criteria Document for an occupational exposure limit value for fluorine for the European Commission. In 2006, I served as a reviewer of the National Research Council's report *Fluoride in Drinking Water: A Scientific Review of EPA's Standards*.

14. During the past 10 years, my research work on fluoride has focused on its developmental effects on the brain. In 2012, I published a meta-analysis of the epidemiological studies on fluoride and IQ (Choi et al. 2012); in 2015, I published an epidemiological study of fluoride and IQ in China (Choi et al. 2015); and, in December of 2019, I published an updated review of fluoride neurotoxicity, which relied in part on the work that I have performed in this case (Grandjean 2019).

15. In addition to my work on fluoride, I also have expertise in Benchmark Dose (BMD) analysis. My experience doing BMD analysis started about 20 years ago in connection with my research on the neurodevelopmental effects of methylmercury in the Faroe Islands that was selected as the critical study for risk assessment by the EPA. Based on this research, the EPA provided me with a contract to produce a BMD analysis of the data, which I carried out in collaboration with my biostatistician colleagues, Dr. Esben Budtz-Jorgensen and Professor Niels Keiding. The EPA relied on this BMD analysis to establish the safe level for methylmercury exposure in the U.S. (U.S. EPA 2001).

16. In 2009, I served on an expert panel that assisted the European Food Safety Authority (EFSA) in developing a guidance document on BMD analysis titled "Use of the Benchmark Dose (BMD) Approach in Risk Assessment."

17. In 2013, Dr. Budtz-Jorgensen and I extended our BMD methodology in collaboration with the International Pooled Lead Study Investigators, which was peer-reviewed and published in the journal *Risk Analysis* (Budtz-Jorgensen et al. 2013). As part of this analysis, we developed a BMD for lead and IQ by analyzing pooled data from multiple different cohort studies. The paper was co-authored by leading scholars on lead neurotoxicity, including Drs. David Bellinger and Bruce Lanphear.

18. More recently, Dr. Budtz-Jorgensen and I conducted an advanced BMD analysis on perfluorinated chemicals, which was published in 2018 in the peer-reviewed journal *PLOS One* (Budtz-Jorgensen and Grandjean 2018). In total, our achievements on BMD approaches and applications have

been published in seven articles so far in international biostatistical and biomedical journals.

19. In addition to my scientific training, I remain mindful, of the importance of translating the results of epidemiological studies in a way that can facilitate public participation in making informed decisions to protect their health, even prior to a "final proof" of causation being available; a final proof that, all too often, has come too late to protect the public from harm, as reviewed most recently in the monograph on *Late Lessons of Early Warnings*, published by the European Environment Agency (EEA Report No 1/2013), for which I served as an editor. As Dr. Selikoff once impressed upon me, "Never forget that the numbers in your tables are human destinies, although the tears have been wiped away."

II. SUMMARY OF OPINIONS

20. The weight of epidemiological evidence leaves no reasonable doubt that developmental neurotoxicity is a serious human health risk associated with elevated fluoride exposure, including those occurring at the levels added to drinking water in fluoridated areas. The IQ losses associated with community water fluoridation are substantial and of significant public health concern.

21. Application of the Benchmark Dose (BMD) methodology to the recent prospective birth cohort data shows that the level of fluoride added to water in fluoridation programs greatly exceeds the science-based limit needed to protect against developmental neurotoxicity.

22. The systematic review conducted by Dr. Ellen Chang, when corrected for its biases and errors in judgment, further supports my opinions on the neurotoxic risks posed by elevated fluoride exposure.

III. SUMMARY OF METHODOLOGY

A. Weight of the Evidence

23. I conducted a weight of the evidence assessment of available research on fluoride neurotoxicity, with an emphasis on the epidemiology. While I place the greatest weight on the strong epidemiological evidence, I also consider toxicokinetics, experimental toxicology data, and background

principles of brain development as part of my comprehensive analysis.

24. My review focuses on the evidence that carries the greatest weight which, as generally accepted, emphasizes the recent prospective cohort studies.

25. My methodology follows the general approach applied by the EPA, in the sense that I did a weight of the evidence analysis that focuses on the best available science (e.g., EPA 2017).

26. In light of my familiarity with the scientific literature on fluoride neurotoxicity, I did not conduct a formal systematic review on this occasion. Instead, my conclusions rely on a comprehensive and thorough review supplemented by a Benchmark Dose analysis of the recent prospective data.

27. I have read and considered the systematic review conducted by Dr. Ellen Chang, which mostly relies on the same evidence and which further confirms and supports my assessment of the literature. My opinions are thus fully informed by the insights offered by a formal systematic search of the literature.

B. Factors Considered When Assessing Epidemiological Literature

28. In evaluating the weight of the evidence, the question must be asked what each study could potentially reveal, given the design and choice of study parameters, including such factors as the precision of the exposure assessment. In the field of epidemiology, there is a well-known bias toward the null, e.g., from imprecise assessment of the exposure, of which epidemiologists (and readers of epidemiology reports) need to be careful, especially when human health is at stake (EPA 2005).

29. The following Table highlights common causes of bias toward the null in epidemiological studies, i.e., reasons that a study might not show the existence of a risk that indeed is present, though hidden due to the bias. While biases in the opposite direction also exist, they are usually of much less significance (Grandjean 2013).

Table 1. Causes of bias toward the null in epidemiology studies (Grandjean 2013a).

Inadequate statistical power in small studies
Lost cases and inadequate follow-up for long-term effects
Exposed or otherwise inappropriate comparison (control) group
Exposure misclassification
Insensitive or imprecise outcome measures
Failure to adjust for confounders with effects in the opposite direction
Disregarding vulnerable subgroups
5% probability level to minimize risk of false positives (Type I error)
20% probability level to minimize risk of false negatives (Type II error)
Pressure to avoid false alarm

30. Studies that do not show a statistical significance are sometimes called "negative," although this term is misleading. Joint analyses of several such studies may well show a significant difference or trend.

31. Observational studies will rarely if ever provide definitive proof of causation, and it is always possible for someone to raise doubts and uncertainties that require additional or improved data to resolve (Michaels 2008). It is important to recognize, however, that the presence of uncertainties often tends to cause underestimations of actual risks, not the opposite. This issue is of importance especially regarding substances that have not yet been studied in the detail desired or cannot be examined in randomized clinical trials. Many unfortunate past errors in regard to industrial chemicals have shown that initial assessments were often erroneous and led to an underestimation of the true risks (European Environment Agency 2001 & 2013).

32. In the context of developmental neurotoxicity, I place greatest weight on prospective studies of population-based birth cohorts followed over time (Grandjean et al. 2008; Grandjean & Landrigan 2014). Birth cohorts are crucial because it is not just the dose that can matter but also the timing of the dosing in regard to the developmental stage of the subjects (Grandjean et al. 2008; Grandjean et al. 2019). Follow-up studies of birth cohorts can thus reveal with greater certainty the

impacts of exposures incurred during early life stages.

C. Benchmark Dose Methodology

33. As part of my assessment in this case, I worked with my biostatistician colleague Dr. Budtz-Jørgensen on a BMD analysis of the prospective cohort data on fluoride and IQ using the same peer-reviewed method that we used for lead (Budtz-Jorgensen et al. 2013).

34. The statistical uncertainty in the BMD estimation is taken into account by calculating its lower one-sided 95% confidence limit, which is called the benchmark dose level (BMDL). The BMDL is then used as the point of departure for calculation of the exposure limit, by dividing the BMDL by an uncertainty factor (usually fixed at 10) to obtain a protective Reference Dose (RfD) or tolerable exposure (EFSA 2009; EPA 2012).

D. Materials Relied Upon

35. In my assessment, I relied upon my existing knowledge of the scientific literature (with citations to specific studies noted in my reports), my own meta-analysis of the epidemiological studies of fluoride and IQ (Choi et al. 2012), the more recent meta-analysis by Duan (2018), all available prospective studies, as well as the reviews by NRC (2006) and NTP (2016).

36. I also considered studies provided by counsel,[1] many of which I was already familiar with, and conducted supplemental searches on PubMed, including searches to see if there were any significant epidemiological studies published that I might have overlooked.

37. A complete list of the studies I relied upon is provided in my expert reports.

IV. GENERAL CONSIDERATIONS

A. Emergence of Brain Development as Vulnerable Target

38. Evidence has been accumulating over several decades that industrial chemicals can cause

[1] I understand that these studies were provided to EPA's experts as well, including Dr. Chang.

neurodevelopmental disorders that include learning disabilities, sensory deficits, developmental delays, and cerebral palsy (NRC 2000), and current evidence also relates to other neurodevelopmental deficits, such as attention deficit hyperactivity disorder (ADHD) (Bennett et al. 2016). Subclinical stages of these conditions also appear to be common, and the suspicion of a link between neurotoxic chemical exposures and widespread neurobehavioral damage has increased since it was first raised by research demonstrating that lead is particularly toxic to the developing brain across a wide range of exposures (Baghurst et al. 1987; Dietrich et al. 1987; Landrigan et al. 1975; Needleman et al. 1979).

39. The developing human brain is inherently much more susceptible to injury caused by toxic agents than the brain of an adult. This susceptibility reflects the fact that in the nine months of prenatal life the human brain must evolve from a strip of cells along the dorsal ectoderm into a complex organ comprised of billions of precisely located, highly interconnected and specialized cells. Optimal brain development requires that neurons move along precise pathways from their points of origin to their assigned locations, that they establish connections with other cells near and distant, and that they generate intercommunications in meaningful ways (Dobbing 1968; Rice and Barone 2000; Rodier 1995).

40. All of these processes must take place within a tightly controlled time frame, in which each developmental stage must be reached on schedule and in the correct sequence. Due to the extraordinary complexity of human brain development, windows of unique susceptibility to toxic interference occur that have no counterpart in the mature brain, or in any other organ. Because of the unique structure of the human brain and its advanced function, no other species shows similar degree of developmental vulnerability. Thus, if a developmental process in the brain is halted or inhibited, there is little potential for later repair, although plasticity will allow some compensation, and the consequences are therefore likely to be permanent (Dobbing 1968; Rice and Barone 2000).

41. To test chemicals for developmental neurotoxicity, standardized protocols have been developed using rodent models (OECD 2007). However, they may not necessarily be sufficiently sensitive, as rodent brains are far less complex than human brains, and intrauterine brain development is completed at a stage where the human fetal brain is still rapidly developing *in utero* for several more weeks with possible continued impact from maternal transfer of neurotoxicants (Bal-Price et al. 2018).

42. During fetal development, the placenta can offer some protection against unwanted chemical exposures, but it is not an effective barrier against most environmental neurotoxicants (Andersen et al. 2000), including fluoride (NRC 2006). In addition, the blood-brain barrier, which protects the adult brain from many toxic agents, is not completely formed until about 6 months after birth (Adinolfi 1985).

43. Postnatally, the human brain continues to develop, and the period of heightened vulnerability therefore extends over many months through infancy and into early childhood. While most neurons have been formed by the time of birth, growth of glial cells and myelinization of axons continue for several years and is not complete until late teenage years (Rice and Barone 2000; Rodier 1995).

44. The susceptibility of infants and children to industrial chemicals is further amplified by their relatively increased exposures in regard to body weight, their augmented absorption rates, and diminished ability to detoxify many exogenous compounds as compared to adults (Ginsberg et al. 2004; NRC 1993).

45. In 2005, when I evaluated the evidence of industrial chemicals regarding developmental neurotoxicity, only five substances (arsenic, lead, methylmercury, polychlorinated biphenyls, and toluene) fulfilled our criteria for causal relationship in humans (Grandjean and Landrigan 2006). Eight years later, when we reassessed the evidence, we added six more substances, including fluoride (Grandjean and Landrigan 2014), based on new evidence that had emerged.

46. Our 2014 assessment was focused on *hazard* (i.e., whether fluoride causes developmental neurotoxicity in humans), not on *risk* (i.e., the exposure level at which this hazard may occur). Substantial new evidence published since that time, particularly the prospective birth cohort studies, now permit an assessment of risk.

B. Toxicokinetics During the Fetal Period

47. In my assessment, I considered the toxicokinetics of fluoride, with a particular focus on the uptake, distribution and retention during the fetal period.

48. It is well accepted that fluoride crosses the placenta and reaches the fetus from the mother's blood stream (NRC 2006; WHO 2006).

49. The first documentation of placental transfer in humans was the observation in 1974 (Shen and Taves 1974) that fluoride concentrations in maternal and cord serum correlated well, with the cord blood showing slightly lower concentrations. These findings were replicated in 1986 (Ron et al. 1986), with results suggesting minor deviations depending on gestational age. A more recent study from an area with water-fluoride levels of 0.4-0.8 mg/L showed that cord serum contained about 80% of the concentrations occurring in maternal serum (Opydo-Szymaczek and Borysewicz-Lewicka 2007). Consistent with this, French researchers measured fetal blood concentrations of fluoride after the mothers were administered a small dose of sodium fluoride, and the elevations were statistically significantly higher (2.6 µmol/l) than in a control group (less than 1 µmol/l) (Forestier et al. 1990).

50. A recent study from scientists at the University of California San Francisco (UCSF) further confirms the placental transfer of fluoride (Uyghurturk et al. 2020). In this study, fluoride concentrations were measured in the urine, blood, and amniotic fluid among pregnant women in fluoridated and non-fluoridated areas of Northern California. Each additional 0.1 mg/L of fluoride in water was associated with a significant increase in the fluoride levels in the amniotic fluid ($p < 0.001$), thus confirming the

transplacental passage of fluoride.

51. As would be expected, given the undeveloped nature of the blood-brain barrier during the fetal period, laboratory studies of animals exposed to prenatal fluoride have found significant elevations of fluoride in the brain (McPherson et al. 2018; Mullenix et al. 1995). Similarly, in aborted human fetuses, fluoride concentrations in the brain have been shown to be higher in geographic areas with endemic fluorosis as compared to controls at lower exposures (Du et al. 2008; He et al. 2008).

C. Toxicological Findings

52. Neurotoxicity is a documented hazard of fluoride exposure in laboratory animals (NRC 2006), which supports the plausibility of fluoride causing neurotoxic effects in humans.

53. One of the first U.S. reports on experimental fluoride neurotoxicity emerged when a new method was developed for computerized surveillance of rat behavior. Fluoride was selected for a test of the new methodology and showed clear neurotoxicity (Mullenix et al. 1995). The authors noted that the behavioral effects they observed in the rats are indicative of fluoride’s potential ability to cause IQ deficits in humans. This assessment, which was made prior to the publication of any studies of fluoride and IQ in western journals, proved prescient.

54. Since the Mullenix study was published in 1995, many additional animal studies have documented neurochemical and anatomic changes in the brains of fluoride-treated animals. By 2006, the NRC concluded that there was enough neurochemical and anatomic data to conclude that fluoride interferes with brain functions by both direct and indirect means.

55. Among prominent adverse outcome pathways, the NRC concluded that fluoride is an endocrine disrupter that can affect thyroid function at intake levels as low as 0.01 to 0.03 mg/kg/day in individuals with iodine deficiency (NRC 2006).[2] Thyroid toxicity supports the plausibility of fluoride

[2] Large epidemiological studies published since the NRC report suggest that thyroid dysfunction is a relevant risk at elevated fluoride exposures in fluoridated communities, especially in

neurotoxicity because availability of thyroid hormone is crucial for optimal brain development (Rovet 2014).

56. At the time of the NRC's review, there was little data yet available on fluoride's impact on behavior and cognition in animals, but considerable data has since been published. In 2016, the National Toxicology Program (NTP) conducted a systematic review of these behavioral/cognitive studies (NTP 2016). Although NTP did not consider any of the neurochemical/anatomical effects, it still concluded that the evidence is "suggestive of an effect on learning and memory" (NTP 2016, p. vii). The NTP characterized its confidence in the evidence as "moderate" for adult studies, and "low" for the few available developmental studies.

57. Additional animal research on learning/memory has been published subsequent to the NTP review, and most of it has reported adverse effects. As is often the case, the animal studies on learning/memory have limitations or discrepancies but given the general consistency in their findings they continue to be *at least* "suggestive" of fluoride being a neurocognitive hazard.

V. EPIDEMIOLOGICAL STUDIES (CROSS-SECTIONAL)

A. Neurotoxicity from Occupational Fluoride Exposure

58. The neurotoxicity of chemicals is often first discovered from workplace exposures (Grandjean and Landrigan 2006), which are later followed by case reports that involve children or pregnant women from the general population (Grandjean 2013). The same is true of fluoride.

59. Although largely overlooked or ignored, Roholm first reported evidence of nervous system effects in his seminal study of cryolite workers in Copenhagen (Roholm 1937): "The marked frequency of nervous disorders after employment has ceased might indicate that cryolite has a particularly harmful effect on the central nervous system" (p. 178). The nervous system effects reported by Roholm included tiredness, sleepiness, indisposition, headaches, and giddiness (p. 138).

adults with iodine deficiency (Malin et al. 2018; Peckham et al. 2015).

60. My own mortality study of the cryolite workers studied by Roholm showed an excess of violent deaths (Grandjean et al. 1985), but information on the causes of death did not allow any conclusions on deaths from nervous system disease.

61. One of the challenges with occupational studies of fluoride-exposed workers is that the fluoride exposure usually occurs as part of a mixture. In the 1940s, scientists at the Manhattan Project recorded CNS effects in workers exposed to uranium hexafluoride gas (UF_6). They observed a "rather marked central nervous system effect with mental confusion, drowsiness and lassitude as the conspicuous features" and attributed it to the fluoride rather than uranium (Ferry 1944; Mullenix 2005).

62. Consistent with the observations of the Manhattan Project scientists, published case reports have highlighted difficulties with concentration and memory accompanied by general malaise and fatigue following occupational fluoride exposures (Spittle 1994).

63. More recently, skeletal fluorosis in workers was found to be associated with gradually progressive effects on the normal function and metabolism of the brain and other aspects of the nervous system (Duan et al. 1995), and application of neuropsychological tests (i.e., WHO's Neurobehavioural Core Test Battery) have reported significant associations between workplace fluoride exposures and cognitive problems (Guo et al. 2008; Yazdi et al. 2011).

64. The available evidence from occupationally exposed workers supports the neurotoxicity of fluoride but does not allow any detailed consideration of its dependence on dose, timing, and duration.

B. Neurotoxicity in Endemic Fluorosis Areas

65. Fluoride toxicity has received particular attention in China, where widespread dental fluorosis indicates pervasive high exposures (Wang et al. 2012). Areas with high prevalences of dental (and skeletal) fluorosis are known as "endemic fluorosis" areas.

66. Although microbiologically safe, water supplies from wells, small springs or mountain

sources have created pockets of increased fluoride exposures near or within areas of low exposures, thus representing optimal settings for epidemiological research because only the fluoride exposure would likely differ between nearby neighborhoods. In addition, rural families in China move much less frequently than U.S. families, thus facilitating assessment of impacts from long-term exposures. Chinese researchers took advantage of this fact and published their findings, though mainly in Chinese journals, and according to the standards of science at the time. The early research dates to the 1980s but has not been widely cited, in part because of limited access to Chinese journals, in part because the notion of adverse effects from fluoride intake has often been considered unwelcome.

67. Most of the studies on fluoride neurotoxicity from China, and other countries (i.e., India, Iran, and Mexico), have focused on IQ measures as the endpoint of concern, with the clear majority of these studies reporting inverse associations (i.e., higher levels of fluoride exposure are associated with lower IQ).

68. Many of the studies from China have significant limitations, including lack of information on covariates, missing information on study details, assessment of exposures on a community basis, and use of cross-sectional study designs. The reports have also tended to be relatively brief and simple in design. These deficiencies, which in some cases are rather severe, limit the conclusions that can be drawn, but are unlikely to explain the almost uniformly consistent inverse associations that have been reported.

69. While most of the endemic fluorosis studies have rather simple designs and may have failed to control for confounding factors of possible importance, they also have important strengths, including: 1) Stable populations with stable water-fluoride concentrations; many of the studies specifically limited the populations to those who had lived in the community their entire life. 2) Unlike in the U.S., children in rural China have very little exposure to fluoridated dental products (Zhu et al.

2003), thus making water a more important and reliable metric of fluoride exposure. 3) The studies in endemic fluorosis areas that have controlled for or excluded key confounding factors (arsenic exposure, iodine deficiency, parental education) were still capable of identifying clear associations between elevated fluoride exposure and cognitive deficits (Choi et al. 2012).

70. I will discuss the Chinese research on IQ in fluoride-exposed communities in more detail, but I begin first with studies that have examined other neurotoxicity endpoints, including neuropathological outcomes in aborted fetuses, neurobehavioral effects during infancy, and cognitive deficits and other neurological problems in adults.

1. <u>Neurotoxic Endpoints in Fetuses and Neonates</u>

71. In brain tissue obtained from aborted fetuses in endemic fluorosis areas, electron microscopy showed retarded cell growth in the cerebral cortex, with substantial cytology changes (He et al. 2008). A similar study used stereology to examine nerve cell numbers and volumes in fetal brain tissue and found lower densities (Du et al. 2008). A third study focused on neurotransmitters and receptors and found deviations that suggested neural dysplasia (Yu et al. 2008). Another study of aborted fetal brain tissue showed similar neurotransmitter results (Dong et al. 1993). These studies are consistent with prenatal fluoride exposure causing anatomic and biochemical changes in the fetal brain, as concluded by the NRC. A limiting factor, however, is that the elevated fluoride exposure in these studies came primarily from coal burning, which may have contributed other contaminants besides fluoride that were not assessed.

72. The impact of elevated fluoride in drinking water on neurological behavior in 91 neonates was assessed by Li et al. (2008). The study found that neonates born in an endemic fluorosis area (water-fluoride concentrations of 1.7 - 6.0 mg/L) scored more poorly on the standard Neonatal Behavioral Assessment, and that visual and auditory responses were also deficient, as compared to controls from

areas with less than 1 mg/L (Li et al. 2008). These findings are again consistent with the notion that fluoride can affect the brain during the prenatal period, although neonatal neurological assessments can be somewhat imprecise and may be only weakly predictive of subsequent brain development.

73. In a separate study, infants from an endemic area were examined at ages 3, 6, 9 and 12 months and scored significantly lower in mental and psychomotor development indices than those of the control group (Chang et al. 2017). The exposed group also showed lower birth weight, and it is unclear whether this difference can lead to confounding or if a lower birth weight is a concomitant effect of the fluoride exposure. As with the fetal neuropathology studies, the source of fluoride exposure in this study was coal, not water, which limits the conclusions that can be drawn due to the potential for confounding.

2. Neurotoxic Endpoints in Adults

74. Studies in China using cross-sectional designs have also found cognitive problems and neurological symptoms in adults with skeletal fluorosis living in endemic fluorosis areas. Using neuropsychological tests, including the Wechsler scale, 49 adult fluorosis patients (it is not clear whether the patients were from a coal- or waterborne fluorosis area) were compared with controls and showed deficits in language fluency, recognition, similarities, associative learning, and working memory (Shao et al. 2003). Likewise, cognitive impairment in elderly subjects was clearly elevated in a waterborne fluorosis area, although within-group assessment of urine-fluoride concentrations failed to show a clear gradient of effect (Li et al. 2016). Excess occurrence of neurological symptoms has also been recorded in both adults and children from waterborne fluorosis areas, with headaches being the primary manifestation (Sharma et al. 2009).

3. Childhood IQ

75. As noted above, most of the epidemiological studies on fluoride neurotoxicity have focused on IQ scores in childhood. In 2012, my colleagues and I published a meta-analysis of the

available 27 studies, most of which were published in China[3] (Choi et al. 2012). Because these published studies were conducted independently, we used meta-analysis—a quantitative, formal, statistical technique—to systematically review and assess these published research studies to derive conclusions about the neurotoxicity of fluoride. The outcome of the meta-analysis includes a more precise estimate of the association than any individual study that contributes to the pooled analysis. The variability or heterogeneity in study results was also examined. We did not attempt to generate any dose-response relationship, and the fluoride concentrations were used only for definitions of high and low (reference) groups in each study.

76. Among the 27 studies we reviewed, two involved populations exposed to fluoride from coal burning (Guo et al. 1991; Li et al. 2010); the rest of the studies involved exposure to fluoride through drinking water containing fluoride from soil minerals. The Combined Raven's Test – The Rural Edition in China (CRT-RC) was used to measure the children's intelligence in 16 studies. Other intelligence measures included the Wechsler Intelligence scale (3 studies), Binet IQ test (2 studies), Raven's test (2 studies), Japan IQ test (2 studies), Chinese comparative intelligence test (1 study), and the mental work capacity index (1 study). As each of the intelligence tests used is designed to measure general intelligence, we used data from all eligible studies to estimate the possible effects of fluoride exposure on the children's intelligence. We conducted a sensitivity analysis restricted to studies that used similar tests to measure the outcome (specifically, the CRT-RC, Wechsler Intelligence test, Binet IQ test, or Raven's test), and an analysis restricted to studies that used the CRT-RC. We also performed an analysis that excluded studies with possible concerns about co-exposures, such as iodine status and arsenic exposure, or with non-drinking water fluoride exposure from coal burning, without finding appreciable differences, as described below.

[3] Two of the 27 studies included in the analysis were conducted in Iran (Poureslami et al. 2011; Seraj et al. 2006), otherwise the study cohorts were populations from China.

77. The levels of fluoride exposure in the studies we examined, while higher than those associated with fluoridation programs (0.7 mg/L), are not as high as some have claimed. A surprising number of commentators, including the EPA, have only mentioned the *highest* concentration examined in the studies (11.5 mg/L) (Allukian et al. 2018; EPA 2018), although this high concentration occurred in only one of the 27 studies. The majority of studies that reported the water-fluoride level in the exposed group had between 1.5 and 4 mg/L, which is elevated, but only modestly.[4] Similarly, Duan's more recent meta-analysis of waterborne fluoride exposures reported that 18 of 27 studies addressed water-fluoride concentrations below 4 mg/L, and IQ reductions were observed at elevated concentrations of 1 to 2 mg/L (Duan et al. 2018 Table 2).

78. Among the 27 studies, all but one showed random-effect standardized mean difference (SMD) estimates that indicated an inverse association, ranging from -0.95 to -0.10 (one study showed a slight, non-significant effect in the opposite direction). The overall random-effects SMD estimate (and the 95% confidence interval, CI) were -0.45 (-0.56, -0.34). Given that the standard deviation (SD) for the IQ scale is 15, an SMD of -0.45 corresponds to a loss of **6.75 IQ points**.[5] I shall return to this result later. Among the restricted sets of intelligence tests, the SMD for the model with only CRT-RC tests and drinking-water exposure was lower than that for all studies combined, but the difference was not significant, and heterogeneity remained at a similar magnitude in the restricted analyses.

79. Several studies (Hong et al. 2001; Lin et al. 1991; Wang et al. 2001; Wang et al. 2007; Xiang et al. 2003; Zhao et al. 1996) reported other risk factors, such as iodine status, and exposure to arsenic or lead, both neurotoxicants, and our sensitivity analyses showed similar associations between

[4] The fluoride levels in the control groups in the studies often approximated the concentrations (~0.7 mg/L) used in fluoridation programs. Some ill-informed commentators have mistakenly interpreted this to mean that these control levels are thereby safe. This is false. The control groups are not being compared to *lower* or zero fluoride groups, and, as such, provide no information about the safety, or lack thereof, of the control values.

[5] The effect size we found is consistent with the prior meta-analysis of Tang (2008), who reported a mean difference of 5.03 IQ points between the high- and low-fluoride areas.

high fluoride exposure and the outcomes even after exclusion of these studies. Although large tracts of China have superficial fluoride-rich minerals, there is little, if any, likelihood of contamination by other neurotoxicants that would be consistently associated with fluoride concentrations in drinking water and thereby systemically confound the results. For example, follow-up testing documented lower levels of blood-lead concentrations and waterborne arsenic in the high-fluoride community than the control (Xiang et al. 2003; Xiang et al. 2003; Xiang et al. 2013). In some instances, therefore, potential co-exposure to other neurotoxicants may cause reverse confounding (i.e., may attenuate the real relationship between fluoride and IQ), as we have documented for methylmercury exposure from seafood (Choi et al. 2008).

80. Additional IQ studies in endemic fluorosis areas have been published since our 2012 review. As with the previous studies, these newer studies continue to replicate the consistent inverse association between fluoride exposure and IQ, although many—but not all—suffer from similar limitations. Two of the studies reported linear relationships between urinary fluoride excretion and IQ (one study also included plasma-fluoride) among children living in areas with mean water-fluoride contents of 1.4 mg/L and 1.5-2.5 mg/L (Cui et al. 2018; Zhang and Cheng 2015).[6] Another study published since our meta-analysis is the one I conducted with colleagues in China, which I will now discuss.

81. To ascertain the validity of the Chinese reports on fluoride neurotoxicity, we carried out a pilot study in Sichuan using methods commonly applied in neurobehavioral epidemiology (Choi et al. 2015). The children examined had lived in their respective communities since conception. Although we examined only 51 children, our results are consistent with elevated fluoride exposure being a cause of cognitive deficits. Interestingly, negative associations were found for cognitive function tests regarding

[6] These results are consistent with the findings of Ding et al. (2011), who reported a dose-response relationship between urine-fluoride concentrations (range = 0.24-2.84 mg/L) and reduced IQ in a population without any severe dental fluorosis (Ding et al. 2011).

all three measures of fluoride exposure. One was the known water-fluoride concentration at the residence where the child was born and had grown up, another was the child's morning urine-fluoride after having ingested fluoride-free water the night before (neither measure reached formal statistical significance as a predictor of cognitive deficits). The strongest and statistically significant association was seen with the degree of dental fluorosis that served as a marker of early-life fluoride exposure. While the milder forms of dental fluorosis have been considered a cosmetic effect (Aoba and Fejerskov 2002; WHO 2006), our study suggested that fluorosis can serve as a useful marker of early fluoride exposure in studies of neurodevelopmental toxicity.[7]

C. Studies of Fluoride and ADHD in North America

82. Four epidemiological studies have investigated the relationship between fluoride and ADHD behaviors in North America, the most important of which is the prospective cohort study by Bashash (2018). Two of the other three studies examined ADHD-related outcomes in the Canadian Health Measures Survey (CHMS) (Barberio et al. 2017; Riddell et al. 2019).[8]

83. In 2017, Barberio et al. examined two cycles of the CHMS to investigate the relationship between randomly measured urine-fluoride levels (in 3-to-12-year-old children) and parental reports or self-reported learning disabilities. When the two cycles of the CHMS were combined (both including at least 1,100 subjects), unadjusted urine-fluoride was significantly correlated with an increased incidence of learning disabilities. However, this effect lost its statistical significance after controlling for urine dilution by creatinine and specific gravity. The authors concluded that there was no robust association

[7] A prior study that was co-authored by my colleague David Bellinger failed to observe a relationship between dental fluorosis and behavior, as determined from parental questionnaires (Morgan et al. 1998). Due to several weaknesses, the conclusions were cautious and, in the authors' wording, "cannot lay this issue to rest." The relationship between dental fluorosis and neurobehavioral deficits is an issue that thus requires further study, including the possibility that the relationship is only apparent for fluorosis of certain teeth that share windows of susceptibility that overlap the windows of susceptibility for developmental neurotoxicity.

[8] The third study (Malin and Till 2015) was an ecological study that found an association between ADHD and water fluoridation in the U.S. This association was not robust, however, as it lost its significance after adjustment for altitude, although this adjustment is questionable.

between fluoride exposure and reported learning disability among Canadian children at the ages studied.

84. A more sophisticated study using the same CHMS data has now been completed and shows a significant association between fluoridated water and ADHD diagnoses/symptoms (Riddell et al. 2019). The latter study controlled for more potential covariates than Barberio and focused on an older subset of children (6 to 17 years old). Riddell's focus on an older group of children is an improvement because 90% of children with ADHD are diagnosed after age 6 (Riddell et al. 2019). Riddell also focused specifically on ADHD symptoms and diagnoses, rather than the broader category of "learning disabilities." The Riddell team also analyzed fluoride in water as well as in urine and conducted regression analyses to test the association with specific ADHD parameters: i.e., ADHD diagnosis and the hyperactivity/inattention score on the Strengths and Difficulties Questionnaire (SDQ).

85. After adjustment for covariates, including lead exposure, Riddell and colleagues found that fluoridation of the home water supply significantly increased the risk of an ADHD diagnosis. An increase in water-fluoride by 1 mg/L was associated with a (statistically significant) 6-fold higher odds of an ADHD diagnosis in the 710 children known to rely on community water, although this association was not replicated using urine concentrations that may have been more variable. Similar tendencies were seen for the SDQ scores of hyperactivity/inattention, especially among the older youth (not covered by the Barberio study).

86. With its individual exposure data, more specific ADHD outcomes in adolescents, and large effect size, the Riddell study, along with Bashash et al. (2018) that I will discuss below, provide additional weight to the evidence of fluoride being a neurotoxicant at current levels of exposure in fluoridated areas.

VI. EPIDEMIOLOGICAL STUDIES (PROSPECTIVE)

A. Prospective Cohort Studies with Individual Assessment of Prenatal Exposure

87. The most reliable evidence of developmental neurotoxicity is obtained through prospective studies that include real-time recording of information about exposure in early life followed by subsequent clinical assessments of the child. (Grandjean & Landrigan 2014; Grandjean 2008). In our meta-analysis we recommended that prospective studies be conducted to formally evaluate dose-response relations based on individual-level measures over time, including more precise prenatal measurements (Choi et al. 2012). Five such studies have now been conducted, and they have each found significant adverse associations between prenatal fluoride exposure and neurodevelopmental harm (Bashash 2017; Bashash 2018; Green 2019; Valdez-Jiminez 2017), with an additional study finding an association between fluoride exposure during early infancy and IQ loss (Till 2020). The quality of these studies, coupled with the consistency of their findings, also in regard to the cross-sectional studies, add *substantial* weight to the evidence for developmental neurotoxicity from fluoride exposure.

88. I understand that Dr. Hu and Dr. Lanphear will be discussing the ELEMENT and MIREC cohort studies in detail, so I will forego doing so here. As I explained in my initial expert report, these are high-quality studies given their prospective birth cohort design, individual measurements of fluoride exposure, and extensive control for potential confounders.

89. In addition to the ELEMENT and MIREC studies, a prospective birth cohort study has also been published from a separate area of Mexico where there are elevated levels of fluoride in drinking water (Valdez Jiminez 2017). In this study, maternal urine-fluoride (corrected for specific gravity) was examined for its association with scores on the Bayley Scales among 65 children evaluated at age 3-15 months. The mothers in the study had average urine-fluoride concentrations at each of the three trimesters of pregnancy of 1.9, 2.0, and 2.7 mg/L. These fluoride exposure indicators during the first and second trimesters were associated with large and significant reductions in the Bayley Mental Development Index (MDI) (cognitive) score after adjusting for covariates, including gestational age.

While this study is not as robust as the ELEMENT and MIREC studies due to the limited size, its findings are consistent with and reinforce their findings, and add further weight to the neurotoxicity assessment given its prospective cohort design.

B. Prospective Cohort Studies without Prenatal Exposure Assessment

90. Two additional prospective studies have been previously published on fluoride and neurodevelopment (Shannon et al. 1986; Broadbent et al. 2015), both from New Zealand. They have substantial limitations that make them much less informative than the North American studies, including a failure to obtain individual measurements of fluoride exposure, and a failure to ascertain prenatal fluoride exposure.

91. The first of the New Zealand studies was published in 1986 by Shannon. It found no association between childhood behavior (as scored by mothers and teachers) and the duration of time the child had lived in a fluoridated area during the first 7 years of life. The authors, however, made no attempt to ascertain prenatal and early postnatal exposures. Postnatal exposures were measured by simply tallying the number of years a child resided in a fluoridated area, with no distinctions made for the *timing* of postnatal exposure. Under this exposure metric, a child who lived her first year of life in a fluoridated area (a period of increased vulnerability) would be treated the same as a child who lived her seventh year of life in a fluoridated area.

92. A second prospective study from New Zealand was based on a birth cohort established from births in 1972-1973 (Broadbent et al. 2015). The 1,037 children were recruited at age 3 years, and IQ tests were administered at ages 7, 9, 11 and 13 years, and again at age 38. Urine samples were again not available for analysis, and the authors had no individual data on water intake. Instead, the authors compared individuals who had lived for an undefined period of time in a fluoridated area during their first five years of life, with individuals who had not lived in a fluoridated area during their first five

years. No significant differences in IQ were noted using this exposure metric, and this finding was independent of potential confounding variables, including sex, socioeconomic status, breastfeeding, and birth weight.[9]

93. The Broadbent study also made no attempt to ascertain prenatal exposures, including maternal tea consumption, which is an important limitation given the high rate of tea consumption in New Zealand. Tea contains elevated levels of fluoride, and tea consumption can be a major source of fluoride intake among adults (Waugh 2017). During the time that the children in this study were born (1972-1973), New Zealanders consumed as much as 2.6 kg of tea per capita per year (corresponding to 3-4 teabags per day), as compared to the consumption of 0.5 kg in Canada in the approximate time the MIREC cohort was recruited (Grigg 2002). The failure of both New Zealand studies to consider maternal tea consumption may have introduced substantial imprecision into the exposure classification.

94. An additional concern is that the 10% of cohort subjects who had not lived in fluoridated areas very likely received fluoride supplements, which would eliminate much of the (postnatal) difference in exposure between the fluoridated and non-fluoridated areas. In a letter published subsequent to the study, the authors estimated that the average difference in exposure between children in fluoridated vs. non-fluoridated areas was only 0.3 mg/day (Broadbent et al. 2016).

95. Based on the absence of individual measurements of exposure; failure to control for the timing of exposure, including prenatal exposures; and the relatively small difference in postnatal exposures in the Broadbent study, the New Zealand studies provide virtually no information about the neurotoxic impact of early-life fluoride exposures. They carry little weight in my assessment.

VII. SYSTEMATIC REVIEW

96. Although I decided not to conduct a formal systematic review for my weight-of-the-

[9] Despite the fact that lead exposure in this cohort was later reported to cause IQ deficits (Reuben et al. 2017), the authors of the fluoride study chose not to control for exposure to lead or other chemicals that can affect neurodevelopment.

evidence analysis, I had the opportunity to consider and analyze the review conducted by Dr. Ellen Chang of Exponent. As I described in my expert rebuttal report, Dr. Chang's systematic review provides no credible grounds for questioning my assessment of the literature; in fact, it further supports it.

A. Dr. Chang's Systematic Review Confirms that I Considered All Significant Data

97. Dr. Chang stated that her systematic review identified numerous studies that I did not address, with the apparent implication that these studies are somehow at odds with my opinion (p. 8). What Dr. Chang failed to reveal, however, is that the great majority of these studies reported significant associations between fluoride exposure and neurotoxic outcomes, further confirming my own assessment.

98. Of the 31 studies that Dr. Chang has identified and which I did not specifically address, 27 found associations of elevated fluoride exposure with adverse effects.[10] These studies, which provide further *support* for my opinions, were not cited in my report because most are repetitions of the cross-sectional study design in endemic fluorosis areas that I have already discussed at length; some are only available in abstract form;[11] some are secondary analyses of primary studies that I already addressed;[12] and one was not available to me at the time of submitting my report (Till et al. 2020). As explained in my report, I do not consider it necessary to address and discuss each and every paper that reports on fluoride effects, especially when peer-reviewed systematic reviews are available, including our own (Choi et al. 2012). I consider it more informative to examine the various *types* of studies, including toxicokinetics (e.g., distribution of fluoride throughout the body, including transfer through the placenta and blood-brain barrier); toxicological findings from animals; and different endpoints relevant to

[10] Aravind (2016); Asawa (2014), Calderon (2000), Das (2016), Khan (2015), Kundu (2015), Liu (2000); Lu (2019), Manju (2017), Mustafa (2018), Nagarajappa (2013); Qin (1990), Razdan (2017), Rocha-Amador (2007), Rocha-Amador (2008), Rocha-Amador (2009), Saxena (2012), Shivaprakash (2011), Singh (2013), Sudhir (2009), Thomas (2018), Till (2019), Trivedi (2007), Wang (2005), Xiang (2015), and Yu (2018).

[11] Calderon (2000); Thomas (2018).

[12] Xiang (2015); Wang (2012).

neurotoxicity (e.g., cognitive tests, thyroid function, histological assessments of fetal brain).

99. Conversely, many of the studies that I addressed in my report[13] were not considered by Dr. Chang for unexplained or spurious reasons. Dr. Chang's review, for example, never addressed or considered fluoride's (i) passage through the placenta, (ii) uptake into fetal brain, and (iii) neurochemical and anatomical effects, and she spuriously dismisses the evidence of neurotoxicity in adults as irrelevant to developmental effects in humans (p. 31). In several important ways, therefore, Dr. Chang's review is not as systematic as my own.

100. Dr. Chang's systematic search of the literature identified four papers that reported no significant associations with neurodevelopmental effects and that I did not rely on, but upon inspection, they have no material effect on the conclusions that can be drawn, as I will now discuss.

101. One study highlighted by Dr. Chang is a publication by Spittle and colleagues (Spittle 1998) that Dr. Chang refers to repeatedly throughout her review. Although noted in a lengthy table at the end, Dr. Chang fails to acknowledge in the body of her review that this report is in the form of an abstract and relates to a previous (full) publication (Shannon et al. 1986) that I addressed in my report (and above). I did not cite the Spittle abstract in my report, just as I did not cite abstracts of studies reporting harm.[14] It is standard practice for systematic reviews to omit abstracts, as practiced in systematic reviews conducted by the authoritative Cochrane group (Iheozor-Ejiofor et al. 2015). Dr. Chang provides no justification for including abstracts in her review, such as the one by Spittle (Spittle 1998). Dr. Chang's prominent references to the Spittle abstract is particularly surprising given that it does not describe *any* confounder adjustment,[15] and uses an ecological metric for exposure (group water

[13] *E.g.*, Dong (1993); Duan (1995); Ekstrand (1981); Li (2016); Spittle (1994); Guo (2001); Malin (2018); Opydo-Szymaczek (2005, 2007); Peckham (2015); Ron (1986); Salgarello (2016); Shao (2003); Shen & Taves (1974); Yazdi (2011); Yu (2008).

[14] Calderon (2000); Thomas (2018).

[15] On p. 132 of her Table, Dr. Chang "assume[s]" that the Spittle analysis controlled for the same confounders as the Shannon analysis. I understand that neither Dr. Chang, nor anyone else in her

F level) – features which Dr. Chang has used to dismiss many papers that support the neurotoxicity of fluoride.

102. The other three "no-effect" studies that Dr. Chang cites and that I did not address are similarly unavailing. Two are cross-sectional studies from China which fail to show statistically significant associations between fluoride exposure and IQ,[16] and one is an ecological analysis (Perrott 2018) of the Malin & Till (2015) study on ADHD which I addressed but placed little weight on. As I explained in my report, there are many reasons why an ecological/cross-sectional study can fail to detect an effect even when one is present. The failure of these three studies to find statistically significant effects does nothing to contradict the robust literature that I rely upon, including the prospective birth cohort studies that I placed the greatest weight on. Even Dr. Chang appears to recognize this, as she does not include any of these three studies in her causal analysis, and correctly notes that the analysis by Perrott (Perrott 2018) is a "relatively low quality" ecological study (p. 66).

103. In summary, despite asserting that my review failed to consider "numerous" papers, Dr. Chang's own review confirms that I addressed and considered the most relevant epidemiological studies on cognitive outcomes. Dr. Chang's literature search also confirms that the majority of studies that I did not specifically address are consistent with and further support the association between fluoride and cognitive impairment, in accordance with my conclusions.

B. Dr. Chang's Review Fails to Identify Any Systematic Biases that Explain Fluoride's Consistent Association with Neurodevelopmental Harm

104. Dr. Chang's systematic assessment of study quality provides a lengthy discussion of real or perceived methodological limitations in the available studies. Importantly, however, Dr. Chang failed to identify a likely explanation for how these limitations can explain the consistent adverse associations

office has contacted Dr. Spittle to confirm this statement (Personal email communication with Bruce Spittle, August 13, 2019). According to Dr. Spittle, the abstract provided all important methodological details.

16 He (2010); Kang (2011).

between fluoride and IQ across both cross-sectional and prospective studies. For example, Dr. Chang referred to "high potential for selection bias" but did not consider how unlikely it is that dozens of studies should all suffer from some particular exposure misclassification or selectivity that would all cause bias *away* from the null, e.g., selection bias that would result in participation of intellectually disabled children only in the high-fluoride group, or residual confounding resulting in bias only away from the null in the many different study settings.

105. Dr. Chang claimed that "methodological uncertainties remain about the assessment of fluoride exposure and neurodevelopmental outcomes; and the reported findings are plausibly explained by confounding, bias, and chance" (p. 9). However, she did not provide any convincing evidence that such issues could have resulted in erroneous conclusions, especially in the high-quality prospective studies.

106. Throughout her analysis, Dr. Chang failed to grapple with the fact that random (i.e., non-differential) error is unlikely to cause a bias away from the null, as is well-known in epidemiology, as I have also discussed in past publications (Grandjean and Budtz-Jorgensen 2007, 2010). Dr. Chang thus did not articulate a plausible basis for why the limitations she claimed to have identified can *systematically* bias the results across the many study settings, including the North American birth cohorts.

107. Dr. Chang described cross-sectional studies as if they are all equal and as if the exposure parameter always represents a current and short-lasting exposure only. In so doing, Dr. Chang failed to acknowledge in her causal analysis that exposure measures in many studies represent long-term community conditions, in some studies also likely covering prenatal exposures, a critical detail.

108. Dr. Chang referred to exaggerated associations that can result from lack of blinding (p. 59), but failed to acknowledge that at least 11 of the studies reporting adverse neurocognitive effects

have clearly been blinded, including the recent birth cohort studies, where the exposure was determined *after* the cognitive tests had been completed. Thus, while lack of blinding can create observation bias, it cannot explain the inverse association between fluoride and IQ because similar associations have been consistently found in studies known to be blinded. Despite producing a 56-page table to address "key characteristics" of the studies, Dr. Chang failed to mention this methodological strength in her summary of the studies (pp. 90-146).

109. Dr. Chang repeatedly highlighted the risk of publication bias, e.g., in the biomedical journal *Fluoride*, which is not indexed by PubMed. However, she does not mention the bias *against* publication, i.e., a bias that acts in the opposite direction. The examples that I mentioned in my report illustrate that such bias exists.[17] Further, Dr. Chang speculated that Chinese-language studies that did not find adverse effects may not have been translated into English (p. 37). Instead of speculating about this, Dr. Chang's systematic review could have included a search of online databases of Chinese-language research (e.g., CNKI) but, for unexplained reasons, did not do so.[18]

110. In summary, although Dr. Chang's systematic assessment of study quality correctly identified limitations in a number of studies, she failed to credibly explain how these limitations can plausibly explain the significant inverse associations that have consistently been found across many study settings and designs.[19] Although I recognize the issues that Dr. Chang has raised and have fully

[17] The desire to use fluoride in caries prevention programs has sometimes made it difficult for researchers, including myself, to present findings of potential toxic effects. In addition to my own personal experiences, published case reports suggest that some studies reporting adverse results have been suppressed, and, in at least one instance, a respected scientist at the Forsythe Dental Institute lost her job after publishing evidence of neurotoxicity.

[18] A search of PubMed for "CNKI database" shows that many systematic reviews include CNKI as one of the databases to retrieve studies, and the Institute of Medicine recommendations for systematic review (which Chang relies on) calls for searching for foreign language studies when appropriate (IOM 2011, p. 8). CNKI is publicly available online at: http://oversea.cnki.net/kns55/default.aspx.

[19] Unable to explain why so many studies have found significant associations between fluoride and IQ, Dr. Chang claims that "most published scientific research findings are anticipated to be false" (p. 38, citing Ioannidis (2005)). Although the original report by John Ioannidis (Ioannidis 2005)

considered them in my assessment, it remains extremely unlikely, if not impossible, that the overwhelming evidence of fluoride neurotoxicity is a mirage caused by bias, as Dr. Chang apparently believes. A far more likely and plausible explanation for the consistent findings in the epidemiological studies is that fluoride is a developmental neurotoxicant that reduces IQ and that this association is strong enough to be apparent also in studies with less-than-ideal designs.

C. Bradford Hill Aspects Support, Rather than Detract from, the Causal Nature of Fluoride's Association with Neurodevelopmental Harm

111. Dr. Chang used the Bradford Hill aspects to evaluate the causal relationship between fluoride and neurotoxicity. As I explained in my rebuttal report, her causal analysis is superficial and pays lip service only to Sir Austin's wise advice. An appropriate and systematic assessment of the Bradford Hill guidelines supports, rather than refutes, the causal relationship between elevated fluoride exposure and IQ loss. I will summarize here:

112. *Strength*: Dr. Chang dismissed the strength of the association between fluoride and IQ on the grounds that a loss of 3 to 5 IQ points is relatively small in comparison with normal, expected variation (p. 69). Under this arbitrarily high standard, other well-known neurotoxicants (e.g., lead, methylmercury, arsenic) would fail Dr. Chang's strength criterion. By failing to consider the strength of association of other well-known neurotoxicants, Dr. Chang subjectively analyzed the data on fluoride in a meaningless vacuum. Had Dr. Chang considered the strength of association for other neurotoxicants, she would have found that the effect size for fluoride actually is actually large, not small (i.e., it rivals the effects of lead), which *supports*, rather than detracts, from a causal relationship.

113. *Consistency*: One of the most compelling aspects about the epidemiological research on

did provide some stunning examples of how clinical medicine could be misled by single reports, it would be reckless and counterproductive if we were to ignore all published reports, as Dr. Chang seems to prefer. This nihilistic view was also not the intent of the author. In a more recent paper in the same journal, Dr. Ioannidis highlighted the need for balanced review of scientific evidence in the interest of inspiring responsible policy decisions (Ioannidis 2018).

fluoride is how consistent it has been in finding significant associations with IQ (Choi et al. 2012). Dr. Chang obscured this by highlighting non-informative studies that made no attempt to measure or investigate prenatal or early postnatal fluoride exposures (Barberio et al. 2017; Broadbent et al. 2015; Morgan et al. 1998; Shannon et al. 1986; Spittle 1998) as being on the same level as, and contradicting, the highly significant findings from the prospective ELEMENT (Bashash et al. 2017) and MIREC (Green et al. 2019) prospective birth cohort studies (pp. 70-71). A particularly poor judgment by Dr. Chang was to place the Spittle abstract on the same level as the ELEMENT and MIREC studies despite the fact that Spittle's abstract does not describe *any* confounder adjustment. Dr. Chang cited the "mixed" nature of the findings as a basis to conclude that the consistency factor has not been met, while failing to acknowledge the inappropriate apples-to-oranges nature of comparing the prospective ELEMENT/MIREC studies to much, much weaker studies.

114. In her assessment of consistency, Dr. Chang failed to mention the fact that every single prospective birth cohort study with prenatal exposure measurements has found a significant adverse effect of prenatal fluoride on neurodevelopment (Bashash et al. 2017; Bashash et al. 2018; Green et al. 2019; Valdez Jimenez et al. 2017). Dr. Chang also gave short shrift to the consistent association between fluoride exposure and reduced IQ reported in the numerous cross-sectional studies (Choi et al. 2012; Duan et al. 2018; Tang et al. 2008). This latter shortcoming may be a result of Dr. Chang's critical misunderstanding of our meta-analysis (Choi et al. 2012), which I will now address.

115. Dr. Chang claimed that our meta-analysis found an average loss of 0.45 IQ points in the high-fluoride areas and characterizes this as a 10-fold difference with the Tang meta-analysis (Tang et al. 2008). This, however, is not what we reported (see paragraph 78). Because different intelligence scales had been used in the studies considered, we expressed the outcome as a random-effect standardized weighted mean difference estimate, as we clearly explained (Choi et al. 2012). In order to

translate this measure to a difference on the same IQ scale, the joint result must be multiplied by the standard deviation of the IQ scale, i.e., 15. An SMD of -0.45 thus corresponds to a loss of 6.75 IQ points. Contrary to Dr. Chang's mischaracterization, therefore, the results of our meta-analysis are consistent with the Tang meta-analysis, a fact that we actually mention in our published paper (Choi et al. 2012).

116. Dr. Chang's assessment of consistency also entirely ignored the findings from occupational studies, as well as the neuropathology data from examinations of fetal brains in endemic fluorosis areas. As I explained in my report, each of these types of studies is consistent with, and provide support for, fluoride being a neurotoxic agent.

117. *Specificity*: As Kenneth Rothman (2012) and others (Neutra 2018) have emphasized, and as Dr. Chang recognized, lack of specificity between an exposure and an outcome (e.g., asbestos and mesothelioma) does not weigh against or in favor of a causal conclusion.

118. *Temporality*: Dr. Chang's assessment of temporality mirrored her assessment of consistency in that she cited the two New Zealand studies as contradicting the findings from the ELEMENT and MIREC cohorts. Once again, Dr. Chang fails to acknowledge the absence of prenatal or early postnatal fluoride exposure assessments in the New Zealand studies, nor any of the other serious shortcomings of these studies. Instead, Dr. Chang focused on non-differential measurement uncertainties of the urine-fluoride data in the far superior ELEMENT and MIREC cohorts to cast doubt on the findings of these studies. As already discussed in my report, however, the imprecision of the fluoride exposure parameters would likely *bias the results toward the null*, not the reverse. The temporality requirement is thus met with fluoride, as each of the prospective birth cohort studies has found a significant association between early-life exposure to fluoride and the offspring's subsequent performance on neurobehavioral testing. The exposure preceded the effect in these studies, which is

what the temporality factor is supposed to assess.

119. *Biological Gradient*: The ELEMENT and MIREC studies have reported monotonic dose-response relationships between elevated prenatal fluoride exposure and IQ deficits in the offspring (Bashash et al. 2017, Green et al. 2019), as well as ADHD behaviors (Bashash et al. 2018). Dr. Chang dismissed the biological gradient of these effects by showing scatterplots from the ELEMENT and MIREC cohorts without the trend lines. However, this approach proves little insight, other than illustrating the undisputed fact that there is substantial natural variation in IQ across the population and that an appropriate statistical analysis is needed to extract a reliable estimate of the average effect of the toxicant exposure. Similar scatterplots have been published showing the effects of lead and IQ, as can be seen in the figure to the right (Rothenberg & Rothenberg 2005, Figure 1). Lead would thus fail the biological gradient test that Dr. Chang has used for fluoride. Dr. Chang also argued that outliers may have distorted the effects seen in the ELEMENT and MIREC cohorts (p. 77-78), without acknowledging that statistical analyses on the impact of outliers have been conducted and that the results did not meaningfully change (Bashash et al. 2017).

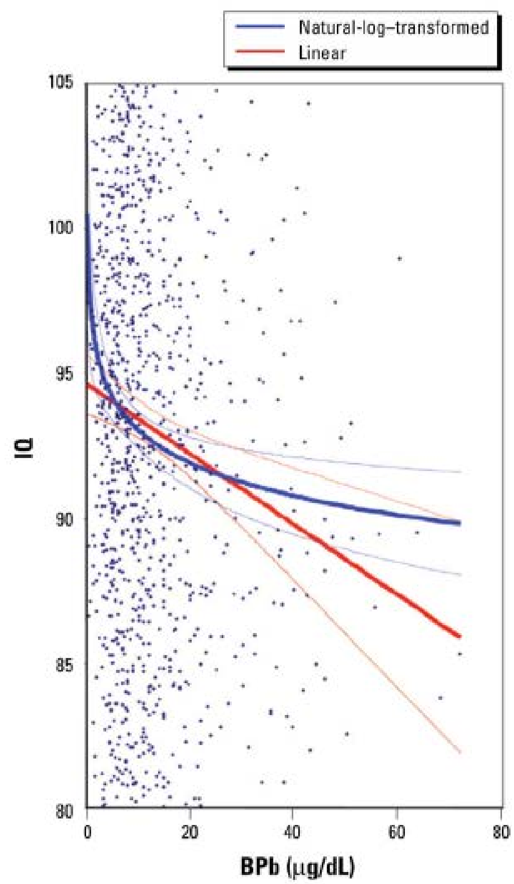


Figure 1. Child blood-lead and IQ. From Rothenberg & Rothenberg 2005.

120. *Plausibility*: Dr. Chang limited her assessment of biologic plausibility to NTP's assessment of learning and memory (NTP 2016) in animal models, and to Dr. Tsuji's expert report. In so doing, Dr.

Chang completely ignored the large body of animal literature showing adverse neuroanatomical and neurochemical effects from fluoride exposure, as already reviewed by the National Research Council (NRC 2006) and by Dr. Thiessen in her report. The NRC concluded that the neuroanatomical and neurochemical effects are sufficient to determine that fluoride interferes with brain function (NRC 2006). Dr. Chang ignored this information in favor of the NTP's more narrow assessment on learning/memory, but even the NTP assessment found suggestive evidence that fluoride impairs learning and memory. In contrast to Dr. Chang's assessment, EPA's own experts on developmental neurotoxicity, including internationally recognized scientists such as William R. Mundy and Kevin M. Crofton (Mundy et al. 2015), have identified fluoride as a chemical with substantial evidence of developmental neurotoxicity.

121. *Coherence*: Dr. Chang dismissed the coherence of fluoridated water reducing IQ on the grounds that IQ scores in US children steadily improved throughout the 20th century (the so-called "Flynn Effect"). Dr. Chang even went so far as to suggest that fluoridation may be responsible for the increased scores, although more plausible explanations are known. Under Dr. Chang's simplistic framework, leaded gasoline could not have reduced IQ and may have increased it, as it was introduced in the early part of the 20th century and IQ scores continued to increase during the entire duration of its use. It is well accepted, however, that low-level lead exposure reduces IQ, and thus the Flynn Effect argument—while perhaps superficially appealing—does *not* demonstrate "incoherence."

122. In her assessment of coherence, Dr. Chang failed to consider other relevant considerations, including the association between neonatal fluoride exposure mediated by infant formula feeding and reduced IQ (Till et al. 2020), as further discussed below. While the studies prior to the recent Canadian analysis did not evaluate the potential role of neonatal fluoride exposure, formula feeding is well established to increase a baby's fluoride exposure, even in areas without fluoridated water (Harriehausen

et al. 2019; Zohoori et al. 2019). Although other factors are of likely importance, the relationship between formula-feeding and reduced IQ is coherent with maternal fluoride exposure during pregnancy being associated with a lowered IQ in the child and supports a causative relationship between early-life exposure to fluoride and IQ deficits.

123. *Experiment*: Dr. Chang ignored the NRC's observation (NRC 2006) that case reports of fluoride toxicity constitute "experimental studies" of neurologic symptomatology following fluoride exposure (NRC, p. 208). The case reports involve "one or more individuals who underwent withdrawal from their source of fluoride exposure and subsequent re-exposures under 'blind' conditions." In most cases, the symptoms (which included lethargy, weakness, and impaired ability to concentrate) "disappeared with the elimination of exposure to fluoride and returned when exposure was reinstated." Although experimental support is not an obligatory criterion (Neutra 2018), the existence of such support should not be missed in what is dubbed a systematic assessment.

124. *Analogy*: I agree with Dr. Chang that "analogies can be drawn to other naturally occurring elements, especially certain metals" like lead (p. 85). As discussed above, many of the exaggerated criteria that she uses to reject a causal relationship between fluoride exposure and IQ could be equally used to erroneously dismiss the causal relationship between low-level lead exposure and IQ.

125. In summary, after correcting for Dr. Chang's errors and biases in judgment, the Bradford-Hill aspects support, rather than detract from, a causal relationship between fluoride in water and neurotoxicity. After analyzing and considering Dr. Chang's systematic review, I have more, not less, confidence that developmental neurotoxicity is a serious risk of elevated fluoride exposure.

VIII. BENCHMARK DOSE (BMD) ANALYSIS

A. Selection of Source Data

126. Regulatory agencies are in overall agreement in using Benchmark Dose (BMD) analyses to

calculate non-cancer health-based limits for dietary intakes of contaminants, such as those found in drinking water (EFSA 2009; EPA 2012).

127. As with the Faroe Islands cohort that the EPA relied upon in its risk assessment for methylmercury, the ELEMENT and MIREC studies are high-quality birth cohorts suitable for dose-response analysis (Bashash et al. 2017; Green et al. 2019). Further, as the data refer to the critical effect in a highly vulnerable population, they constitute appropriate data to use for identifying a safe exposure limit for fluoride. I worked, therefore, with my colleague, Dr. Budtz-Jorgensen, on BMD analyses of these studies, which I describe below.

128. Our selection of the ELEMENT and MIREC studies for BMD analysis is consistent with an analogous assessment conducted by both Dr. Chang and her colleague, Dr. Joyce Tsuji (Tsuji et al. 2015) for another neurotoxicant. In their paper, Drs. Chang and Tsuji sought to determine if the existing RfD for arsenic is adequately protective of neurotoxicity. To answer this question, they conducted a systematic review of the literature to see if there were any studies that would permit a dose-response analysis for quantitative risk assessment. After reviewing the literature, they found a study that, in their judgment, was suitable for the purpose: a study from Bangladesh by Hamadani et al. (2011).

129. The ELEMENT and MIREC studies are at least equally suitable for dose-response analysis as the one study Dr. Chang and Dr. Tsuji found sufficiently reliable to use for their risk assessment of arsenic exposure. As with the Hamadani study, the ELEMENT and MIREC studies have a (i) prospective birth cohort design; (ii) large sample size; (iii) control for potential confounders;[20] (iv) urine measurements[21] of the toxicant of interest during pregnancy; (v) and extended follow-up (up to 5 years

[20] Drs. Chang and Tsuji considered studies to have sufficiently controlled for potential confounders if they controlled for SES or HOME Score and parental education/IQ (Tsuji et al. 2015, p. 93).

[21] As with the ELEMENT and MIREC studies, the Bangladesh study measured prenatal exposure through several samples of maternal urine (adjusted for specific gravity) (Hamadani 2011). The Bangladesh study collected urine twice during the pregnancy (at gestational weeks 8 and 30), which

after birth). In fact, the ELEMENT and MIREC studies have an important advantage: the average arsenic exposure in Bangladesh substantially exceeded exposures in the U.S.,[22] which is not the case with the North American fluoride cohort studies.

130. My calculations of benchmark values for fluoride from the ELEMENT and MIREC cohorts are therefore in accordance with the criteria that Drs. Chang and Tsuji have previously used when generating benchmark calculations for arsenic (where adverse effects were seen in girls, but not boys, at 5 years of age).

B. Selection of Benchmark Response (BMR)

131. The benchmark dose (BMD) is defined as the dose that leads to a specific loss (or degree of abnormality) known as the benchmark response (BMR) in the outcome variable. The BMR must be defined before the analysis (EPA 2012), and general guidelines been developed for the selection of a BMR (EFSA 2009).

132. According to the EPA Clean Air Scientific Advisory Committee, a 1-to-2 IQ point reduction at the population level is "highly significant from a public health standpoint," and should be prevented in up to 99.5% of the population (EPA 2008). Consistent with this, previous BMD analyses of human neurotoxicity have selected 1 IQ point as the BMR (Budtz-Jorgensen et al. 2000; Budtz-Jorgensen et al. 2013; EFSA 2010; Tsuji et al. 2015).

133. Economists have calculated the substantial losses in lifetime incomes from a decrease of 1 IQ point[23] (Gould 2009), as also practiced by economists at the EPA in regulatory impact analyses (EPA 2008).

134. Research on other neurotoxicants (Grandjean 2013) has shown that shifts to the left of IQ

is a lower number of samples than the MIREC cohort, and roughly the same as the ELEMENT study.

[22] The Bangladesh study addressed a population with mean urinary arsenic levels ranging from 35 to 80 ug/L, which is about 10-to-40 times the levels measured in the US population.

[23] In terms of 2006-dollars, the value of 1 IQ point was calculated to be about $18,000 (Gould 2009; Spadaro and Rabl 2008).

distributions in a population (i.e., reductions in average IQ) can have substantial impacts, especially among those in the high and low ranges of the distribution (Bellinger 2007).

135. Consistent with prior analyses, including our own, we therefore selected 1 IQ point as the BMR (Budtz-Jorgensen et al. 2000; Budtz-Jorgensen et al. 2013).

C. Analyses of ELEMENT and MIREC Data

136. For our BMD analysis, we used the same formula that we used in our prior assessment of lead (Budtz-Jorgensen et al. 2013). The formula is as follows:

> The BMD is defined by
>
> $$f(0) - f(BMD) = BMR \;\rightarrow\; BMD = f^{-1}(-BMR)$$
>
> In a linear model, $(Y = \alpha + \beta d + \varepsilon)$, from which we get $BMD = -BMR/\beta$.
>
> Likewise, the BMDL is defined as a lower one-sided 95% confidence limit of the BMD. In the linear model,
>
> $$BMDL = -BMR/\beta_{lower}$$
>
> where β_{lower} is the one-sided lower 95% confidence limit for β. Information on the (linear) regression coefficients and their standard deviations, from which the confidence intervals can be calculated, is available from the published articles on the two major prospective cohort studies.

137. For the ELEMENT study (Bashash et al. 2017), a linear dose-response model could be used for the effect of urine-fluoride concentrations on both measures of childhood IQ (i.e., the General Cognitive Index (GCI) results at age 4 and IQ results at ages 6-12). In this model, the BMD and BMDL can be calculated based only on the regression coefficient and its precision. In Table 4 of the publication (Bashash et al. 2017), this information is available both for a crude model and for a model A with confounder adjustment. The table below shows the benchmark results for these two models for both the age-4 GCI and school-age IQ.

***Table 2. Benchmark dose results (mg/L urine adjusted for creatinine) obtained from the ELEMENT study results* (Bashash et al. 2017).**

Model	GCI		IQ	
	BMD	BMDL	BMD	BMDL
Crude	0.133	0.085	0.211	0.121
Adjusted	0.159	0.099	0.200	0.130
Read from plot	0.159	0.102	-	-

138. As these calculations are based on assumptions of Gaussian distributions, we checked the validity by scanning the numbers from the plot in the published article. We tentatively used the *WebPlotDigitizer* software to read the plot shown in the published paper (see Figure 1, page 22) (Bashash et al. 2017), to obtain the individual adjusted GCI results for more accurate BMD calculations. Of the original 287 observations, the software provided 286 observations, probably due to two overlapping observations. Thus, missing a single point only, our calculations based on the scanned data should be considered fairly reliable.

139. Using the standard benchmark approach to epidemiological data and a linear dependency, we find that the BMD for GCI is approximately 0.16 mg/L, and that the BMDL is 0.10 mg/L (bottom line of Table 2). These results are in excellent agreement with the results calculated only from the regression data presented in Table 4 of Bashash et al. (2017).

140. To assess the robustness of the calculation, we included a logarithmic conversion of the exposure parameter. We also used a split linear dose-response curve as in one of our previous studies (Budtz-Jorgensen et al., 2013). These sensitivity analyses showed BMD results that deviated only marginally from the calculation using the default linear association. In conclusion, Table 2 shows

reliable BMD results that have been calculated in accordance with standard EPA procedures.

141. We also conducted a BMD analysis of the MIREC data. As with the ELEMENT study, we calculated the BMD and BMDL from the reported regression coefficients and standard deviations, with the assumption of a Gaussian distribution (Green 2019, Table 2). In addition to calculating the BMD and BMDL from the urine-fluoride data (U-F), we also calculated a BMD and BMDL from the maternal fluoride intake data. Our results are shown in the table below:

Table 3. Benchmark dose results (mg/L urine adjusted for specific gravity, or mg estimated daily intake) obtained from MIREC study results on IQ (Green et al. 2019).

Study	Exposure	Sex	BMD	BMDL
MIREC	Maternal U-F	Both sexes	0.51	0.21
	Maternal U-F	Boys	0.22	0.13
	Maternal U-F	Girls	(–)	0.58
	Maternal F intake	Both sexes	0.27	0.15

142. As shown in the above table, the prenatal BMD for girls is not defined when relying on urine-fluoride, but the BMDL is still meaningful and is, as expected, higher than the other estimates obtained. No sex difference was found when relying on estimated fluoride intake.

143. Overall, the results derived from the two studies are comparable. In the ELEMENT study, the BMDL for maternal urine among ~4-year olds is approximately **0.1 mg/L** (both sexes), while in the MIREC study, it is **0.13 mg/L** (boys) and **0.21 mg/L** (both sexes).[24] The respective BMDL for the 6-to-12-year olds from the ELEMENT study is **0.13 mg/L**, thus overall approximately 0.15 mg/L. Consistent with these maternal urinary excretion values, the BMDL for maternal fluoride *intake* in the MIREC

[24] Based on how the authors reported the data, our BMDL values from the ELEMENT cohort are creatinine-adjusted, while our BMDL values from the MIREC cohort are specific gravity-adjusted.

study is **0.15 mg/day** (both sexes).

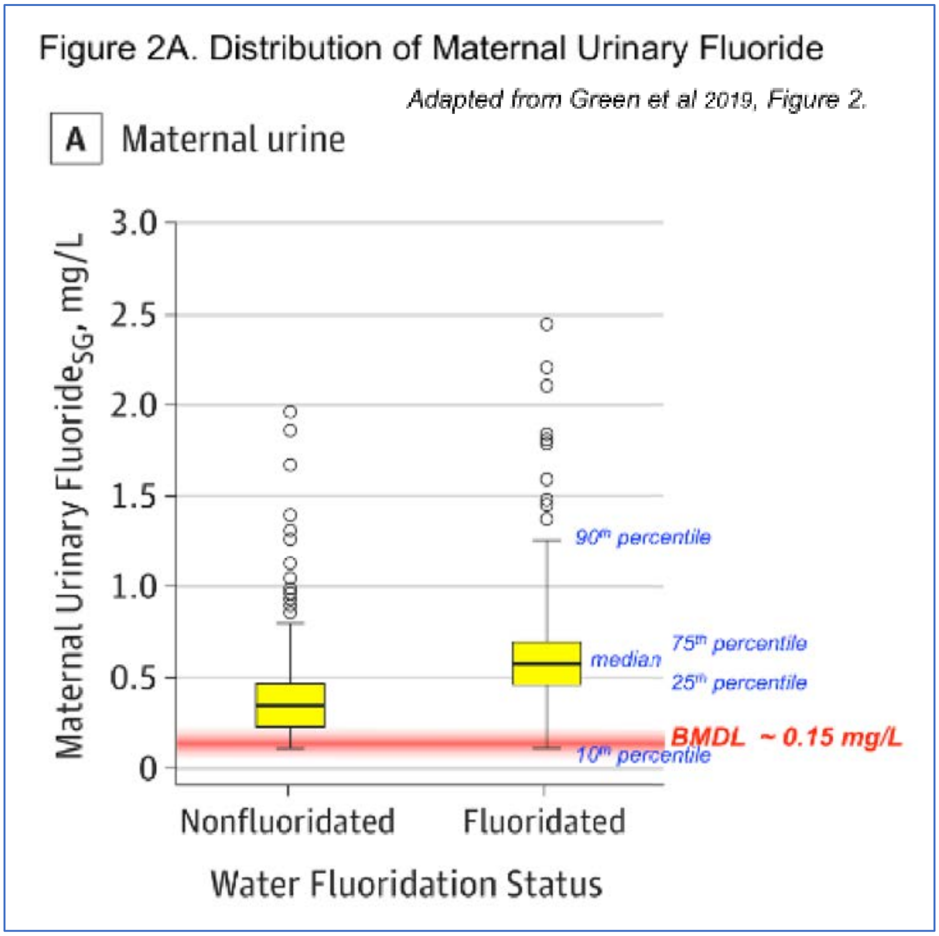
IX. ASSESSMENT OF RISK

A. Comparing BMDLs with Current Exposures in Fluoridated Areas

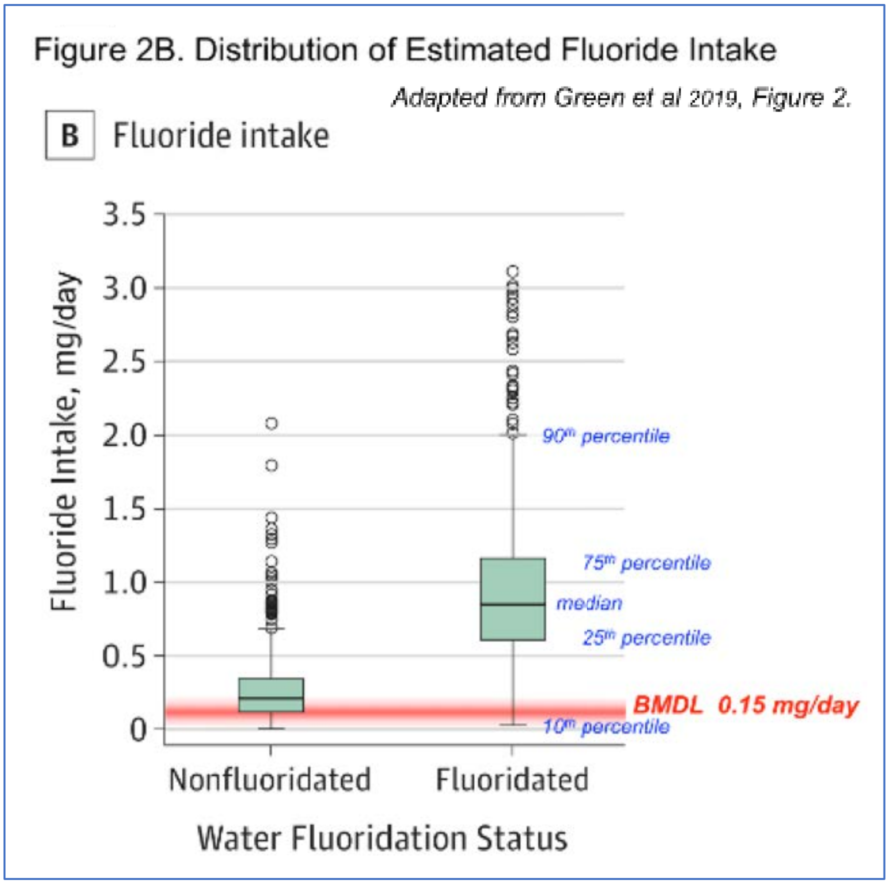
144. As benchmark dose calculations constitute a routine approach applied by the EPA for establishing safe limits on chemical exposure, the above calculations that rely on overall associations between fluoride exposure and cognitive deficits provide a basis for assessing the risk of cognitive deficits from current fluoride exposure levels.

145. Typically, the EPA uses the BMDL to calculate a Reference Dose (RfD) by dividing by an uncertainty factor for the purpose of accounting for variations in human susceptibility. The default value that EPA uses for the uncertainty factor is 10. Here, if we round up the overall BMDL to **0.2 mg/L**, or about 0.2 mg/day, the RfD would likely be 0.02, which is very much below current exposure levels, especially in communities with fluoridation programs (Till et al. 2018). But, *even if no uncertainty factor is applied, and even if relying on the BMD rather than the BMDL* (both of which would be unusual), the RfD would still be well below current exposure levels in fluoridated areas (Till et al. 2018).

146. The serious risk that we are confronted with can be appreciated by visually comparing the BMDLs against documented exposure levels in fluoridated communities. The following Figure 2A, adapted from Green et al. (2019), compares an overall BMDL for maternal urine-fluoride (0.15 mg/L) with the maternal urine-fluoride concentrations reported in the study. As can be seen, the urine-fluoride levels far surpass the levels associated with IQ loss.



147. The below Figure 2B compares the approximate BMDL for maternal fluoride *intake* from beverages (0.15 mg/day) against the reported fluoride intakes in Green et al (2019). As can be seen, the estimated fluoride intakes in fluoridated areas far surpass the fluoride intake level associated with a clear IQ loss.



148. There are no contemporary large-scale studies of urine-fluoride concentrations in the United States, as the CDC has not yet reported urinary fluoride excretion levels as part of its ongoing National Health and Nutrition Examination Survey (NHANES) studies. One can reasonably infer, however, that urinary fluoride excretion levels in fluoridated areas of the U.S. are generally comparable to those in fluoridated areas of Canada. The reasonableness of this inference is supported by the following facts:

149. Canada and the U.S. add fluoride to water to reach the same target concentration (0.7 mg/L), although empirical data suggests Canadian cities only reach 0.6 mg/L which is slightly less than the U.S. (Till et al. 2018).

150. Fluoridated water is recognized as the largest source of fluoride exposure for adults, particularly when indirect sources are accounted for, such as beverages and foods prepared with the

water, including commercially prepared beverages such as soda and reconstituted juice (EPA 2010).

151. Urine-fluoride has been shown to be a good indicator of total daily fluoride intake, and has a close, linear correlation with the fluoride content in water (Villa et al. 2000; McClure 1944; Smith et al. 1950).

152. The largest study of urine-fluoride levels in the U.S. found that pooled urine samples from healthy young males generally mirrored the fluoride concentration in the drinking water (McClure 1944). Based on U.S. data, therefore, a person drinking water with 0.7 mg/L fluoride would be expected to have about 0.7 mg/L in their urine, which is similar to what was found in the MIREC cohort (Till et al. 2018). Although these U.S. data were published prior to widespread fluoridation, the levels today would, if anything, tend to be *higher* today, not lower because fluoride is now available from more sources than was the case in the 1940s (e.g., including commercial beverages made with fluoridated water, dental products, etc.).

153. To address the lack of contemporary US data on fluoride exposures in pregnant women,[25] the recent UCSF study measured the concentrations of fluoride in urine (and blood[26]) of 48 pregnant women living in fluoridated and non-fluoridated areas of California. As with the Canadian study, the UCSF team had the urine tested by Dr. Angeles Martinez-Mier at the University of Indiana and adjusted the fluoride measurements for specific gravity. The study found an average (specific gravity-adjusted) urine-fluoride concentration of **0.69 mg/L** among pregnant women in areas with at least 0.7 mg/L in the community water, which is clearly in the same range as the MIREC team found in Canada (Uyghurturk

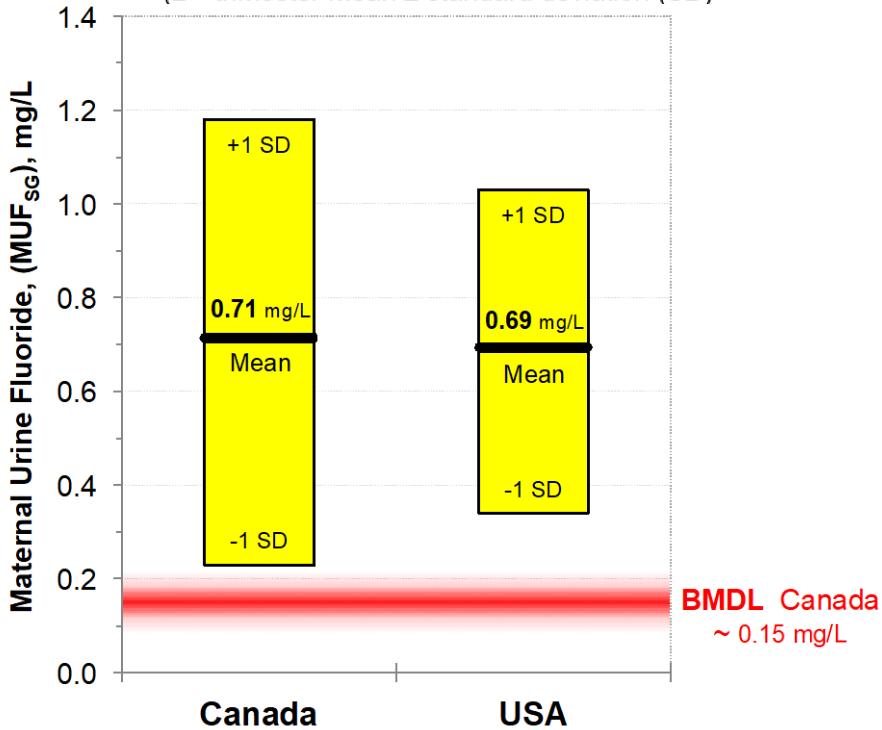
[25] As noted by the UCSF team, there is only one prior published study of urine-fluoride levels among pregnant women in the US: this is the study by Shen & Taves (1974) that I discussed in my expert report. This study found an average of 1.02 mg/L in maternal urine among a group of 16 pregnant women, but the authors did not report the concentrations of fluoride in water.

[26] The study found an average of 0.021 mg/L fluoride in the blood (=1.1 µmol/L) of the women from fluoridated areas (~0.8 mg/L), which is higher than the predicted value (0.015 mg/L = 0.8 µmol/L). As discussed by the NRC, it has been historically estimated that adult populations will have 1 µmol/L in their blood for each 1 mg/L of fluoride in the water (NRC 2006).

2020, Table 2). While the small-scale nature of the study likely introduced some random scatter in the results of the UCSF study,[27] the study supports the general similarity in fluoride exposures in the Canadian and U.S. populations.

154. Finally, the relevance of the Canadian IQ data (Green et al., 2019) to the US can be appreciated by comparing the BMDL to the maternal urine-fluoride concentrations reported by the UCSF team (Uyghurturk 2020). As can be seen in the following Figure 3, maternal urine-fluoride concentrations found in pregnant women in the Californian cohort greatly exceed the BMDL for fluoride-associated IQ loss. This, ultimately, is the most important consideration.

Figure 3. Specific gravity adjusted urine-fluoride concentrations in fluoridated communities of Canada and the U.S. (2nd trimester mean ± standard deviation (SD))



Urine F in Canada from Till 2018, Suppl., Table S4; urine F in USA from Uyghurturk 2020, Table 2.

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[27] The study found some high levels of urine-fluoride in the mid-range fluoride communities (0.3-0.5 mg/L), which slightly skewed the distribution (Uyghurturk 2020, Fig. 2). As the authors note, this may be the result, in part, of the fact that the women had their urine tested while visiting a clinic in San Francisco, which is fluoridated (Uyghurturk 2020, p. 6-7). In the areas with >0.3 mg/L in water, the average (specific-gravity adjusted) fluoride level was 0.74 mg/L (Uyghurturk 2020, Table 5).

B. Comparing Fluoride's Population-Level Effects with Other Causes of IQ Loss

155. In order to compare fluoridation's population-level effects with other neurotoxicant exposures, some approximate estimates of fluoride-associated IQ losses can be made. The calculations rely on several assumptions that are necessary in the absence of actual data and are therefore meant only to identify relative orders of magnitude. On the conservative side, I shall assume that all children are equally vulnerable and that the dose-dependent IQ losses observed in the recent prospective studies can be used to assess the impact on the population at large (i.e., that genetic and other predisposition can be ignored).

156. My analysis focuses on the average difference in maternal urine-fluoride levels between fluoridated and non-fluoridated areas. The Canadian study (Till et al. 2018) showed that this difference is approximately 0.4 mg/L. It bears emphasizing that this difference likely understates the true contribution of fluoridated water because part of the exposure in "non-fluoridated" areas comes from the "halo" effect. The halo effect refers to the fact that many commercial beverages and foods made in fluoridated areas are shipped to and consumed in non-fluoridated areas and that residents from non-fluoridated communities may work or spend time in fluoridation areas.[28]

157. Given the BMD results, an average increase of 0.4 mg/L in maternal fluoride concentrations is above the threshold for developmental neurotoxicity, even if assuming a zero background exposure. Using the dose-dependent losses observed in the recent prospective studies (Bashash et al. 2017; Green et al. 2019), this elevated exposure will correspond to an IQ loss of approximately 2 IQ points.

158. Because about two-thirds of the U.S. population receives fluoridated drinking water, one can assume that a similar proportion of the 4 million annual U.S. births (i.e., more than 2.5 million

[28] This widespread dispersal of fluoridated water in commercial products helps to explain the relatively high urine-fluoride levels now seen in non-fluoridated communities versus the situation back in the 1940s (Till et al. 2018).

births) are affected by fluoridation-associated exposure increases. With the 2-point average IQ loss associated with fluoridation, the 2.5 million births will lose a total estimated number of 5 million IQ points annually.

159. This approximate estimate can be compared with calculations made by Professor David Bellinger on IQ losses due to major pediatric diagnoses affecting 0-to-5-year-old children (Bellinger 2012). According to CDC data and Bellinger's calculations, the top pediatric etiologies for IQ loss are preterm birth at 34 million IQ points lost and lead exposure representing 23 million IQ points lost. For fluoridation, the estimate for children aged 0 to 5 years is approximately 25 million IQ points. Even if this estimate is somewhat imprecise, and unevenly distributed, the order of magnitude is likely to be correct and is very considerable.

160. Finally, even if we assume that a threshold exists at approximately 0.8 mg/L in maternal urine (as suggested by mere inspection of the IQ plots in the ELEMENT study for the 6-to-12-year-old cohort members), water fluoridation would still result in substantial IQ losses. As documented in the MIREC study, the 75th percentile maternal urine-fluoride levels (adjusted for creatinine) are 1.04 mg/L in the fluoridated areas versus 0.52 mg/L in the non-fluoridated areas (Till et al. 2018, Table S4). In fluoridated areas, pregnant women above the 75th percentile are already at least 0.29 mg/L above the hypothetical threshold, and thus 25% of the children would then experience an average IQ loss of at least 1.5 points. This would amount to over 4.5 million lost IQ points among 0-to-5-year-olds. Even this smaller amount of IQ losses exceeds the IQ losses attributed to methylmercury exposure in the U.S. (Grandjean et al. 2012).

161. I have made these calculations only to illustrate the significance and impact of neurotoxicity outcomes from fluoridation exposures, and I offer these crude estimates to emphasize my concern that developmental neurotoxicity due to early-life exposure to fluoride is a serious public health

hazard with substantial societal impacts that must be controlled.

X. CONCLUSIONS

162. Recent research has shown that the most vulnerable life stage for many toxicants, particularly those that adversely affect the brain, is during intrauterine and early postnatal development. Fluoride fits into this paradigm, and efforts to control human fluoride exposures must therefore focus on pregnant women and small children.

163. Research on fluoride-exposed workers and laboratory animals suggest that elevated fluoride exposure is toxic to the brain and nerve cells. Epidemiological studies have identified links to learning, memory, and intelligence deficits, though most of the past studies focused on populations with fluoride exposures higher than those typically provided by U.S. water supplies.

164. Epidemiology studies of birth cohorts from the most recent years document that adverse effects on brain development happen at elevated exposure levels that occur widely in North America, in particular in communities with fluoridated drinking water. These new prospective studies are of very high quality and show very similar results, thus leaving little doubt that developmental neurotoxicity is a serious risk associated with elevated fluoride exposure. This evidence shows that community water fluoridation is associated with IQ losses that are substantial and of economic and societal concern.

165. Applying methods for standards setting routinely used by the EPA (i.e., Benchmark Dose analysis), the recent studies on IQ deficits in children allow the estimation of a recommended limit that would protect against neurotoxicity. Such calculations show that current allowable limits for fluoride in drinking water and the levels of fluoride added in community water fluoridation programs both greatly exceed a science-based limit that would protect against developmental neurotoxicity.

166. The evidence on fluoride neurotoxicity in the general population is fairly recent and

unlikely to represent the full toxicological perspective, including adverse effects that may occur at longer delays. As has been seen on numerous occasions, the evidence available today may well underestimate the true extent of the fluoride toxicity. With a reasonable degree of scientific certainty, I therefore consider the elevated levels of fluoride exposure in the U.S. population as a serious public health concern.

I declare under penalty of perjury, under the laws of the United States, that the foregoing is true and correct to the best of my knowledge and belief.

Executed on May 20, 2020, in Copenhagen, Denmark.

PHILIPPE GRANDJEAN, MD, DMSc

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Academic degrees

1974, M.D., University of Copenhagen
1975, Diploma in basic medical research, University of Copenhagen
1979, D.M.Sc. (dr.med.), University of Copenhagen

Chronology of employment

1974-1975	Postgraduate training fellowship, University of Copenhagen
1975-1978	Research fellow, Institute of Hygiene, Univ. Copenhagen
1978-1980	Senior research fellow, University of Copenhagen Visiting fellow, Department of Community Medicine, Mount Sinai School of Medicine, New York
1980-1982	Director, Department of Occupational Medicine, Danish National Institute of Occupational Health
1982-	Professor of Environmental Medicine, Odense University
1983-2017	Consultant in Toxicology, Danish Health Authority
1994-2002	Adjunct Professor of Public Health (Environmental Health) and Neurology, Boston University School of Medicine, Boston
2003-	Adjunct Professor of Environmental Health, Harvard T.H. Chan School Public Health, Boston

Awards and honors

Prize essay in medicine, University of Copenhagen (1972)
Fulbright senior research scholarship (1978)
Keynote speaker, Odense University anniversary (1983)
Gitlitz Memorial Lecture, Association of Clinical Scientists, USA (1985)

Fellow, Collegium Ramazzini (1987)
Knight of the Dannebrog, awarded by the Queen of Denmark (1990)
The Dannin prize for medical research (1991)
Fellow, American Association for the Advancement of Science (1994)
Irish Congress Lecturer, Royal College of Physicians of Ireland and Irish Society of Toxicology (1996)
Knight of the Dannebrog, First Degree, awarded by the Queen of Denmark (2003)
'Mercury madness award' for excellence in science in the public interest from eight US environmental organizations (2004)
Emeritus Fellow, International Union of Pure and Applied Chemistry, IUPAC (2009)
Honorary Research Award, International Order of Odd Fellows (2010)
Science Communication Award, University of Southern Denmark (2012)
Bernardino Ramazzini Award (2015)
Basic & Clinical Pharmacology & Toxicology Nordic Award (2015)
Margrethegaarden honorary prize (2016)
John R. Goldsmith Award, International Society for Environmental Epidemiology (2016)

Editorial boards

American Journal of Industrial Medicine (1987-2017)
Applied Organometal Chemistry (1985-1991)
Arbejdsmiljø (Occupational Environment, in Danish, 1983-1990)
Archives of Environmental Health (European Editor, 1986-1992)
Archives of Toxicology (1987-)
Biomarkers (1996-2001)
Central European Journal of Occupational and Environmental Medicine (2015-)
Critical Reviews in Toxicology (1985-2012)
Danish Medical Bulletin (1994-2003)
Environmental Health (Editor-in-Chief, 2002-)
Environmental Health Perspectives (2003-)
Environmental Research (1981-1994 and 2014-2017, Associate Editor, 1995-2014)
Industrial Health (2000-2005)
International Journal of Hygiene and Environmental Health (2001-)
International Journal of Occupational and Environmental Health (1994-2011)
International Journal of Occupational Medicine & Environ Health (1991-
Journal of Clean Technology, Environmental Toxicology, and Occupational Medicine (1992-1998)
Journal of Environmental Medicine (1998-1999)
Naturens Verden (Natural Science, in Danish) (1987-1991)
Ugeskrift for Læger (Danish Medical Journal, in Danish) (1991-2007)

Scientific societies

American Association for the Advancement of Science (Fellow, 1994)
American Public Health Association
Collegium Ramazzini (Fellow, 1987; Member of the Council, 2005-2013)
Danish Medical Association

Danish Societies of Clinical Chemistry, Epidemiology, Occupational and Environmental Medicine, and Public Health
Faroese Society of Science and Letters
International Society for Environmental Epidemiology

Teaching experience

Professor of Environmental Medicine, Odense University (University of Southern Denmark) (1982-). Member of curriculum committee. Coordinator, Global Health class.
Adjunct Professor of Public Health (Environmental Health) and Neurology, Boston University School of Medicine, Boston (1994-2002)
Adjunct Professor of Environmental Health, Harvard T.H.Chan School of Public Health, Boston (2003-)
Invited teacher, École des hautes études en santé publique (EHESP, French school of public health) (2009-)
International: Numerous teaching assignments, including guest lectures at universities and related tasks, e.g. as external examiner, National University of Singapore (1995). External evaluator of PhD theses from other universities, including University of Sydney and University of South Pacific (Fiji).

Research support as Principal Investigator since 2000

2000-2006 NIEHS
Mercury associated neurobehavioral deficit in children
2001-2003 Nordic Arctic Research Programme (NARP)
Changing patterns of biomagnified pollutants in the northern marine environment
2001-2004 Danish Medical Research Council
Exposure assessment for endocrine disruptors
2002-2004 Danish Medical Research Council
Environmental epidemiology research
2003-2004 European Commission
Assessment of Neurobehavioral Endpoints and Markers of Neurotoxicant Exposures (ANEMONE)
2003-2005 Danish Medical Research Council
Research in hormone related substances
2003-2006 NIEHS ES11687
Effects of perinatal disruptors in children
2003-2007 EPA STAR RD-83075801-0
Children's vulnerability to environmental immunotoxicant
2004-2011 NIEHS ES12199
Epidemiology of immunotoxicant exposure in children
2006-2011 NIEHS ES13692
Health effects of lifetime exposure to food contaminants
2006-2012 NIEHS ES14460
Three-generation human study of reproductive effects of marine food contaminants
2008-2012 Danish Council for Strategic Research
Environmental pollutant impact on antibody production against current and new childhood vaccines
2007-2013 NIEHS ES009797

Mercury associated neurobehavioral deficit in children
2011-2017 NIEHS ES012199
Epidemiology of immunotoxicant exposure in children
2012-2020 NIEHS ES021993 and NSF OCE-1321612
Immunotoxity in Humans with Lifetime Exposure to Ocean Pollutants
2013-2019 NIEHS ES021477
Glucose Metabolism in Adults Prenatally Exposed to Diabetogenic Pollutants
2013-2018 NIEHS ES021372
Pollutant-related diabetes in the Nurses' Health Study II
2014-2020 NIEHS ES023376
Gut Microbiome in Adults with Early Life Exposures to Environmental Chemicals

Major Current Funding as Principal Investigator
2017-2020 NIEHS ES026596
Inflammation and metabolic abnormalities in pollutant-exposed children
2017-2022 NIEHS P42ES027706
Sources, Transport, Exposure and Effects of PFASs (STEEP)
2019-2024 ATSDR TS000313
Assessment of PFAS exposures and health effects in two Massachusetts communities with PFAS drinking water contamination
2019-2023 NIEHS ES030394
Vulnerability During Infancy to Immunotoxic Contaminant Exposures

Major committees, boards and elective offices
Danish:
Danish Medical Association: Member, Prevention Council (2011-2014)
Danish Medical Research Council: Consultant on environmental medicine (1985-1990); Member, Joint Research Council Committee on Environmental Research (1986-1991); Member of DMRC (1992-1998)
Danish Society of Community Medicine: Secretary (1977-1978)
Danish Society of Industrial Medicine: Board Member (1974-1983)
Ministry of Education: Member, Committee on Toxicology (1984-1986); Member, Committee on Environmental Education (1986-1987)
Ministry of the Environment: Member, Council on Environmental Chemicals (1983-1989); Member, Environmental Appeal Board (1986-2010); Member, Environmental Research Council (1990-1992); Member, Advisory Committee on Pesticide Research (1995-2004 and 2018-2020); Member, Advisory Committee on Arctic Research (1996-2004)
Ministry of Health: numerous committee appointments; Chair, Committee on Risk Perception (2000-2001)
Ministry of Labour: Consultant on Occupational Health, Council on Occupational Safety and Health (1983-1993); Member, Occupational Health Council Research Committee (on behalf of the Danish Medical Research Council) (1984-1990 and 1999-2003)
Ministry of Research: Chair, Committee on Research at the Faroe Islands (1995-1996); Member, Committee on Scientific Dishonesty (2004-2006); Chair, Program Committee on Non-Ionizing Radiation (2004-2009)
Odense University (from 2000 University of Southern Denmark), elected

offices: Chairman, Institute of Community Health (1982-1985; 1996-1999); Member of Executive Committee, Institute of Community Health (from 2000 Institute of Public Health) (1986-1995; 2000-2005); Member, Faculty Research Committee (1983-1985); Member, Curriculum Committee (1984-1986); Member, Faculty Council (1985-1993); Vice-Dean (1991-1993); Member, Scientific Integrity Committee (2003-2022)

United States and international:

Academy of Finland: member of panel evaluating the National Institute of Public Health (1995), site visit of center of excellence (2001)

Agency for Toxic Substances and Disease Registry: Workshop Rapporteur, Neurobehavioral Test Batteries for Use in Environmental Health Field Studies (1992); Member, Expert Panel of Mercury (1998)

Association of Schools of Public Health in the European Region: Treasurer (1975-1977)

BioMedCentral: Member, Editors Advisory Group (2011-2013)

Boston Environmental Hazards Center: Consultant (1994-1999)

Collegium Ramazzini: President, International Conference, The precautionary principle: Implications for research and prevention in environmental and occupational health (2002); Member, Executive Council (2005-2013)

Commission of the European Communities: National Expert, Working Party on Environmental and Lifestyle-Related Diseases (1988-1990); ad hoc Consultant for evaluation of research applications; ad hoc Scientific Advisor on Risk Assessment (2009-); Member, Scientific Committee on Emerging and Newly Identified Health Risks; – Working group on Dental Amalgam (Human Health)(2012-2013)

European Environment Agency: Member, Scientific Committee (2012-2020)

European Food Safety Authority: Member, Panel on Contaminants in the Food Chain responsible for 85 opinions (2003-2009); Member of Working Groups on mercury, polychlorinated biphenyls, cadmium, lead, and benchmark dose

Food Advisory Committee, U.S.FDA, Methylmercury: invited expert (2002)

INMA (Infancia y Medio Ambiente), Spain: Member, Project Steering Committee (2010-)

Institut de Recherche Santé, Environnement et Travail, France: Member, Board of Advisers (2015-)

International Agency for Research on Cancer: Member of Task Group, Monographs on the Evaluation of Carcinogenic Risks to Humans, Vol. 47 (1988), Vol. 49 (1989), as chairman, Vol. 58 (1993), and as Subgroup chair, Vol. 100C (2009)

International Commission on Occupational Health: Danish Delegation Secretary (1982-90); Member, Scientific Committee on the Toxicology of Metals (1987-); Member of the Board (1990-1996)

International Programme on Chemical Safety: Member of Task Group, Environmental Health Criteria, Vol. 36 (1984) and 72 (1986)

International Society for Environmental Epidemiology: Councillor (1991-1994)

International Union of Pure and Applied Chemistry: Member, Subcommittee on the Toxicology of Nickel (1979-1989); Titular

Member (1985-1991) and Chairman (1987-1991), Commission on Toxicology; Chairman, Subcommittee on Risk Assessment (1985-1989)

Instituto de Saude Ambiental, Lisboa, Portugal: Member, External Advisory Committee (2018-2020)

Karolinska Institute (Stockholm, Sweden): Member of international evaluation panel on environmental medicine (1993)

Ministry for Scientific Policy (Belgium): Consultant on national research program on health hazards (1990 and 1994)

National Institutes of Health (USA): Member of Special emphasis panels (2009-)

NATO Priority Area Panel on Environmental Security: Member (1996-1997)

Norwegian Research Council: ad hoc reviewer (2001-2008); Chairman of Environment and Health Review Group (2009-2010); member of steering committee (2011-2015)

Prenatal Programming and Toxicity (PPTOX) conferences: Organizer/ Chair/Co-chair, Torshavn (2007), Miami (2009), Paris (2012), Boston (2014), Kita-Kyushu (2016), Torshavn (2018)

Society of Occupational and Environmental Health: Member, Governing Council (1990-1993)

Swedish Council for Work Life Research: Member, Priority Committee on Chemical Health Risks (1997-1998)

U.N.Environment Programme: Member, Global Mercury Assessment Working Group (2002)

U.S. Environmental Protection Agency: Member, SAB/SAP Endocrine Disruptor Screening Program Subcommittee (1998-1999); Member, Food Quality Protection Act (FQPA) Science Review Board (SRB)(1999-2003)

White House Office of Science and Technology Policy: Team leader and presenter, Workshop on Scientific Issues Relevant to Assessment of Health Effects from Exposure to Methylmercury (1998)

World Health Organization: Temporary Adviser or Consultant on several occasions, five times elected Rapporteur; Member, European Advisory Committee on Health Research (2011-2017)

Books

1. Grandjean P, ed. Standards setting. Copenhagen: Occupational Health Foundation, 1977, 210 pp.
2. Grandjean P, Nielsen T. Organiske blyforbindelser, forurening og toksikologi (Organolead compounds, pollution and toxicology, in Danish). Report No. SNV PM 879. Stockholm: Naturvårdsverket, 1977, 78 pp.
3. Grandjean P. Occupational health aspects of construction work. EURO Reports and Studies 86. Copenhagen: World Health Organization, Regional Office for Europe, 1983, 28 pp. (also published in German, French and Russian)
4. Grandjean P, ed. Biological effects of organolead compounds. Boca Raton, FL: CRC Press, 1984, 278 pp.
5. Grandjean P, Tarkowski S, eds. Toxic oil syndrome: mass food poisoning in Spain. Copenhagen: World Health Organization, Regional Office for Europe, 1984, 92 pp. (also published in Spanish)
6. Grandjean P. Miljø og forebyggelse. (Environment and prevention, student's guide in Danish). Copenhagen: F.a.d.L.'s Forlag, 1984, 109 pp.
7. Gilioli R, Grandjean P, Johnson B, Seppäläinen AM, Tarkowski S, eds. Neurobehavioural methods in occupational and environmental health. Environmental Health No. 3. Copenhagen: World Health Organization, Regional Office for Europe, 1985, 209 pp.
8. Grandjean P, ed. Neurobehavioural methods in occupational and environmental health. Environmental Health No. 6. Copenhagen: World Health Organization, Regional Office for Europe, 1985, 72 pp.
9. Grandjean P, ed. Miljømedicin (Environmental medicine, textbook in Danish). Copenhagen: F.a.d.L.'s Forlag, 1986, 257 pp.
10. Grandjean P, ed. Trace elements in human health and disease: extended abstracts. Environmental Health No. 20. Copenhagen: World Health Organization, Regional Office for Europe, 1987, 230 pp.
11. Grandjean P, ed. Trace elements in human health and disease: symposium report. Environmental Health No. 26. Copenhagen: World Health Organization, Regional Office for Europe, 1987, 134 pp.
12. Grandjean P, Kimbrough RD, Rantanen J, Tarkowski S, Yrjänheikki E. Assessment of health risks in infants associated with exposure to PCBs, PCDDs and PCDFs in breast milk. Environmental Health No. 29. Copenhagen: World Health Organization, Regional Office for Europe, 1988, 116 pp.
13. Grandjean P, ed. Miljømedicin, 2. udg. (Environmental medicine, 2nd ed., textbook in Danish). Copenhagen: F.a.d.L.'s Forlag, 1988, 311 pp.
14. Kimbrough RD, Mahaffey KR, Grandjean P, Sandø SH, Ruttstein DD. Clinical Effects of Environmental Chemicals: A Software Approach to Etiologic Diagnosis. New York: Hemisphere, 1989, 110 pp. and one floppy disk.
15. Grandjean P. Skin Penetration: Hazardous Chemicals at Work. (Published on behalf of the Commission of the European Communities.) London: Taylor and Francis, 1990, 187 pp.
16. Grandjean P, ed. Ecogenetics: Genetic Predisposition to Toxic

Effects of Chemicals. London: Chapman & Hall, 1991, 288 pp.
17. Grandjean P, ed. Miljø, sundhed og samfund (Environment, health and society, in Danish). Copenhagen: Nyt Nordisk Forlag, 1991, 453 pp.
18. Grandjean P, Brown SS, Reavey P, Young DS, Rej R (eds). Biomarkers of Chemical Exposure. Proceedings of the Arnold O. Beckman/IFCC European Conference on Environmental Toxicology. Clin Chem 1994; 40 (issue 7B).
19. Toppari J, Larsen JC, Christiansen P, Giwercman A, Grandjean P, Guillette LJ, Jr, Jégou B, Jensen TK, Jouannet P, Keiding N, Leffers H, McLachlan JA, Meyer O, Müller J, Rajper-DeMeyts E, Scheike T, Sumpter J, Skakkebaek N. Male reproductive health and environmental chemicals with estrogenic effects. Copenhagen: Danish Environmental Protection Agency, 1995, 166 pp.
20. Grandjean P, Brown SS, Reavey P, Young DS, Sampson E (eds). Biomarkers. Proceedings of the Second Arnold O. Beckman/IFCC European Conference on Environmental Toxicology. Clin Chem 1995; 41 (issue 12B).
21. Grandjean P. Farlig forurening (Dangerous pollution, in Danish). Copenhagen: Nyt Nordisk Forlag and National Board of Health, 1998, 174 pp.
22. Grandjean P ed. Human health effects of environmental mercury exposure (special issue). Environ Res 1998; 77 (67-177).
23. Grandjean P, Sofritti M, Minardi F, Brazier J (eds). The Precautionary Principle. Implications for research and prevention in environmental and occupational health. Eur J Oncol Library 2003; 2: 1-245. Also published in Int J Occup Med Environ Health 2004; 17: 3-201.
24. Grandjean P (ed). Prenatal programming and toxicity. Basic Clin Pharmacol Toxicol. 2008; 102(2): 71-273.
25. Gee D, Grandjean P, Hansen SF, van den Hove S, MacGarvin M, Martin J, Nielsen G, Quist D, Stanners D, eds. Late Lessons from Early Warnings, volume II (EEA Report No 1/2013). Copenhagen, European Environment Agency, 2013, 746 pp.
26. Grandjean P. Only one chance. How Environmental Pollution Impairs Brain Development – and How to Protect the Brains of the Next Generation. New York: Oxford University Press, 2013 (232 pp.).
27. Grandjean P, Hermann P. Kemi på hjernen – går ud over enhver forstand. København: Gyldendal, 2015 (334 sider).
28. Grandjean P. Cerveaux en danger (Brains in danger, in French). Translated by Odile Demange. Paris: Buchet Chastel, 2016 (336 pp.).
29. Kishi R, Grandjean P, eds. Health Impacts of Developmental Exposure to Environmental Chemicals. Singapore: Springer, 2020 (555 pp.)

Publications in international peer-reviewed journals

1. Grandjean P, Holma B. A history of lead retention in the Danish population. Environ Biochem Physiol 1973; 3: 268-73.
2. Grandjean P. Lead in Danes, historical and toxicological studies. Environ Qual Saf 1975; Suppl. Vol. 2: 6-75.
3. Grandjean P. Possible effect of lead on egg-shell thickness in kestrels 1874-1974. Bull Environ Contam Toxicol 1976; 16: 101-6.
4. Grandjean P. Regional distribution of lead in human brains. Toxicol Lett 1978; 2: 65-9.
5. Nielsen T, Jensen KA, Grandjean P. Organic lead in normal human brains. Nature (Lond.) 1978; 274: 602-3.
6. Grandjean P. Lead concentration in single hairs as a monitor of occupational lead exposure. Int Arch Occup Environ Health 1978; 42: 69-81.
7. Grandjean P, Lintrup J. Erythrocyte-Zn-protoporphyrin as an indicator of lead exposure. Scand J Clin Lab Invest 1978; 38: 669-75.
8. Grandjean P, Arnvig E, Beckmann J. Psychological dysfunctions of lead-exposed workers: Relation to biological parameters of exposure. Scand J Work Environ Health 1978; 4: 295-303.
9. Grandjean P. Widening perspectives of lead toxicity, a review of health effects of lead exposure in adults. Environ Res 1978; 17: 303-21. (Also published as a special report to the U.S. National Institute of Environmental Health Sciences)
10. Grandjean P. Occupational lead exposure in Denmark: Screening with the haematofluorometer. Br J Ind Med 1979; 36: 52-8.
11. Grandjean P, Nielsen OV, Shapiro IM. Lead retention in ancient Nubian and contemporary populations. J Environ Path Toxicol 1979; 2: 781-7.
12. Grandjean P, Nielsen T. Organolead compounds, environmental health aspects. Residue Rev 1979; 72: 97-148.
13. Arnvig E, Grandjean P, Beckmann J. Neuropsychological effect of heavy lead exposure determined with psychological tests. Toxicol Lett 1980; 5: 399-404.
14. Hertz MM, Bolwig TG, Grandjean P, Westergaard E. Lead poisoning and the blood-brain barrier. Acta Neurol Scand 1981; 63: 286-96.
15. Grandjean P, Selikoff IJ, Shen SK, Sundermann FW Jr. Nickel concentrations in plasma and urine of shipyard workers. Am J Ind Med 1981; 1: 181-9.
16. Olsen NB, Hollnagel H, Grandjean P. Indicators of lead exposure in an adult Danish suburban population. Dan Med Bull 1981; 28: 168-76.
17. Grandjean P, Olsen NB, Hollnagel H. Influence of smoking and alcohol consumption on blood lead levels. Int Arch Occup Environ Health 1981; 48: 391-7.
18. Grandjean P, Kon SH. Lead exposure of welders and bystanders in a ship repair yard. Am J Ind Med 1981; 2: 65-70.
19. Grandjean P, Lintrup J. Sources of variation in fluorometry of zinc-protoporphyrin in blood. Scand J Work Environ Health 1981; 7: 311-2.
20. Grandjean P, Olsen NB, Hollnagel H. Occupationally related lead exposure in the general population. Scand J Work Environ Health 1981;

7: 298-301.
21. Grandjean P. Occupational fluorosis through 50 years: clinical and epidemiological experiences. Am J Ind Med 1982; 3: 227-36.
22. Nielsen OV, Grandjean P, Bennike P. Chemical analyses of archaeological bone samples: Evidence for high lead exposure on the Faroe Islands. J Dan Archaeol 1982; 2: 145-8. (also published in Faroese: Blyggj i føroyingum, Mondul 1983; 9: 27-31)
23. Grandjean P. Storage depots in the body: Passive retention or time bomb? (Editorial) Am J Ind Med 1983; 4: 489-90.
24. Grandjean P, Wulf HC, Niebuhr E. Sister chromatid exchange in response to variations in occupational lead exposure. Environ Res 1983; 32: 199-204.
25. Grandjean P, Thomsen G. Reversibility of skeletal fluorosis. Br J Ind Med 1983; 40: 456-61.
26. Grandjean P. Lead poisoning: Hair analysis shows the calendar of events. Hum Toxicol 1984; 3: 223-8.
27. Grandjean P, Hansen ON, Lyngbye K. Analysis of lead in circum-pulpal dentin of deciduous teeth. Ann Clin Lab Sci 1984; 14:270-5.
28. Eskildsen J, Grandjean P. Lead exposure from lead pellets: Age-related accumulation in mute swans. Toxicol Lett 1984; 21: 225-9.
29. Grandjean P, Juel K, Jensen OM. Mortality and cancer morbidity after heavy occupational fluoride exposure. Am J Epidemiol 1985; 121: 57-64.
30. Lyngbye T, Hansen ON, Vangberg L, Grandjean P. Lead as a cause of SIDS. N Engl J Med 1985; 10: 954-5.
31. Grandjean P. Reference intervals for toxic metals: Problems and prospects. Ann Clin Lab Sci 1986; 16: 67-74.
32. Grandjean P, Bach E. Indirect exposures: The significance of bystanders at work and at home. Am Ind Hyg Assoc J 1986; 47: 819-24.
33. Grandjean P, Lyngbye T, Hansen ON. Lead concentration in deciduous teeth: Variation related to tooth type and analytical technique. J Toxicol Environ Health 1986; 19: 437-45.
34. Grandjean P. After Chernobyl (Editorial). Arch Environ Health 1986; 41: 277.
35. Andersen O, Grandjean P. Effects of inorganic and organic lead compounds on chromosomal length in human lymphocytes. Appl Organomet Chem 1987; 1: 15-19.
36. Grandjean P, Andersen O, Nielsen GD. Carcinogenicity of occupational nickel exposures: An evaluation of the epidemiological evidence. Am J Ind Med 1988; 13: 193-209.
37. Christoffersen J, Christoffersen MR, Larsen R, Rostrup E, Tingsgaard P, Andersen O, Grandjean P. Interaction of cadmium ions with calcium hydroxyapatite crystals: A possible mechanism contributing to the pathogenesis of cadmium-induced diseases. Calcif Tissue Int 1988; 42: 331-9.
38. Grandjean P, Berlin A, Gilbert M, Penning W. Preventing percutaneous absorption of industrial chemicals: The "skin" denotation. Am J Ind Med 1988; 14: 97-107.
39. Lyngbye T, Hansen ON, Grandjean P. Bias resulting from non-participation in childhood epidemiological studies: A study of low-level lead exposure. Scand J Soc Med 1988; 16: 209-15.

40. Grandjean P. Ancient skeletons as silent witnesses of lead exposures in the past. CRC Crit Rev Toxicol 1988; 19:11-21.
41. Madsen HHT, Skjødt T, Jørgensen PJ, Grandjean P. Blood lead levels in patients with lead shot retained in the appendix. Acta Radiol 1988; 29: 745-6.
42. Andersen O, Grandjean P. Effects of tetraethylthiuram disulfide on the toxicokinetics of cadmium in mice. Pharmacol Toxicol 1989; 64: 210-5.
43. Lyngbye T, Hansen ON, Grandjean P. Neurological deficits in children: Medical risk factors and lead exposure. Neurotoxicol Teratol 1989; 10: 531-7.
44. Grandjean P, Hollnagel H, Hedegaard L, Christensen JM, Larsen S. Blood lead-blood pressure relationships: Alcohol intake and hemoglobin as confounders. Am J Epidemiol 1989; 129: 732-9.
45. Hansen ON, Trillingsgaard A, Beese I, Lyngbye T, Grandjean P. A neuropsychological study of children with elevated dentine lead level: Assessment of the effect of lead in different socioeconomic groups. Neurotoxicol Teratol 1989: 11: 205-13.
46. Grandjean P, Jensen BM, Sandø SH, Jørgensen PJ, Antonsen S. Delayed blood regeneration in lead exposure: An effect on reserve capacity. Am J Publ Health 1989; 79: 1385-8.
47. Grandjean P. Bone analysis: Silent testimony of lead exposures in the past. Medd Grønland Man Soc 1989; 12: 156-60.
48. Grandjean P, Hørder M, Thomassen Y. Fluoride, aluminum and phosphate kinetics in cryolite workers. J Occup Med 1990;32:58-63.
49. Grandjean P, Kristensen K, Jørgensen PJ, Nielsen GD, Andersen O. Trace element status in alcoholism before and during disulfiram treatment. Ann Clin Lab Sci 1990; 20: 28-35.
50. Nielsen GD, Jepsen LV, Jørgensen PJ, Grandjean P, Brandrup F. Nickel-sensitive patients with vesicular hand eczema: Oral challenge with a diet naturally high in nickel. Br J Dermatol 1990; 122: 299-308.
51. Lyngbye T, Hansen ON, Trillingsgaard A, Beese I, Grandjean P. Learning disabilities in children: significance of low-level lead-exposure and confounding factors. Acta Paed Scand 1990; 79: 352-60.
52. Jensen BM, Sandø SH, Grandjean P, Wiggers P, Dalhøj J. Screening with zinc-protoporphyrin for iron deficiency in non-anemic female blood donors. Clin Chem 1990; 36: 846-8.
53. Lyngbye T, Grandjean P, Hansen ON, Jørgensen PJ. Validity and interpretation of blood lead levels: A study of Danish school children. Scand J Clin Lab Invest 1990; 50: 441-9.
54. Bonde I, Beck H-I, Jørgensen PJ, Grandjean P, Brandrup F. Nickel in intercellular fluid, comparison between nickel-allergic patients and controls. Acta Derm Venereol (Stockh) 1990; 70: 300-3.
55. Lyngbye T, Hansen ON, Grandjean P. Predictors of tooth-lead level with special reference to traffic. Int Arch Occup Environ Health 1990; 62: 417-22.
56. Grandjean P, Jørgensen PJ. Retention of lead and cadmium in prehistoric and modern human teeth. Environ Res 1990; 53: 6-15.
57. Lyngbye T, Hansen ON, Grandjean P. Lead concentration in deciduous teeth from Danish school children. Dan Med Bull 1991; 38: 89-93.

58. Grandjean P, Jacobsen IA, Jørgensen PJ. Chronic lead poisoning treated with DMSA. Pharmacol Toxicol 1991; 68: 266-9.
59. Grandjean P, Jørgensen PJ, Viskum S. Temporal and interindividual variation in erythrocyte zinc-protoporphyrin in lead-exposed workers. Br J Ind Med 1991; 48: 254-7.
60. Grandjean P, Sandoe SH, Kimbrough RD. Nonspecificity of clinical signs and symptoms caused by environmental chemicals. Hum Exp Toxicol 1991; 10: 167-73.
61. Grandjean P, Lyngbye T, Hansen ON. Lessons from a Danish study on neuropsychological impairment related to lead exposure. Environ Health Perspec 1991; 94: 111-5.
62. Grandjean P, Andersen O. Lung cancer in filling station attendants. Am J Ind Med 1991; 20: 763-8.
63. Grandjean P, Weihe P, Jørgensen PJ, Clarkson T, Cernichiari E, Viderø T. Impact of maternal seafood diet on fetal exposure to mercury, selenium, and lead. Arch Environ Health 1992; 47: 185-95.
64. Grandjean P, Nielsen GD, Jørgensen PJ, Hørder M. Reference intervals for trace elements in blood: Significance of risk factors. Scand J Clin Lab Invest 1992; 52: 321-337.
65. Grandjean P, Olsen JH, Jensen OM, Juel K. Cancer incidence and mortality in workers exposed to fluoride. J Natl Cancer Inst 1992; 84: 1903-9.
66. Grandjean P. Individual susceptibility to toxicity. Toxicol Lett 1992; 64/65: 43-51.
67. Grandjean P. International research on the relation between health and the environment (summary in French). Santé Publique 1992; 4: 103-8.
68. Grandjean P. Symposium synthesis, Application of neurobehavioral methods in environmental and occupational health. Environ Res 1993; 60: 57-61.
69. Grandjean P, Weihe P. Neurobehavioral effects of intrauterine mercury exposure: potential sources of bias. Environ Res 1993; 61: 176-83.
70. Damm D. Grandjean P, Lyngbye T, Trillingsgaard A, Hansen ON. Early lead exposure and neonatal jaundice: Relation to neurobehavioral performance at 15 years of age. Neurotoxicol Teratol 1993; 15: 173-81.
71. Grandjean P, Andersen D. Scientific dishonesty: a Danish proposal for evaluation and prevention. J Exposure Anal Environ Epidemiol 1993; 3, Suppl. 1: 265-70.
72. Grandjean P. International perspectives of lead exposure and lead toxicity. Neurotoxicol 1993; 24: 9-14.
73. Olsen S, Grandjean P, Weihe P, Viderø T. Seafood intake in pregnancy as a determinant of birth weight: Evidence for a dose-dependent relationship. J Epidemiol Comm Health 1993; 47: 436-40.
74. Grandjean P, Jørgensen PJ, Weihe P. Human milk as a source of methylmercury exposure in infants. Environ Health Perspec 1994; 102: 74-7.
75. Dalgård C, Grandjean P, Jørgensen PJ, Weihe P. Mercury in the umbilical cord: Implications for risk assessment for Minamata disease. Environ Health Perspec 1994; 102: 548-50.
76. Grandjean P, Weihe P, Nielsen JB. Methylmercury: Significance of

intrauterine and postnatal exposures. Clin Chem 1994; 40: 1395-1400.
77. Grandjean P, Brown S, Reavey P, Young D. Biomarkers of chemical exposure: state of the art. Clin Chem 1994; 40: 1360-2.
78. Nielsen JB, Andersen O, Grandjean P. Evaluation of mercury in hair, blood and muscle as biomarkers for methylmercury exposure in male and female mice. Arch Toxicol 1994; 68: 317-21.
79. Johnson BL, Grandjean P, Amler R. Neurobehavioral testing and hazardous chemical sites. Neurotoxicol Teratol 1994; 16: 485-7.
80. Grandjean P, Weihe P, White RF. Milestone development in infants exposed to methylmercury from human milk. Neurotoxicol 1995; 16: 27-33.
81. Grandjean P. Individual susceptibility in occupational and environmental toxicology. Toxicol Lett 1995; 77: 105-8.
82. Grandjean P. Biomarkers in epidemiology. Clin Chem 1995; 41: 1800-3.
83. Grandjean P, Brown SS, Reavey P, Young DS. Biomarkers in environmental toxicology: State of the art. Clin Chem 1995; 41: 1902-4.
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UNITED STATES DISTRICT COURT
FOR THE NORTHERN DISTRICT OF CALIFORNIA
AT SAN FRANCISCO

FOOD & WATER WATCH, et al., Plaintiffs, vs. U.S. ENVIRONMENTAL PROTECTION AGENCY, et al., Defendants.	Civ. No. 17-CV-02162-EMC **DECLARATION OF HOWARD HU, MD, MPH, ScD**

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I, Howard Hu, MD, MPH, ScD, declare that:

1. I am a physician-scientist trained in internal medicine, occupational/environmental medicine, epidemiology and general public health who has held leadership positions in science and academia for over 2 decades.

2. I am also the Principal Investigator of ongoing research that is examining the impact of early-life exposures to fluoride on neurobehavioral development in the offspring participating in the Early Life Exposures in Mexico to Environmental Toxicants (ELEMENT) project.

I. SUMMARY OF QUALIFICATIONS

3. A complete summary of my qualifications and publications can be found in my Curriculum Vitae, which has been marked as Plaintiffs' Exhibit 5 and attached herein.

4. As relevant to my testimony here, I am an epidemiologist with decades of research experience investigating the impact of environmental toxicants on human health. In 1990, I received a Doctor of Science degree in Epidemiology from the Harvard School of Public Health, and since that time have taught epidemiology at Harvard, University of Michigan, and University of Toronto, where I served as Dean of the School of Public Health.

5. I hold editorial positions on leading environmental and occupational health journals, including the *American Journal of Industrial Medicine, Current Environmental Health Reports* and *Environmental Health Perspectives*, and serve as a peer reviewer for the *American Journal of Epidemiology*, *Epidemiology*, *Journal of the American Medical Association*, *Lancet*, *New England Journal of Medicine*, and *Pediatrics,* amongst others.

6. My own epidemiological research has resulted in hundreds of peer-reviewed publications in leading scientific journals. For the past 29 years, this research has been continuously funded by the National Institutes of Health (NIH) through a number of competitive R01 grants.

7. The Environmental Protection Agency (EPA) has funded several of my epidemiological studies, including a $7.8 million research grant to study the effects of metals mixtures on children's health. I have also served as an expert advisor to the EPA, including as a member of EPA's Science Advisory Board on Relative Risk Reduction Strategies and as an expert reviewer of EPA's recent draft report on the concentration-response functions between lead exposure and cardiovascular disease.

8. In 1993, I co-founded the ELEMENT research project, a pregnancy and birth cohort that has been funded by both the EPA and NIH. Since its inception, ELEMENT has evolved into a highly successful, award-winning project involving collaborators at the University of Michigan, Harvard, and other academic institutions in the U.S., Canada, and Mexico.

9. Through the ELEMENT cohort, we have studied how prenatal exposure to environmental toxicants—including lead, mercury, and fluoride—affect children's health, including their neurodevelopment. Thus far, the ELEMENT cohort has generated over 80 high-impact publications and provided evidence contributing towards environmental health policies around the world, including the EPA's national air standard for lead and the CDC's "Guidelines for the Identification and Management of Lead Exposure in Pregnant and Lactating Women."

10. In 2012, following the National Research Council's (NRC) call for more research to investigate the neurobehavioral risks of fluoride exposure, the team I lead successfully competed for a peer-reviewed NIH RO1 grant to study the neurodevelopmental effects of pre- and post-natal fluoride exposures. This research was funded with an understanding that it would provide a major contribution to fluoride risk assessment and policy decision-making on the neurotoxicity concerns identified by the NRC.

11. To date, my team has published five peer-reviewed studies on fluoride, including two prospective studies on fluoride and neurodevelopment that were published in the world's two most

prominent environmental health journals: *Environmental Health Perspectives* and *Environment International* (Bashash 2017, Bashash 2018, Cantoral 2019, Liu 2019, Thomas 2016). As with our own research, the journal *Environmental Health Perspectives* is funded by the NIH.

II. SUMMARY OF OPINIONS

12. The ELEMENT prospective cohort studies of fluoride's neurodevelopmental effects are methodologically rigorous studies that provide scientifically reliable and robust results.

13. The results of the ELEMENT prospective cohort studies are consistent with and support the conclusion that fluoride is a developmental neurotoxicant at levels of exposure seen in the general population in water-fluoridated communities.

III. BASIS FOR OPINIONS

A. The Methodological Strengths of the ELEMENT Studies

14. ***Prospective Birth Cohort Study Design***: One of the key strengths of our ELEMENT research is that it has utilized a prospective birth cohort design. Prospective studies (aka longitudinal cohort studies designed at the outset to enable research on the topics of interest and that follow a defined group of individuals) are recognized by epidemiologists as the ideal study design for investigating the impact of environmental toxicants on human health, in part, because the measurement of exposure precedes the development of disease/dysfunction. This is important because it permits greater confidence in the causal relationship of an association since the requirement of temporality is satisfied i.e., the exposure precedes the effect, which, in turn, is one of the components of the Bradford Hill criteria. Where there is suspicion that a chemical may exert a toxic effect *in utero*, a birth cohort study design is critical because it allows an assessment of exposure during the prenatal period.

15. ***Effective Control for Other Factors that Influence IQ:*** Our ELEMENT studies have considered and controlled for a large number of factors known to affect neurodevelopment, which

increases the rigor of the results. First, we excluded women from the study who had characteristics known to affect neurodevelopment, including gestational diabetes, renal disease, hypertension, circulatory diseases, use of illicit drugs, and alcohol consumption. Second, our analyses of fluoride and neurodevelopment controlled for a large number of potential confounders, including maternal age, maternal education, maternal IQ, birth weight, gestational age at time of delivery, sex of child, birth order, maternal smoking, and marital status. Third, we performed sensitivity analyses which controlled for the quality of the child's home environment (i.e., HOME),[1] as well as prenatal lead and mercury exposures.

16. ***Blinded Assessments***: Our studies employed a "blinded" study design where neither the examiners nor the subjects were aware of the subject's fluoride exposure status at the time of the neurodevelopmental exams. A blinded study design is superior to a non-blinded study because it helps protect against bias, including unconscious bias, in the assessment.

17. ***Individual Biomarkers of Both Prenatal and Postnatal Exposure***: As cohort studies, our investigations have collected and utilized individual measurements of fluoride exposure, covariates, and the outcomes of interest. Cohort studies with individual measurements of exposure, covariates and outcomes are considered much more robust than studies with group-level metrics (otherwise known as "ecological studies") because the accuracy and precision of individual-level measures are far superior to the estimates of these parameters that are associated with ecological studies. In our studies, we measured prenatal fluoride exposure by testing archived samples of the mother's urine that were collected during pregnancy. Urine fluoride is a well-accepted biomarker of total fluoride exposure. As the EPA has

[1] The HOME Score is a rating that is performed by a research observer who gains permission to enter the home and observe the interactions between the offspring and other family members, as well as other characteristics of the home environment. It is intended to try to capture the ability of the home to enrich a child's educational development and skill development. It is often highly correlated with socio-economic status, but it also has some independent value of its own in determining a child's neurodevelopmental trajectory.

recognized, "archives of biological samples from birth cohort studies . . . provide critical information on the prenatal and childhood determinants of adult disease" (EPA-NIEHS 2017, p. 9). The archived urine samples in our studies were tested under the oversight and direction of Dr. Angeles Martinez-Mier, a leading authority on the measurement of fluoride in urine and plasma.

18. As I acknowledge and discuss further below, there are some limitations with our urine-based exposure estimates. These limitations, however, do not provide a plausible explanation for the results we have observed as they create *non-differential* imprecision in the exposure variable (in this case, the non-differential exposure misclassification is sometimes referred to as "random" or "classical" measurement error associated with exposure). It is a basic epidemiologic axiom that non-differential errors, such as non-differential exposure misclassification, bias the results towards the null (i.e., no association exists), rather than create spurious associations where none otherwise exist. I discuss this further below.

19. ***Large Cohort Sizes***: Our studies have involved a sufficiently large number of mother-offspring pairs to permit statistical analyses that are stable and robust. In our 2017 study ("Bashash 2017"), we investigated fluoride's relationship with intelligence among 299 mother-offspring pairs. This included 287 mother-offspring pairs for the analysis of intelligence at age 4, and 211 mother-offspring pairs for an analysis of intelligence at ages 6-12. In our 2018 study ("Bashash 2018"), we investigated the relationship between fluoride and symptoms of Attention-Deficit Hyperactivity Disorder (ADHD) among a total of 213 mother-offspring pairs, with the ADHD assessment conducted between the ages of 6 and 12.

20. ***Reliable Neurocognitive Tests:*** Our 2017 study on intelligence used validated, standardized neurocognitive tests that were administered by a team of psychologists. For the 4-year old children, we used the McCarthy Scales of Children's Abilities (MSCA), and focused on the General

Cognitive Index (GCI) score. For the 6-12 year old children, we used the Spanish version of the Wechsler Abbreviated Scale of Intelligence (WASI)[2] to assess Full-Scale IQ. All raw scores were standardized for age and sex. The examining psychologists were trained and supervised by an experienced developmental psychologist, and independent testing confirmed a very high correlation (0.99) in the scoring, thus confirming a high degree of inter-examiner reliability.

21. ***Reliable Neurobehavioral Tests:*** Behaviors associated with ADHD were assessed using the Spanish version of the Conners' Rating Scales-Revised (CRS-R)[3] and Conners' Continuous Performance Test (CPT-II, 2nd Edition).[4] All measures of ADHD-behaviors were standardized for age- and sex. Higher T-scores (mean of 50, SD of 10) indicate poorer performance. All psychometric tests were applied under the supervision of an experienced psychologist.

22. ***Appropriate Statistical Analyses that Did Not Assume Linearity***: We used the same standard statistical analyses for our fluoride studies as we have used for our other ELEMENT studies. These included regression analyses that appropriately adjusted for potential confounders, as well as Generalized Additive Models (GAM) that visualized adjusted associations between fluoride and neurodevelopment for purposes of assessing the linearity of the relationship. We did *not* assume

[2] The WASI shows strong criterion validity with the full-length Wechsler Intelligence Scale for Children (WISC-III; ages 6-16 yrs), and the Wechsler Adult Intelligence Scale (WAIS; ages 16+ yrs). The correlation coefficient between the Full Scale IQ of the WASI and WISC-III is 0.81 (Wechsler, 1991), and 0.92 between the WASI and the WAIS-III (Wechsler, 1999), indicating a high covariance between the abbreviated and full-length measures of intellectual ability. The WASI is also correlated with another abbreviated IQ test, the Kaufman Brief Intelligence Test (r = 0.89), providing evidence for convergent validity (Hays et al., 2002). Finally, the WASI demonstrates excellent internal consistency (reliability=0.976) (based on data from Tables 5.1 and 5.8 of the WASI manual).

[3] The CRS-R contains three ADHD scales that correspond with the Diagnostic and Statistical Manual of Mental Disorders – 4th edition (DSM-IV) criteria for ADHD: 1) DSM-IV Inattention Index, 2) DSM-IV Hyperactive-Impulsive Index, and 3) DSM-IV Total Index (inattentive and hyperactive-impulsive behaviors combined). It also examines seven types of behavior problems that were derived through factor analysis, including: Oppositional, Anxious-Shy, Cognitive Problem/Inattention, Hyperactivity, Perfectionism, Psychosomatic, and Social Problems. For our study, we examined the three DSM-IV ADHD scales as our primary outcomes because these scales are intended to screen for ADHD, and are commonly used to study the association between diverse environmental contaminants and ADHD- behavior problems.

[4] The CPT-II is a computer-administered signal detection paradigm. Using the CPT-II, we measured errors of omission and commission, and hit reaction time (response latency).

linearity in the dose-response relationship. Additionally, we took appropriate steps to eliminate the influence of outliers and influential points.

B. Prenatal Fluoride Exposure Is Associated with Substantial and Significant Adverse Effects on IQ and ADHD-Behaviors in the ELEMENT Cohort

23. In the ELEMENT cohort, we found that prenatal fluoride exposure has a linear, dose-response relationship with reduced IQ among both 4-year old and 6-12 year old children (Bashash 2017).[5] In our main model that adjusted for potential confounders, we found that each 0.5 mg/L increase in maternal urinary fluoride (which approximates the interquartile range, i.e., the difference between the 25^{th} v. 75^{th} percentile) was significantly associated with a loss of 3.15 GCI points among the 4-year-olds, and a loss of 2.5 IQ points among the 6-to-12 year olds. These are substantial reductions in intelligence that rival the effect sizes associated with lead exposure. As one measure of practical impact developed and published in 2009 by an expert from the Economics Policy Institute, each IQ point lost due to lead exposure was estimated to represent a loss of $17,815 in present discounted value of lifetime earnings (in 2006 USD) (Gould 2009).

24. Visual assessment of the adjusted associations between fluoride and intelligence confirmed the monotonic, mostly linear nature of the relationships (see Figures A and B). Notably, there was no evidence of a threshold among the 4-year olds, although there was some suggestion of a threshold at approximately 0.8 mg/L among the 6-12 year olds.

[5] We have subsequently reported an analysis, in abstract form, of neurocognitive outcomes at ages 1 to 3, as measured through the Mental Development Index (Thomas 2018). These results are consistent with the age 4 and age 6-12 analyses in that they show significant adverse associations with maternal urinary fluoride. I do not rely on these results here, however, since they have not yet been published in full

Figure A: Visual Association Between Maternal Urinary Fluoride and Intelligence at Age 4

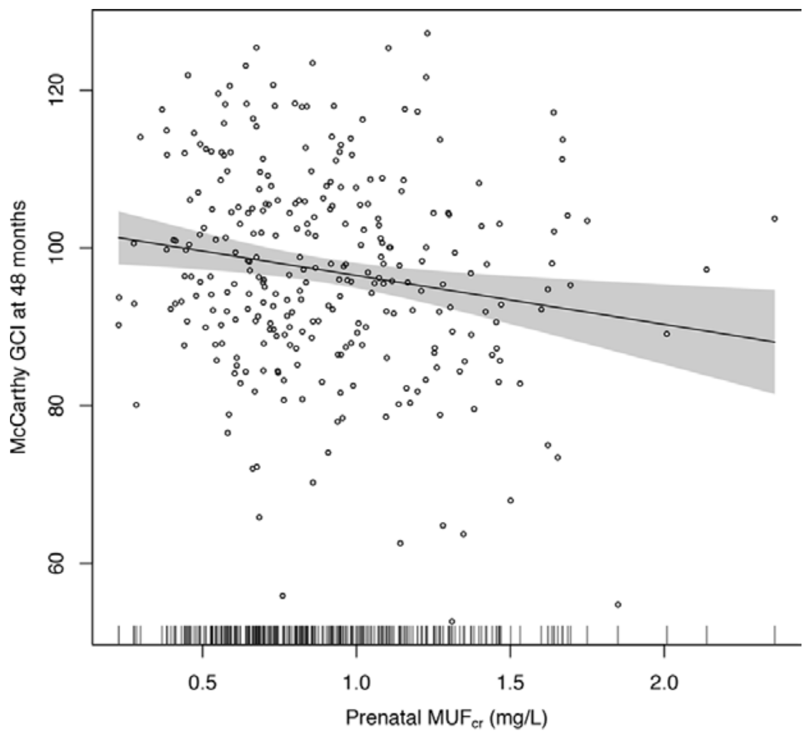
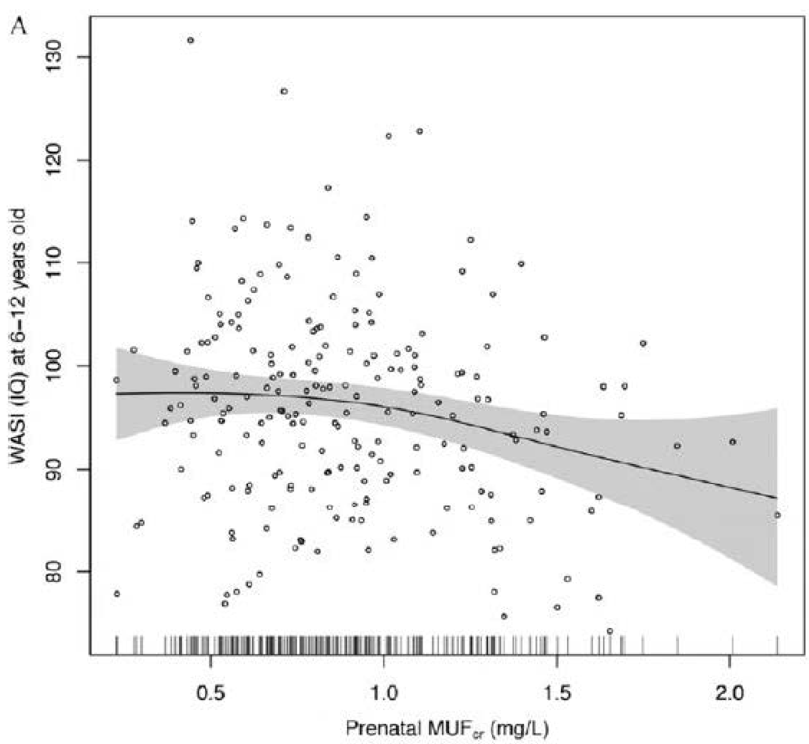


Figure B: Visual Association Between Maternal Urinary Fluoride and Intelligence at Ages 6-12

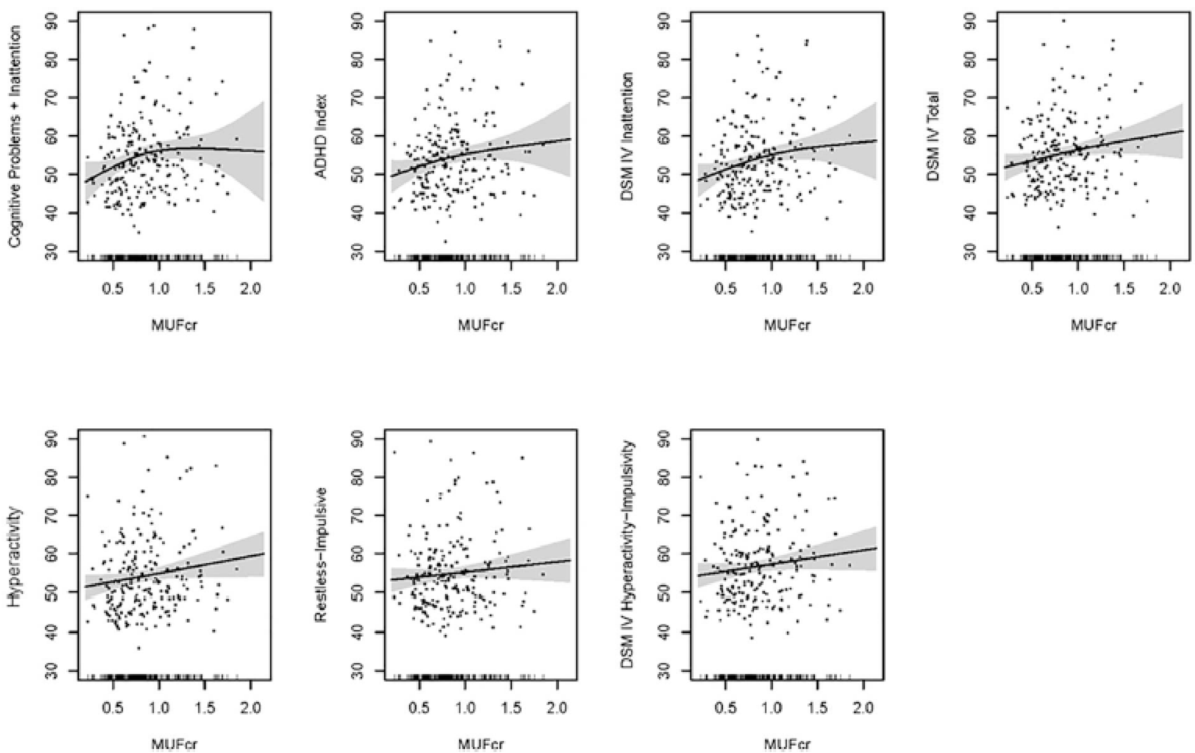


25. In contrast with prenatal exposures, we did not find statistically significant associations between IQ and childhood urinary fluoride levels at ages 6 to 12, although there was some suggestion of an adverse effect. This suggests that the timing of fluoride exposure is an important determinant of fluoride's neurodevelopmental effects, and is consistent with exposures occurring prenatally being more detrimental than those occurring during school-aged years. This is plausible given fluoride's passage through the placental barrier, and the known enhanced vulnerability of the developing brain to neurotoxicants during the *in utero* period.

26. In addition to IQ, we have also found a significant association between prenatal fluoride exposure and some attention deficit hyperactivity disorder (ADHD)-like behaviors on the CRS-R test, including cognitive problems and inattention (Bashash 2018). As with our IQ analyses, the associations were linear, although—as we have found for lead (Huang 2016)—there was some indication in some of the analyses of a ceiling effect at higher doses (i.e., the dose-response curve for cognitive problems and inattention began to flatten above 1 mg/L).

27. The effect sizes between prenatal fluoride and ADHD behaviors in our cohort were substantial. For those effects which reached statistical significance, increases of 0.5 mg/L in maternal urinary fluoride were associated with 2.4 to 2.8-point higher scores (higher scores reflect indicate poorer performance). Whereas IQ is standardized to a mean of 100, the ADHD behavior scales are standardized to a scale of 50. The effect sizes that we found for prenatal fluoride are similar to what we have found for childhood blood lead levels (Huang 2016).

Figure C: Association Between Maternal Urinary Fluoride & ADHD Behaviors



28. We did not find statistically significant associations between fluoride and ADHD-behaviors on the CPT-II test. Other studies of environmental chemicals have reported similar discrepancies between the two tests, suggesting that they are assessing different constructs. The stronger association that we found between fluoride and ADHD behaviors on the CRS-R scale may be explained by the CRS-R's focus on constructs that rely on attention (e.g., new learning, ability to hold information and complete tasks, organizational skills, etc).

29. The relationship we observed between fluoride and inattention is consistent with some animal research that has reported a relationship between prenatal fluoride exposure and hypoactive behavioral patterns (Mullenix 1995), as well as prior epidemiological research associating fluoride with impaired working memory (Choi 2015). Working memory is linked with the ability to control attention and it is common for youth with ADHD to have weaknesses in working memory (Kasper 2012).

Fluoride's effect on working memory may relate to an effect on the dopamine system, which fluoride has been found to alter in animal studies (Pal & Sarkar 2014). Dopamine is an important modulatory neurotransmitter in planning and initiation of motor responses, activation, switching, reaction to novelty and processing of reward (Fararone 2015).

30. Some have suggested that the "scatter" in the above scatterplots is a basis to doubt the relation between fluoride and the neurodevelopmental outcomes. Such scatter, however, is typical in epidemiological studies of neurotoxicants, as can be seen in the following figure from our study on lead and neurocognitive effects which the EPA relied upon as evidence of low-level lead neurotoxicity when the Agency set the national air standard for lead (Tellez-Rojoz 2006, Fig 1, reproduced as Figure D below). We also found similar scatter in our analysis of blood lead and ADHD behaviors, as measured by CRS-R (Huang 2016, Fig. 1, reproduced as Figure E below). The scatter relates to the fact that there are multiple factors that impact on intelligence and behavior; however, unless they are confounders (which we controlled for), they do not preclude the ability to focus on the specific effect of fluoride.

Figure D: Association Between Childhood Blood Lead Levels and Mental Development Index

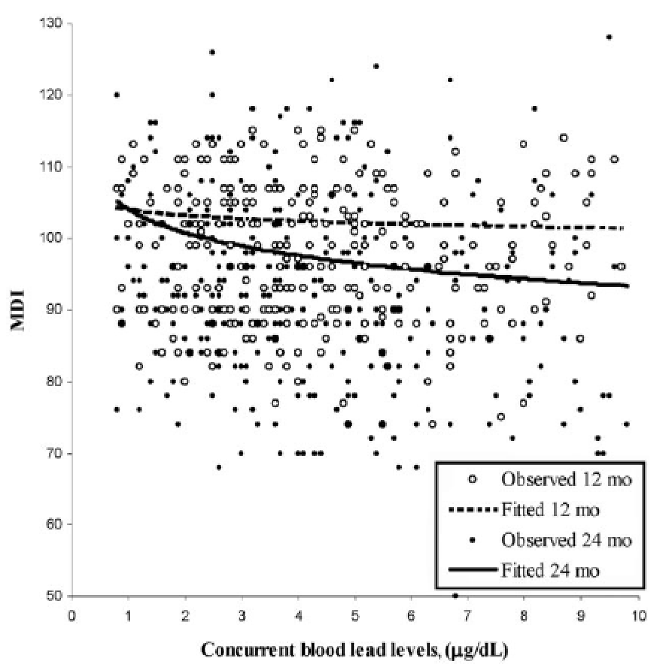
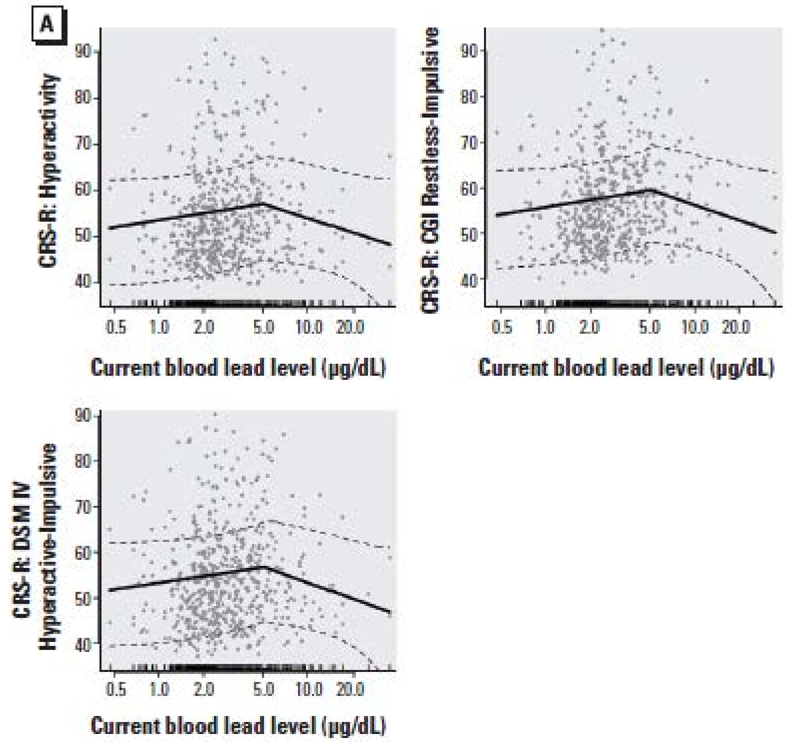


Figure E: Association Between Childhood Blood Lead Levels and ADHD Behaviors



C. The Limitations of Our Studies Do Not Provide Plausible Alternative Explanations for the Results

31. Every epidemiological study, including our studies on fluoride and neurodevelopment, has its limitations. Some of these limitations could theoretically be avoided through the use of human experiments where the toxicant is delivered to the study participants in a controlled and randomized manner. Human experimentation on neurotoxicants, however, is strictly prohibited for obvious ethical reasons. We are thus left with "observational" studies to investigate the impact of environmental toxicants on human health, with prospective cohort studies being the study design best suited for this purpose.

32. As is often the case with epidemiological studies of environmental toxicants, there are limitations in the exposure measurements that we have used in the ELEMENT studies. Our use of spot samples to collect the urine introduces some imprecision into the exposure measurement because urinary

fluoride concentrations can fluctuate somewhat throughout the day. This imprecision would have been reduced through the use of 24-hour urine collection samples, and, to a lesser extent, fasting first-morning voids, which are both considered more rigorous measures of fluoride exposure. We compensated for this limitation by using "timed" samples (i.e., second void in the morning) and adjusting for urinary creatinine. Urinary fluoride concentrations fluctuate during the day in large part because fluctuations in an individual's hydration during the day result in fluctuations in urinary dilution (and therefore, urinary concentrations). Adjusting for urinary creatinine is particularly important because the resulting measure adjusts for urinary dilution. As a result, measures of fluoride in spot urine samples adjusted for creatinine have been found to have excellent agreement with 24-hour samples (Zohouri 2006). Timed spot samples also have an important advantage over 24-hour samples in that they are less imposing on study participants. This is an important consideration when designing cohort studies because the imposition of difficult tasks like collecting 24-hour urines serve to reduce participation, which, in turn, reduces the study's sample size and statistical power.[6]

33. Another limitation in our exposure measurements is that, for most of the mothers, we did not have urine samples for every trimester. In our age 4 IQ analysis, 49%, 42%, and 9% of the mother-offspring pairs had urine samples for one, two, and all three trimesters, respectively. In the age 6-12 analysis, the respective percentages were 56%, 39%, and 5%; while in the ADHD study, the respective numbers were 57%, 36.4%, and 6.5%. It is common for prospective cohort studies to only have one or two exposure measurements during the prenatal period, but this does not introduce undue imprecision in the exposure estimate, as exposures to a toxicant have limited variation during pregnancy.

34. Importantly, the imprecision in our exposure measurements does not explain the large and significant associations we observed between maternal urinary fluoride and neurodevelopmental

[6] To put it in simple terms, less people will volunteer to participate in a study if it requires them to collect all of their urine throughout the entire day, including while they are in public spaces, such as their work environment and restaurants, etc.

effects. Imprecision in exposure measurements (of the classical or random type, as noted above) is a type of non-differential error that introduces scatter into the analysis which has a generally expected effect of biasing the results towards the null hypothesis. To put it another way, imprecisions in exposure measurements make it *harder*, not easier, to detect an association between an exposure and outcome, much like background noise makes it harder to hear a sound or signal of interest. Imprecision in the exposure measurement is thus anticipated to *obscure* an association, rather than create spurious associations where none otherwise exist. Because of this, improvement in the measurement of a particular exposure tends to reveal and strengthen associations associated with that exposure, not eliminate them.

35. Another limitation with our studies is, as with most observational studies, we could not rule out the potential for uncontrolled confounding from factors that we did not measure. For example, we did not have data on arsenic which is a neurotoxicant that has been associated with fluoride in certain rural drinking water supplies. While it always preferable to have more data than less, it is unlikely that arsenic is a meaningful confounder in our cohort. To be a confounder, a covariate must be associated with *both* the outcome *and exposure*. In our cohort, the main source of fluoride is from *salt*, not water.[7] Accordingly, even if arsenic is correlated with fluoride in rural water supplies in Mexico, this association is unlikely to be materially associated with fluoride exposures in our cohort.

36. Finally, an additional limitation in our studies is that we did not have information on the iodine status of our cohort. Iodine has been theorized to be a potential effect-modifier for fluoride's nervous system effects, i.e., deficiencies of iodine may magnify fluoride's effects, and vice versa (NRC 2006). However, failure to control for an effect modifier is unlikely to produce an association between exposure and outcome that does not otherwise exist. Moreover, in Mexico, salt is required by law to be

[7] The water in Mexico City has low levels of fluoride (i.e., 0.16 mg/L) and thus does not present a meaningful exposure in our cohort (Cantoral et al. 2019).

iodized. Fluoride levels in our cohort are thus likely correlated with *increased* iodine. To the extent that iodine modifies the effect of fluoride in our population, it is more likely to be in the direction of *reducing* toxicity rather than magnifying it.

D. Implications of Our Findings to the General Population in Water-Fluoridated Areas

37. In 2016, we published the largest characterization to date of urinary and plasma fluoride levels throughout pregnancy (Thomas, et al. 2016). At the time we published this study, there had yet to be a population-based study of fluoride exposures among pregnant women in North America, although there were two small-scale studies available from Israel and Poland. The lack of data from North America prevented us, at that time, from comparing the urinary fluoride levels in our cohort with populations from Canada or the United States.

38. Urine and plasma fluoride are metrics of the total absorbed dose of fluoride, sometimes referred to as the "internalized" or "bioavailable" dose. These internalized doses do not currently permit one to directly estimate the amount of fluoride that is ingested (i.e., the "external" exposure), nor do they permit the determination of source apportionment of the fluoride exposures. Internalized doses, however, are more relevant than external intake in predicting toxic effects, since they reflect the concentration of toxics that are being delivered to target organs in the body.

39. Our 2016 study presented the urinary and plasma fluoride levels from 825 and 330 pregnant women from our cohort, respectively. The urine samples were collected and measured using the procedures discussed above for our neurodevelopmental papers (i.e., early morning 2nd voids that were adjusted for creatinine), and both the urine and plasma samples were tested under the direction of Dr. Angeles Martinez-Mier from Indiana University, a world leader in the measurement of fluoride in biological samples.

40. The average creatinine-adjusted urinary fluoride level across all three trimesters in the

ELEMENT cohort was 0.91 mg/L, with a standard deviation of 0.4 mg/L.[8] The average plasma fluoride level across all three trimesters was 0.0221 mg/L, with a standard deviation of 0.0164 mg/L.[9]

41. By the time we published our 2018 study on ADHD behaviors, general population data had become available on maternal urinary fluoride levels in pregnancy (Till 2018). As we noted in our 2018 study, the maternal fluoride levels in the Canadian study are similar to the levels in our cohort (Bashash 2018). The mean (creatinine-adjusted) maternal urinary fluoride level among pregnant women living in the water-fluoridated areas of Canada was 0.87 mg/L, with a standard deviation of 0.50 mg/L, which is clearly in the same range as our cohort (i.e., mean = 0.91 mg/L, SD = 0.4 mg/L). The urine samples in the Canadian study were tested by Dr. Martinez-Mier using the same creatinine-adjustment method, which increases the comparability of the data.

42. The similarity in maternal urinary fluoride levels between pregnant women in the ELEMENT cohort and water-fluoridated areas of the Canadian cohort is consistent with the fact that both populations are receiving so-called "optimal" levels of fluoride through fluoridation programs (i.e., salt fluoridation in Mexico, and water fluoridation in Canada). Since salt fluoridation programs are designed to replicate the doses provided by fluoridated water, it is a reasonable, first-order expectation that populations living in salt-fluoridated and water-fluoridated areas will receive similar doses of fluoride.

43. The maternal urinary fluoride data from the ELEMENT cohort and water-fluoridated areas of Canada support the conclusions that the two populations have essentially the same *internalized* doses of fluoride. The internalized doses in water-fluoridated areas are thus in the range that we have

[8] The 75th and 90th percentile values were 1.09 mg/L and 1.37 mg/L, respectively.

[9] We tested for and found no correlation between creatinine-adjusted urinary fluoride and sociodemographic variables, including maternal age, maternal education, child sex, smoking status, birth order, and cohort. Although we found a trend towards increasing urinary fluoride levels through the first 22-23 weeks of pregnancy, and a reduction thereafter, these trends were not statistically significant (Thomas 2016).

found to be associated with substantial and significant neurodevelopmental harms in the ELEMENT cohort.

44. Although direct comparisons of *external* fluoride intake cannot yet be made, such information is not necessary to generalize the neurodevelopmental results from the ELEMENT cohort to water-fluoridated areas.

45. There is no identified reason to believe that the neurodevelopmental effects of fluoride will differ by the source of exposure, be it fluoridated salt or fluoridated water; once inside the body the source of fluoride is immaterial.

46. For the reasons stated, it is my opinion to a reasonable degree of scientific certainty that the results of the ELEMENT studies support the conclusion that fluoride is a developmental neurotoxicant at levels of internalized exposure seen in water-fluoridated communities.

I declare under penalty of perjury, under the laws of the United States, that the foregoing is true and correct to the best of my knowledge and belief.

Executed on May 20, 2020, in Seattle Washington.

HOWARD HU, MD, MPH, ScD

IV. REFERENCES

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Bashash M, Marchand M, Hu H, Till C, Martinez-Mier EA, Sanchez BN, Basu N, Peterson KE, Green R, Schnaas L et al: Prenatal fluoride exposure and attention deficit hyperactivity disorder (ADHD) symptoms in children at 6-12years of age in Mexico City. *Environ Int* 2018, 121(Pt 1):658-666.

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Téllez-Rojo MM, Bellinger DC, Arroyo-Quiroz C, Lamadrid-Figueroa H, Mercado-Garcia M, Schnaas-Arrieta L, Wright RO, Hernández-Avila M, Hu H. Longitudinal associations between blood lead concentrations lower than 10 microg/dL and neurobehavioral development in environmentally exposed children in Mexico City. *Pediatrics* 2006;118(2):e323-e330.

Thomas DB, Basu N, Martinez-Mier EA, Sanchez BN, Zhang Z, Liu Y, Parajuli RP, Peterson K, Mercado-Garcia A, Bashash M et al: Urinary and plasma fluoride levels in pregnant women from Mexico City. *Environ Res* 2016, 150:489-495.

Thomas D, Sanchez B, Peterson K, Basu N, Martinez-Mier EA, Mercado-Garcia A, Hernandez-Avia M, Till C, Bashash M, Hu H, Tellez-Rojo M. Prenatal fluoride exposure and neurobehavior among children 1-3 years of age in Mexico. *British Medical Journal* 2018; 75(Suppl 1):A10.

Till C, Green R, Grundy JG, Hornung R, Neufeld R, Martinez-Mier EA, Ayotte P, Muckle G, Lanphear B: Community Water Fluoridation and Urinary Fluoride Concentrations in a National Sample of Pregnant Women in Canada. *Environ Health Perspect* 2018, 126(10):107001.

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CURRICULUM VITAE OF
HOWARD HU, MD, MPH, ScD

CURRICULUM VITAE Date Prepared: May, 2019

NAME: Howard Hu

PRIMARY AFFILIATION: School of Public Health, University of Washington
SECONDARY AFFILIATION: School of Public Health, University of Michigan

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https://www.linkedin.com/in/howard-hu-059703a/?trk=public-profile-join-page

TABLE OF CONTENTS

EDUCATION:

9/1973-6/1976	Biology	B.Sc.	Brown University
9/1977-6/1982	Medicine	M.D.	Albert Einstein College of Medicine
9/1979-6/1980	(degree in 6/1982*)	M.P.H. (Occ Hlth)	Harvard School of Public Health
9/1985-6/1986	Epidemiology	M.S.	Harvard School of Public Health
7/1986-6/1990	Epidemiology	Sc.D.	Harvard School of Public Health

* Awarding of the Harvard M.P.H. to medical students is delayed until the M.D. degree is conferred

POSTDOCTORAL TRAINING:

Research Fellowships

7/1987-6/1988 Occupational Health Research Fellow, Dept. of Environmental Health
Harvard School of Public Health

Internship and Residencies

7/1982-6/1983	Intern in Medicine	Boston City Hospital
7/1983-6/1984	Junior Assistant Resident, Internal Medicine	Boston City Hospital
7/1984-6/1985	Senior Assistant Resident, Internal Medicine	Boston City Hospital
7/1985-6/1987	Resident, Occupational Medicine	Harvard School of Public Health

CERTIFICATION AND LICENSURE:

1984	Massachusetts Medical License Registration
1985	American Board of Internal Medicine, Diplomate
1987	American Board of Preventive Medicine, Diplomate (Occupational Medicine)
2006	Michigan Medical License Registration
2013	College of Physicians & Surgeons of Ontario
2018	Washington State Medical License Registration

ACADEMIC APPOINTMENTS:

9/1988-6/1992	Instructor in Medicine Department of Medicine, Harvard Medical School
9/1988-6/2006	Associate Physician (Clinical and Research), Channing Laboratory, Department of Medicine, Brigham & Women's Hospital
9/1990-6/1994	Assistant Professor of Occupational Medicine Department of Environmental Health, Harvard School of Public Health

7/1992-6/1997	Assistant Professor of Medicine Department of Medicine, Harvard Medical School
7/1994-6/2002	Associate Professor of Occupational Medicine Department of Environmental Health, Harvard School of Public Health
7/1997-8/2006	Associate Professor of Medicine Department of Medicine, Harvard Medical School
7/2002-8/2006	Professor of Occupational and Environmental Medicine (tenured) Department of Environmental Health, Harvard School of Public Health
9/2006-6/2012	Chair and Professor of Environmental Health Sciences (tenured), Department of Environmental Health Sciences, University of Michigan School of Public Health
9/2006-8/2009	Adjunct Professor of Occupational and Environmental Medicine Department of Environmental Health, Harvard School of Public Health
9/2006-6/2012	Research Associate Physician, Channing Laboratory, Department of Medicine, Brigham & Women's Hospital
5/2007-2012	Professor of Epidemiology, University of Michigan School of Public Health
5/2007-2012	Professor of Internal Medicine, University of Michigan Medical School
1/2009-2012	NSF International Endowed Department Chair, University of Michigan School of Public Health, Department of Environmental Health Sciences
7/2012-2018	Professor of Environmental Health, Epidemiology and Global Health (tenured) Dalla Lana School of Public Health, University of Toronto, Toronto, Ontario, Canada (on sabbatical/administrative leave, 2017-2018)
7/2012-2018	Professor, School of Medicine, University of Toronto, Toronto, Ontario, Canada
7/2012-	Adjunct Professor, Department of Environmental Health Sciences, University of Michigan School of Public Health
7/2012-2013	Director, Dalla Lana School of Public Health, University of Toronto, Toronto, Ontario, Canada
7/2013-6/2017	Founding Dean, Dalla Lana School of Public Health, a Faculty of the University of Toronto, Toronto, Ontario, Canada
7/2017-	Affiliate Professor (started as Visiting Scholar until December, 2017), Department of Occupational and Environmental Health Sciences, University of Washington School of Public Health, Seattle, WA

ADMINISTRATIVE APPOINTMENTS:

7/1991-6/2006 (Founding) Director, Metals Epidemiology Research Group, Channing Laboratory, Department of Medicine, Brigham and Women's Hospital, Harvard Medical School, and Department of Environmental Health, Harvard School of Public Health

7/1992-6/1995 Director, Commission to Investigate the Health and Environmental Effects of Nuclear Weapons Production, International Physicians for the Prevention of Nuclear War

7/1996-6/2006 Director, Residency Program in Occupational and Environmental Medicine, Harvard School of Public Health

7/1996-8/2006 Director, Occupational and Environmental Medicine Core, National Institute for Occupational Safety and Health Educational Resource Center at the Harvard School of

Public Health
7/1998-6/2004 (Founding) Medical Editor, Environmental Health Perspectives (official journal of NIEHS)
7/2000-8/2006 Associate Director, the Harvard NIEHS Environmental Sciences Center, Harvard School of Public Health
7/2004-6/2009 (Founding) Principal Investigator and Director, Harvard Center for Children's Environmental Health and Disease Prevention Research (co-PI and co-Director after 9/1/08)
9/2006-6/2012 Chair, Department of Environmental Health Sciences, University of Michigan School of Public Health
9/2006-2012 Director, Occupational Epidemiology Core, NIOSH Education and Research Center, University of Michigan
9/2006-2012 Co-Director, Michigan-Harvard/Harvard-Michigan Metals Epidemiology Research Group
7/2009-2011 Director, NIA T32 Training Grant in Aging and Public Health, University of Michigan School of Public Health
1/2010-2012 Chair, Faculty Steering Committee on Global Health, University of Michigan School of Public Health
4/2011-2012 (Founding PI) and Director, University of Michigan NIEHS P30 Core Center.
7/2012-2013 Director, Dalla Lana School of Public Health, University of Toronto, Toronto, Ontario, Canada
7/2013-6/2017 Founding Dean, Dalla Lana School of Public Health, a Faculty of the University of Toronto, Toronto, Ontario, Canada

CLINICAL APPOINTMENTS:

7/1985-6/1987 Attending Physician, Emergency Department, Whidden Memorial Hospital
7/1985-6/1988 Assistant Visiting Physician, Department of Medicine, Boston City Hospital
1/1985-6/2006 Consultant in Occupational and Environmental Medicine, Center for Occupational and Environmental Medicine, Northeast Specialty Hospital (formerly known as the Olympus Specialty Hospital, the Massachusetts Respiratory Hospital, and Norfolk County Hospital).
3/1987-9/1987 Attending Physician, Occupational Health Program, University Hospital/Boston University Medical Center
7/1988-9/2006 Associate Physician, Brigham and Women's Hospital
7/1990-6/1995 Occupational/Environmental Medicine Consultant, Brigham and Women's Hospital Employee Health Services
7/2007-2012 Associate Physician, Division of General Medicine, Department of Medicine, University of Michigan Health System
1/2019-present Staff Physician, RotaClinic-Lake City, Seattle, WA

OTHER ACADEMIC POSITIONS and MAJOR VISITING APPOINTMENTS:

7/1987-6/1990 Visiting Physician, South Cove Health Center, Boston (Chinatown)
7/1996-8/2006 Associate, Center for Health and the Global Environment, Harvard Medical School
2/1997 Alice Hamilton Visiting Professor, Division of Occupational and Environmental Medicine, Department of Medicine, University of California at San Francisco
11/2000- Visiting Scientist, Sri Ramachandra Medical College and Research Institute
7/2010- Senior Consultant, Tianjin Centers for Disease Control and Prevention, Tianjin, China
10/2012- Visiting Professor, Shanghai Key Laboratory of Children's Environmental Health, Xinhua Hospital, Shanghai Jiao-Tung University, China
7/2013-6/2016 Visiting Professor, Shanghai Jiao Tong School of Medicine, China
5/2015- Affiliate Scientist to the Li Ka Shing Knowledge Institute, St. Michael's Hospital, Toronto, Canada

MAJOR RESEARCH INTERESTS:

1. Environmental and molecular epidemiologic research related to heavy metals, potential endocrine disruptors, other neurotoxicants, carcinogens, etc.
2. Gene-environment interactions; epigenetic dysregulation
3. Fetal/early life exposures and long-term effects
4. Aging-environment interactions
5. Environmental health, health inequities and health disparities, human rights
6. Health and the global environment
7. "Big Data" for population health
8. Environmental sensitivities/Multiple chemical sensitivities

GRANTS (as PI, Co-PI, or primary mentor only):

Past Funding:

1980 (summer) Montefiore Hospital, Bronx NY,PI; $2,000 (approx)
A study of rural and occupational health in Tulua, Colombia, South America
1982 (summer) Albert Einstein College of Medicine, PI; $3,000 (approx)
A study of occupational/environmental health in Shanghai, China
7/1987-6/1989 NIEHS Center Grant ES00002 Pilot Project, PI; $12,000
The Long-term Renal and Neurologic Effects of Childhood Plumbism
7/1989-6/1990 NIEHS subcontract 7083-1, PI; $50,000 (approx)
The Use of X-Ray Fluorescence to Measure Lead Burden and Childhood Lead Exposure
7/1990-6/1992 Agency for Toxic Substances and Disease Registry, PI; $150,000 (approx)
"Clinical Environmental/ Occupational Medicine Research Fellowship Award",
7/1990-6/1991 NIEHS Center Grant ES00002 Pilot Project, PI; $12,000
The Metabolic Effects of Pregnancy and Lactation on Lead Burden

7/1990-6/1991 Harvard School of Public Health Basic, PI
Research Support Grant; $10,000
K-X-Ray Fluorescence Measured Lead Burden
10/1991-11/1991 NIOSH Special Grants, PI; $50,000 (approx)
The Carpenters Lead Project
4/1991-3/1996 NIEHS/R01, PI; $2,200,000 (approx)
The Epidemiology of Lead, Diet and Blood Pressure
7/1991-6/1996 NIEHS/R01 supplement, PI; $240,000 (approx)
The Epidemiology of Lead, Diet and Blood
Pressure--Research Supplement for Minority Investigator
7/1992-6/1995 NIEHS/R01 (Office of Research on Women), PI; $200,000 (approx)
Lead and Hypertension in Women
7/1993-6/1996 NIEHS/subcontract, PI; $150,000 (approx)
Exposure to Neurotoxins as Risk Factors for Amyotrophic Lateral Sclerosis
7/1995-6/1998 State of Washington, Department of Labor, PI; $350,000 (approx)
SPECT Imaging of the Brain in Patients with Multiple Chemical Sensitivity Syndrome and Controls
7/1996-6/1997 NIEHS Center Grant ES00002 Pilot Project, PI; $15,000
Electrocardiographic abnormalities in association with low-level lead exposure among middle-aged to elderly men: the Normative Aging Study
4/1995-3/2000 NIEHS Project PI (Program Project PI: Richard Monson); $1,800,000 (approx)
Lead Exposure, Accumulation in Bone, and Reproductive Toxicity Among Men and Women In Mexico
4/1995-3/2000 NIEHS Project PI (Program Project PI: Richard Monson); $1,900,000 (approx)
Lead Exposure, Accumulation in Bone, and Cognitive Toxicity Among Elderly Men and Women
6/1997-5/2002 NIEHS/R01 ES05257 PI; $2,312,274
Lead Biomarkers, Aging, and Chronic Disease
7/1997-6/1999 NIEHS Center Grant ES00002 Pilot Project, PI; $10,000
The effect of genetic polymorphisms of metallothionein-IIA on mRNA levels in middle-aged to elderly men: the Normative Aging Study
7/1998-6/2003 NIEHS/R01 PI (with no-cost extension; 5R01ES007821); $2,291,833
Lead Dose Biomarkers, Reproduction, and Infant Outcomes
7/1999-6/2000 NIEHS Center Grant ES00002 Pilot Project, co-PI; $14,000
Magnetic Resonance Spectroscopy in the Evaluation of Lead Neurotoxicity: the Normative Aging Study
7/2000-6/2001 MAVERIC (Massachusetts Area Veterans Epidemiology Resource and Institute Center) Pilot Project PI (with Dr. Robert Wright, co-PI); $10,000
The Use of Magnetic Resonance Spectroscopy in Lead Poisoning
7/2000-6/2001 NIOSH Center Grant Pilot Project, PI (with Dr. Robert Wright, co-PI); $12,000
Interaction between ApoE Genotype and Lead Exposure in the Development of Cognitive Impairment
7/2002-6/2004 The Rasmussen Foundation/Health Care Without Harm; $50,000

Medical Use of Phthalate Containing Products in the Neonatal Intensive Care Unit and Biomarkers of Neonatal Phthalate Metabolites

7/2002-6/2003 NIEHS Center Grant Pilot Project, PI; $8,000
Vitamin D Receptor Gene and Bone Lead in Reproduction

3/2004-2/2005 The Critelli Family Foundation; $10,000
Review of Environmental Cadmium Exposure and Toxicity

4/2000-3/2007 NIEHS Project Leader (Program Project PI: Richard Monson; 5P01ES05947); $2,472,677; Controlled Trial in Pregnancy of Dietary Supplements for the Suppression of Bone Resorption and Mobilization of Lead into Plasma (no cost extension)

4/2000-3/2007 NIEHS Project co-Leader (Program Project PI: Richard Monson; 5P01ES05947); $1,210,000 (approx); A Community-Based Study of Lead Exposure Pathways, Biomarkers of Dose, Health Effects, and Phytoremediation Strategies at the Tar Creek Superfund Site (no cst extension)

4/2002-9/2007 NIEHS/R01 PI (5R01ES010798); $3,011,295
Gene-Metal Interactions and Parkinson's Disease

10/2003-9/2007 NCMHI/P20 Project Leader (MD000501-01; Hughes Harris, PI); $828,781(Project)
"FAMU and Harvard Center for Health and Health Care Disparities"

8/2003-7/2008 NIEHS/R01 PI (2R01ES05257-11A2); $3,357,424 (bécame co-PI in 2007 after move to University of Michigan)
Lead-Gene Interactions and Cognition

6/2004-3/2009 NIEHS/P01 PI (5 P01ES012874-01); $6,662,670 (became co-PI in 2006 after move to University of Michigan)
Metals Mixtures and Children's Health (Center for Children's Environmental Health and Disease Prevention Research)

7/2002-12/2009 NIH/R03 PI (1R03TW005914; no cost ext through 2008); $192,000 (approx)
Lead, Genes, and Cognition in Children in Chennai, India

9/2006-7/2011 NIEHS/R01 PI (R01ES0007821); $3,116,831
Fetal Origins of Neurobehavior: Lead and Cholesterol Metabolism Interactions

7/2006-6/2011 NIEHS/R01 co-PI (R01ES013744; PI Wright), $3,200,000
Stress, Lead, Iron Deficiency and Neurodevelopment

7/2006-6/2011 NIEHS/R01 co-PI (RO1ES014930; PI Wright), $2,800,000
Metal Mixtures and Neurodevelopment

2/2008-2/2010 Michigan Institute for Clinical and Health Research (MICHR; home of the UM CTSA; UL1RR024986) Pilot Project PI; $26,000 (no cost extension)
Epigenetics of Early Life Events and Environmental Toxicants

4/2009-4/2010 Michigan Alzheimer's Disease Research Center Pilot Project PI, $25,000
Environment, Epigenetics and Alzheimer's Disease (no cost extension)

12/2009-12/2010 University of Michigan Center for Global Health Pilot Project PI, $25,000
Climate Variability and Impacts on Mortality and Morbidity in Chennai, India: A Pilot Project Stemming from the 2009 U.S.-India Workshop on Climate Change and Public Health, Goa India (no cost extension)

9/2009-9/2010 Michigan Institute for Clinical and Health Research (MICHR; home of the UM CTSA; UL1RR024986) Pilot Project PI; $26,000 (no cost extension) Epigenetics and Epigenomics in the Etiology of Alzheimer Disease

7/2008-6/2011 NIA/T32 PI (T32AG027708); $450,000 Interdisciplinary Training Program in Aging and Public Health

4/2010-3/2015 NIEHS P42 Superfund Co-Inv, Project 2, Co-investigator (P42ES017198; PI: Alshawabkeh, Project 2 Leader: Meeker) Puerto Rico Testsite For Exploring Contaminant Threats, $12,000,000

4/1/2011-6/2015 NIEHS Core Environmental Health Sciences Center, Founding PI and Director (until 2012; now consultant; P30 ES017885), $ 4,620,100; "Lifestage Exposures and Adult Disease"

4/2010-3/2014 NIEHS/EPA P20 Co-PI and Clin Health Specialist (P20 ES018171; PI Peterson) Formative Children's Environmental Health and Disease Prevention Center, $1,959,960; "Perinatal Exposures, Epigenetics, Child Obesity & Sexual Maturation"

7/1/2013-6/30/2014 CIHR, Canadian Institute for Health Services and Policy Research; Planning Grants-Priority Announcement:Partnerships for Health System Improvement; PI, $24,992 "The Surviving Opioid Overdose with Naloxone (SOON) Project and Roundtable"

07/1/11-06/30/16 NIEHS K01 ES019909 (co-mentor; PI: Somers) "Immune dysfunction associated with early life heavy metal exposure"

4/1/12-3/30/17 NIEHS R01ES013744 (consultant; PI: Wright; Mt Sinai School of Medicine) "Stress-Lead Interactions and Child Development"

7/1/2012-7/1/2017 European Commission (EC), Funded under FP7-Health, Project 304925, co-Investigator; PI, epidemiologic studies, $6,000,000 E "A novel micronutrient-based strategy to prevent hearing impairments: test and road to market for age-related hearing loss and preservation of residual hearing"

Current Funding

6/1/2012-7/1/2019 1R01ES021446-01, PI, $4,140,000 (parent + supplement awards); "Prenatal and Childhood Exposure to Fluoride and Neurodevelopment"

5/15/2015-5/15/2019 Health Canada; PI, $200,000 (Phase 1); $1,400,000 (proposed Phase 2) "A Community-based First Nation Study of Cancer and the Environment in Northern Ontario"

4/1/13-3/31/23 NIEHS/EPA P01ES022844 (co-inv; PI: Peterson at the University of Michigan) "Lifecourse Exposures & Diet: Epigenetics, Maturation & Metabolic Syndrome."

7/1/16-6/30/21 CIHR (co-PI; Director; PI: Jeffrey Brook at the Dalla Lana School of Public Health) $4,700,000 CNDN "CANadian Urban Environmental (CANUE) Health Research Consortium"

9/1/16-8/31/21 NIH 5R01ES026033-02, (consultant; PI: Arora at Mt. Sinai School of Medicine) $648,000 "Novel Biomarker to Identify Critical Windows of Susceptibility to Metal Mixture"

Applications Under Review

Wellcome Trust, co-investigator (PI: P Landrigan)
"Quantifying the Cognitive and Economic Benefits of Reducing Air Pollution to Achieve Climate Change Mitigation"

Competitive Renewal Application In Progress

R01ES021446-01, PI, $4,140,000
A Prospective Study of Early Life Exposure to Fluoride, Thyroid Function, and Neurobehavioral Outcomes

Amended Application In Progress

R01ES007821-11, PI, $4,800,000;
Early Life Toxicants and Cardiovascular Outcomes" (priority score 27 by CASE Study section; 25th percentile)

New Application in Progress

Wellcome Trust, xxx, multiple-PI
Addressing Two Critical Gaps in Understanding the Impacts of Lead Exposure on the Global Burden of Disease: (a) Impacts on Cardiovascular Disease; (b) Exposures and Sources in Low and Middle-Income Countries

HONORS AND AWARDS:

1978-1982 National Health Service Corps Scholarship
1985-1988 National Research Service Award
1990-1992 Agency for Toxic Substances and Disease Registry Clinical Environmental Medicine Award
1994 Will Solimene Award of Excellence, American Medical Writers Association, for: Chivian E, McCally M, Hu H, Haines H, eds. *Critical Condition: Human Health and the Environment*. Cambridge: The MIT Press, 1993.
1997 Alice Hamilton Lecturer, University of California at San Francisco
1998 First Prize for Best Infant Nutrition Research, Instituto Danone, Mexico (for González-Cossío T, Peterson KE, Sanín L, Fishbein SE, Palazuelos E, Aro A, Hernández-Avila M, Hu H. "Decrease in birth weight in relation to maternal bone lead burden." Published in *Pediatrics*)
1999 National Institute for Environmental Health Sciences "Progress and Achievement of the

Year Award", 1998-1999
1999 True Memorial Lecturer, Maine Medical Center, Portland ME.
2000-2001 Faculty Sabbatical Award, Harvard School of Public Health
2000-2001 Senior Fulbright Scholar in India
2001 Hoopes Prize, Faculty Mentorship (for Senior Thesis of Charles Lin, "More than Black and White: Lead Poisoning as an Environmental Justice Issue in Boston")
2003 Best Paper in Preventive Medicine by a Medical Student (for Senior Thesis of Vanitha Janakiraman; Janakiraman V, Hu H, Mercado-Garcia A, Hernandez-Avila M. A randomized crossover trial of nocturnal calcium supplements to suppress bone resorption during pregnancy. Am J Prev Med 2003;24:260-4.). American College of Preventive Medicine, Ulrich and Ruth Frank Foundation for International Health.
2004 Das Travel Grant Award, The South Asia Initiative, Harvard University (for Travel in India)
2005 Adolph G. Kammer Merit in Authorship Award, the American College of Occupational and Environmental Medicine (for Rhodes D, Spiro A, Aro A, Hu H "Relationship of Bone and Blood Lead Levels to Psychiatric Symptoms: The Normative Aging Study", Published in the *Journal of Occupational and Environmental Medicine*)
2006 Teacher of the Year Award, Occupational/Environmental Medicine Residents, Harvard School of Public Health
2006 Harriett Hardy Award, the New England College of Occupational and Environmental Medicine
2009 Linus Pauling Award for Lifetime Achievements, American College for the Advancement of Medicine
2011 Award for Excellence, American Public Health Association
2015 John R. Goldsmith Award for Outstanding Contributions to Environmental Epidemiology, International Society for Environmental Epidemiology
2016 Election to Fellowship, Canadian Academy of Health Sciences

MEMBERSHIPS IN PROFESSIONAL SOCIETIES

Memberships

1981- American Public Health Association (APHA)
1982-2006 Massachusetts Coalition for Occupational Safety and Health
1983-1989 American College of Physicians
1985- Physicians for Social Responsibility
1987- Physicians for Human Rights
1990- International Society for Environmental Epidemiology (ISEE)
1990-2000 American Association for the Advancement of Science
1990-2006 Association of Occupational and Environmental Clinics (AOEC)
1991- International Physicians for the Prevention of Nuclear War (IPPNW)

1994-1996 Society for Occupational and Environmental Health (SOEH)
2000-2012 American College of Occupational and Environmental Medicine (ACOEM)
2009-2012 Society of Toxicology
2012- Canadian Public Health Association (CPHA)

Committee Assignments

1981-1982 Program Committee, Occupational Safety and Health Section, APHA
1987-1988 Program Committee, Asian-American Caucus, APHA
1992-1998 Membership Committee, ISEE
1995-1998 Quality Assurance Committee, AOEC
1997-1998 Program Committee, 1998 Superfund Basic Research Program, Annual National Meeting
2001-2006 Program Committee, New England College of Occupational and Environmental Medicine Annual Meetings

EDITORIAL POSITIONS AND BOARDS:

1977-1982 Einstein Community Health Newsletter
1988-1992 Bookreview Co-Editor, Section on Occupational Safety and Health, Am Public Health Assoc.
1993- Journal of Health and Human Rights
1998- Environmental Health Perspectives (Founding Medical Editor, 1998-2004; Associated Editor, 2004-)
2004- American Journal of Industrial Medicine
2007-2009 Faculty of 1000 Medicine
2017- Current Environmental Health Reports
2017- Faculty of 1000 Medicine

PEER REVIEW SERVICE

American Journal of Epidemiology
American Journal of Industrial Medicine
Archives of Environmental and Occupational Health
Biomed Central
Circulation
Environmental Health
Environmental Health Perspectives
Environment International
Environmental Research
Epidemiology

Indian Journal of Medical Research
Journal of Health and Human Rights
Journal of the American Medical Association
Kidney International
Lancet
New England Journal of Medicine
Pediatrics
PLOS One
Science of the Total Environment

TEACHING:

1. LOCAL CONTRIBUTIONS (at the Harvard School of Public Health, 1985-2006)

1985-	"Toxicology of the Kidney and Urinary Tract" Guest Lecturer for TOX204a,b
1988-	"Occupational Health" Guest Lecturer for EH201a,b
1989-1992	"Lead Toxicology" Guest Lecturer for TOX204a,b
1990-	<u>Grand Rounds in Occupational/Environmental Medicine</u> Director
1990-2000	<u>Introduction to Occupational and Environmental Medicine (EH232c,d)</u> Course director, lecturer
1990-	"The Epidemiology of Lead Exposure, Dose, and Toxicity" Guest Lecturer for EPE215c,d and EPE215t
1990-	"Solvent toxicity" Fundamentals of Industrial Hygiene, Continuing Education Department
1992	"Current Research on Lead", Metals Epidemiology Research Group Seminar Presenter
1992	"Lead Poisoning Without a Known Source in a Hyperthyroid Patient" Case discussant, Grand Rounds in Occupational and Environmental Medicine
1992-	"Biological Markers of Lead Dose" Guest Lecturer, EHE280c,d
1994-	"Screening for Lead Toxicity" Guest lecturer, EPI227d
1994-	"Lead Exposure and Biological Monitoring" Guest Lecturer, ID263b
1994-	"Case Study: Lead" Guest Lecturer and Case Discussant, EH202d
1996-	<u>Introduction to Environmental Health (EH201b)</u>

	Course director and lecturer
1997-	Human Health and Global Environment Change (EH278a,b)
	Course Co-developer, Co-director, and lecturer

Hospital courses and Invited Teaching Presentations (Harvard-affiliated Hospitals)

1990	Guest Lecturer on Occupational Medicine Residency Program, Department of Medicine, Brigham and Women's Hospital
1994	Speaker, Grand Rounds; "Is Lead a Ticking Time Bomb?" Department of Obstetrics and Gynecology, Brigham and Women's Hospital
1994	Speaker, Grand Rounds; "Is Lead a Ticking Time Bomb?" Department of Medicine, Brockton V.A. Hospital
1994	Speaker, Symposium on Preventive Medicine and Clinical Epidemiology,; "Is Lead a Ticking Time Bomb"; Brigham and Women's Hospital
1995	Discussant, "Multiple Chemical Sensitivity", Occupational/Environmental Medicine Grand Rounds, Occupational Health Program, Harvard School of Public Health
1996	Guest lecturer, "Lead Toxicity as a Paradigm for a Regional and Global Health Hazard", Environmental Health Student Group, Holmes Society, Harvard Medical School
1997	Speaker, "Mobilization of maternal bone lead as a hazard to the fetus", Grand Rounds, Dept. of Neonatology, Beth Israel Hospital, Boston, MA
2000	Guest lecturer, "Update on Lead Toxicity Research", Program in Pediatric Toxicology, Children's Hospital
2000	Discussant, "Adult Lead Toxicity", Weekly Case Round, Department of Medicine, Brigham and Women's Hospital, Boston.
2000	Lecturer, "Update on Lead Toxicity, Hypertension, and Chronic Renal Failure", Renal Rounds, Division of Nephrology, Department of Medicine, Brigham and Women's Hospital, Boston.
2002	Lecturer, "Maternal Bone Lead as a Threat to Fetal Development", Program in Neonatology, Beth Israel-Deaconness Hospital, Boston, MA

Doctoral student committees
Chair and member:

Dr. Rokho Kim	Dr.P.H. Occupational Health and Epidemiology, '96
Dr. Yawen Cheng	Sc.D. Epidemiology, '98
Dr. Sharon Tsaih	Sc.D. Epidemiology, '99
Dr. Hung Yi Chuang	Sc.D. Occupational Health, '99
Dr. Adrienne Ettinger	Sc.D. Environmental Health, '03
Dr. Florence Wang	Sc.D. Environmental Health, '05
Dr. Sung K. Park	Sc.D. Environmental Health, '05
Dr. Pradeep Rajan,	Sc.D. Occupational Health, '06

Member/Advisor:

Dr. How Ran Guo	Sc.D. Occupational Health, ‘94
Dr. Joshua Cohen	Sc.D. Health Policy and Management, ‘94
Dr. Jane Hoppin	Sc.D. Environmental Health, ‘95
Dr. Salma Elreedy	Sc.D. Environmental Health, ‘97
Dr. Mary Jean Brown	Sc.D. Maternal and Child Health, ‘00
Dr. Brisa Sanchez	Sc.D. Biostatistics, '06
Dr. Ami Zota	Sc.D. Environmental Health, ‘07
Dr. Ananya Roy	Sc.D. Environmental Health, '08
Dr. Elissa Wilker	Sc.D. Environmental Health, '09

Post-doctoral fellow mentor:

Dr. Marinelle Payton (Channing Lab), Dr. Susan Korrick (Channing Lab), Dr. Rokho Kim (Channing Lab), Dr. Viji Potula (HSPH Research Fellow), Dr. Barbara Nowak (Visiting Scientist from Silesian University School of Medicine, Poland), Dr. Robert Wright (Channing Lab), Dr. Ming Tsuang Wu (HSPH Research Fellow), Dr. Yawen Cheng (Channing Lab), Dr. Geeta Mathur (neonatology fellow at the Brigham and Women’s Hospital), Dr. Sri Hari Bojja (HSPH Research Fellow), Dr. Hae-Kwan Cheong (Visiting Scientist from Dongguk University School of Medicine, S. Korea), Dr. Sahar Elmarsafawy (HSPH Research Fellow), Dr. Jing Lu (Visiting Scientist from the Chinese Academy of Preventive Medicine), Dr. Dieter Affeln (Occ/Env Med Fellow), Dr. Ahmed Gomaa (Occ/Env Med Fellow), Dr. Chris Leffler (Occ/Env Med Fellow), Dr. Ronald Dykeman (Occ/Env Med Fellow), Dr. Uma Dhanabalan (Occ/Env Med Fellow), Dr. Hsien-Wen Hsu (Occ/Env Med Fellow), Dr. Betty Ann Cohen (Occ/Env Med Fellow), Dr. Arvin Chin (Occ/Env Med Fellow), Dr. Daniel Rhodes (Occ/Env Med Fellow), Dr. Richard Wittman (Occ/Env Med Fellow), Dr. Sun-Dong Lee (Visiting Scientist from Sangji University, Korea), Dr. Ronald Green (Occ/Env Med Fellow), Dr. Erma Lawson (Environmental Health Fellow), Dr. Marc Weisskopf (Environmental Health Fellow), Dr. Bridget Bagert (Occ/Env Med Fellow), Dr. John Jarrell (Visiting Scientist from University of Calgary), Dr. Jennifer Weuve (Environmental Health Fellow), Dr. Karen Chou (Visiting Scientist from Michigan State), Dr. Nitin Jain (Channing Laboratory Fellow), Dr. Adrienne Ettinger (Children’s Center Scientist), Dr. Sam Myers (Fellow in Alternative and Complementary Medicine), Dr. Marcelo Targino (Occ/Env Med Fellow), Dr. Manish Arora (Post-doctoral fellow from University of Sydney), Dr. Huiling Nie (Post-doctoral fellow from McMaster University).

Other faculty mentorship:

Elizabeth Rubinstein (HMS Summer research), Alicia Marier (HMS Summer research), Vanitha Janakiraman (HMS Summer research), Young-Sook Lim (Harvard College Summer research), Charles Lin (Harvard College Senior thesis research), Ed Hsieh (Harvard College Summer research), Naveen Thomas (Emory University Medical School Senior thesis research). Shreekrishna Akilesh (Harvard Dental School summer research), Christine Pace (HMS Summer research)

Advisory and supervisory responsibilities

1985-1987	Attending Physician, outpatient general medicine clinic, Boston City Hospital; weekly precepting for housestaff and medical students

1990-2006	Preceptor, Residency in Occupational and Environmental Medicine, Harvard School of Public Health at the Mass Respiratory Hospital
1990-2006	Advisor to general M.P.H. students, Harvard School of Public Health.

2. LOCAL CONTRIBUTIONS (at the University of Michigan, 2006-2012)

2006-	Principles of Environmental Health (EHS-500) Course director and lecturer
2006-	Environmental Epidemiology (EHS-608) Guest lecturer on birth cohorts and environmental epidemiology
2006-	Occupational and Environmental Disease (EHS-501) Guest lecturer on metals exposure and health effects; Course Director (2009-)
2007-	Metals Exposure, Biomarkers and Toxicity: A Multi-disciplinary Environmental Epidemiology Approach (EHS-698 reading course) Course director and lecturer
2008-2009, 2010-2011	Topics in Environmental Health Sciences (EHS-688) Course director and lecturer
2009	Occupational and Environmental Disease (EHS-501) Course director and lecturer
2009-	On-line (Long-distance Foundations in Public Health Certificate Program): Principles of Environmental Health (EHS-500-801) Course director and lecturer
2009	Introduction to Public Health (HMP-200) Guest lecturer on environmental health
2009-	Seminars in Aging and Public Health (EPID 813) Course director and lecturer
2011	Seminar on Public Health in China (HMP-xxx) Guest lecturer on “Environmental Health in China”

Post-doctoral fellow mentor:

Dr. Sung Kyun Park (Environmental Health Sciences Fellow, now Research Assistant Professor), Dr. Brisa Sanchez (Biostats Research Assistant Professor, now Assistant Professor), Dr. Richard Pilsner (Robert Wood Johnson Health & Society Fellow), Dr. Aimin Zhang (Environmental Health Sciences Fellow, Toxicology Training Grant), Dr. Ananya Roy (Environmental Health Sciences Fellow), Dr. David Cantonwine (Reproductive Sciences Fellow).

Doctoral Student Advisor (principal)

David Cantonwine	Ph.D.	Environmental Health Sciences (2009)
Myriam Afeiche	Ph.D.	Environmental Health Sciences (co-mentor with Karen Peterson; 2010)
Yoon-Hyeong Choi	Ph.D.	Environmental Health Sciences (co-mentor with Sung Kyun Park; 2011)

Katie F. Bush	Ph.D.	Environmental Health Sciences (co-mentor with Marie O'Neill; 2011)
Kelly Bakulski	Ph.D.	Environmental Health Sciences (2012)
Gamola Fortenberry	Ph.D.	Environmental Health Sciences (co-mentor with John Meeker; 2013)
Siying Huang	Ph.D.	Environmental Health Sciences (2013)
Deena Thomas	Ph.D.	Environmental Health Sciences (2014)
Rebecca Tutino	Ph.D.	Environmental Health Sciences (2015)
Zishaan Farooqui	Ph.D.	MD-PhD Medical Scientist Training Program (2015)

Masters Student Thesis Advisor

Bradley Lampe (OEE), Troy Meissner (OEE), Pheba Alexander (OEE), Brian Davis (OEE & HBHE), Aaron Leftwich (OJOC program), Suengwon Lee (Nutrition), Allen Zhong (OEE), Graham Newman (OEE), Jacqueline Barkoski (OEE)

Undergraduate Thesis Advisor

Lauren Schwartz (Neuroscience, LSA)

3. LOCAL CONTRIBUTIONS (at the University of Toronto, 2012-present)

2012	Determinants of Community Health (Faculty of Medicine) Guest lecturer on 'The Future of Medicine & Public Health in a Crowded, Diverse, Aging, Stratified, Urbanized, Polluted, Hot, Thirsty, Hungry, Debt-Ridden World".
2012-	CHL5004H Introduction to Public Health Guest lecturer on "The Future of Public Health (and Your Role !) in a Hot, Flat, Crowded…and Diverse, Aging, Stratified, Urbanized, Polluted, Thirsty, Hungry, Debt-Ridden World". "What is Public Health?", "Climate Change and Health"
2012-	CHL 5912F Industrial Toxicology. Guest lecturer on the "Toxicology of Metals".
2013-2014	Department of Family & Community Medicine "Building Blocks" (short course for International post-graduate primary care trainees); Guest lecturer on "Public Health & Primary Care"
2013-	CHL5701H Doctoral Seminar, Collaborative Doctoral Program in Global Health Guest lecturer on "The Challenges of Environmental Health in a Rapidly-Changing World, from the Molecular to the Global".
2014	JCR1000 "Interdisciplinary Approach to Global Challenges" Guest lecturer on "Global Environmental Health"
2014-	PHS100H1 "Grand Opportunities in Global Health"; Guest lecturer on "Urban Environments"
2015	Public Health & Preventive Medicine Residency Rounds "Physicians, Climate, and other Global Environmental Changes: Our Role"
2016	CHL5004H Introduction to Public Health, Course Co-Director (with Professor Erica

DiRuggierro)
2016 CHL 7001H F6 Environmental-Molecular Epidemiology, Course Co-Moderator (with Professor Morteza Bashash)
2016 CHL5701H Doctoral Seminar, Collaborative Doctoral Program in Global Health, Course Co-Director (with Professors Erica DiRuggierro and Abdallah Daar)
2016 Joint Seminar, "The Impact on Intelligence, Behaviour, and Society of Lead Exposure: A Case Study of a Global Pollutant and On-going Research"; Collaborative Program in Neurosciences and Collaborative Global Health Doctoral Program, University of Toronto
2016 CHL5420H "Global Health Research Methods"
Guest lecturer on "The Early Life Exposures in Mexico to Environmental Toxicants Project (ELEMENT): A Global Health Collaboration Case Study"

Masters student research advisor
Maelle Marchand

Doctoral student advisor
Adele Carty

Doctoral student thesis committee member
Laura Bogaert

Doctoral student thesis examination committee member
Claudie CY Wong (doctoral student in epidemiology, Jockey School of Public Health and Primary Care, Chinese University of Hong Kong)
Zilong Zhang (doctoral student in epidemiology, Jockey School of Public Health and Primary Care, Chinese University of Hong Kong)

Post-doctoral fellow mentor:
Siying Huang, Ph.D.; Morteza Bashash, Ph.D.; Roman Pabayo, Sc.D. (Harvard School of Public Health); Tripler Pell, M.D., M.P.H.

4. LOCAL CONTRIBUTIONS (at the University of Washington, 2017-present)

Doctoral student thesis research mentor
Megan Suter

Doctoral student special projects advisor
Rachel Shaffer
Joey Frostad
Rebecca De Buen

5. NIH K-grant mentorship:

Robert Wright, M.D., M.P.H. (K-23 ES000381, "*Neurochemical and Genetic Markers of Lead Toxicity"*), 2000-2005; Dr. Wright is now Prof of Pediatrics, Mt. Sinai School of Medicine
Marc Weisskopf, Ph.D. (K-01 ES012653, "*New Biomarkers of Neurotoxicity*"), 2004-2009; Dr. Weisskopf is now Associate Prof of Occup Health, Harvard Sch Public Health
Sung Kyun Park, Sc.D. (K-01 ES016587; "*Environment, Novel Aging Outcomes, and Genetics*"), 2009-2014; Dr. Park is now Assistant Prof, Department of Epidemiology, University of Michigan Sch Public Health
Emily Somers, Ph.D. (K-01 ES019909; *"Immune Dysfunction Associated with Early Life Heavy Metals Exposure"*), 2011-2016; Dr. Somers is now Associate Prof, Division of Rheumatology, Department of Internal Medicine, University of Michigan Medical School

COMMITTEE, ORGANIZATIONAL, AND VOLUNTEER SERVICE

National/International

1978-1982 Taskforce on Occupational and Environmental Health, Co-coordinator, Am Med Stu Assoc
1989 Ad Hoc Study Committee, National Institute for Environmental Health Sciences Council
1989-2006 Association of Occupational and Environmental Medicine Clinics (AOEC)-- (through the Northeast Specialty Hospital Center for Occupational and Environmental Medicine)
1989-1990 Member, Relative Risk Reduction Strategies Committee, Science Advisory Board, U.S. Environmental Protection Agency
1989-1992 Member, Board of Directors, Physicians for Human Rights, Boston, MA
1991 National Institutes of Health, General Clinical Research Center Program, Site Visit Team
1992- Member, National Advisory Committee, Physicians for Human Rights, Boston, MA
1992 Special Study Section member (R3/S1/B3), National Institutes of Health
1994 Ad Hoc Reviewer, National Institutes of Health, General Dental Research Center Program
1994- Advisory Board, Institute for Energy and Environmental Research
1994-1996 Associate, Project on Global Environmental Change and Health, Physicians for Social Responsibility
1995 Ad Hoc Reviewer, National Institutes of Health, Diagnostic Radiology Study Section
1996- Membor, Editorial Board, Health and Human Rights—an International Journal
1995-1998 Advisory Committee, Consortium for Environmental Education in Medicine, Cambridge, MA.
1996-1997 Reviewer, Agency for Toxic Substances and Disease Registry
1997-1998 Program Committee, Annual Mtg, NIEHS Superfund Basic Research Group Centers
1998-2013 (Founding) Medical Editor (1998-2004); Associated Medical Editor (2004-), Environmental Health Perspectives (official journal of NIEHS)
2001 Ad Hoc Reviewer, National Institutes of Health, R-13 applications

2002-2006 External Advisory Committee, Program Project on Lead and Osteoporosis, University of Rochester
2003-2005 Member, Ad-Hoc Expert Panel to Form Medical Management Guidelines for Lead-Exposed Adults, (supported by NIOSH and AOEC)
2003-2009 Member, Working Group on Lead and Pregnancy, Advisory Committee on Childhood Lead Poisoning Prevention, U.S. Centers for Disease Control and Prevention
2004 Ad Hoc Reviewer, National Institutes of Health, K-23 applications
2004 Ad Hoc Reviewer, Draft of "Immunization Safety Review: Vaccines and Autism" Immunization Safety Review Committee, Institute of Medicine, National Academies of Science
2004 Finalist (one of 8), Search for Director, National Institute for Environmental Health Sciences, U.S. National Institutes of Health
2005 Member, Strategic Planning Conference, National Institute for Environmental Health Sciences, Research Triangle Park, NC
2006 Ad Hoc Reviewer, Draft of "Preterm Birth: Causes, Consequence, and Prevention" Committee on Understanding Premature Birth and Assuring Health Outcomes, Institute of Medicine, National Academies of Science
2006 Member, External Advisory Committee, NIEHS Center, University of Rochester
2007 Member, Ad Hoc Study Section, Special Emphasis Panel/Scientific Review Group 2007/05 ZES1 JAB-C (DI) (NIEHS Discover Centers)
2007-2010 Member, Board on Population Health and Public Health Practice, Institute of Medicine, National Academies, Washington DC.
2007 Member, Ad Hoc Review Panel, Centers of Excellence Program, Swedish Council for Working Life and Social Research.
2007-2008 Member, Search Committee for Director of Extramural Research, NIEHS
2007 Special Consultant, Ad Hoc Study Section, Special Emphasis Panel/Scientific Review Group 2008/01 ZAR1 CHW-G (NIAMS Arthritis Centers)
2008 Report Reviewer, Draft National Research Council Report, "The National Children's Study Research Plan: A Review", National Academies
2008 Report Reviewer, Draft National Research Council Report, "Gulf War and Health: Updated Literature Review of Depleted Uranium", Institute of Medicine, National Academies
2008-2009 Data Safety Monitoring Board, "d-Penicillamine Chelation in lead-poisoned Children—A Phase II/III Trial" (R01FD003361; PI: Michael Shannon)
2008 Subcommittee to review Draft Report on Bisphenol A, Science Board, Food and Drug Administration
2008 Planning Committee, International Symposium on the Environmental and Health Consequences of Metal Mining and Smelting
2008-2009 Co-Chair, Planning Committee, "Climate Change Impacts on Public Health in India", Workshop that took place in Goa, India in Aug-Sept 2009 co-sponsored by UM Center for Global Health, the US Centers for Disease Control and Prevention and the Indian Council for Medical Research

2008 Finalist (one of 2), Search for Director, National Institute for Environmental Health Sciences, U.S. National Institutes of Health
2009-2012 Member, Board on Environmental Studies and Toxicology, National Research Council
2009 Reviewer, NIH Challenge Grants, Special Emphasis Panel/Scientific Review Group 2009/10 ZRG1 GGG-F
2009-2010 External Member, Academic Program Review Site Visit Committee, Department of Environmental and Occupational Health Sciences, University of Washington School of Public Health
2010-2012 Member, External Advisory Committee, University of Rochester NIEHS P30 Core Center
2010 Member, Ad-hoc review committee, National Health Research Institutes of Taiwan, Special Emphasis Panel—NHRI-Kaoshiung Medical College Program Project on ": "Gene Environment Interaction in the Genesis of Asthma and Allergic Diseases"
2010-2012 Member, Advisory Board, Institute of Public Health, Florida Agricultural & Mechanical University, Tallahassee, FL
2011 Reviewer, NIEHS Career Development Awards, Special Emphasis Panel/Scientific Review Group 2011/05 ZES1 LKB-J (K9)
2011-2016 Member, NIEHS National Advisory Environmental Health Sciences Council
2012 Member, Editorial Board, Journal of Alzheimer's Disease
2015 Member and External Reviewer, School of Population and Public Health Review Committee, University of British Columbia, Vancouver, B.C.
2016- Chair, Board of Directors, Canadian Urban Environmental Health Research Consortium, (National Consortium based out of the Dalla Lana School of Public Health)
2017- Member, Energy Research Committee, Health Effects Institute, Boston, MA
2017-2018 Executive Co-Chair, Workshop on the Global Burden of Disease-Pollution and Health Initiative, March 1-2, 2018, Institute for Health Metrics and Evaluation, Seattle, WA
2017- Executive Co-Leader, Global Burden of Disease-Pollution and Health Initiative
2019- Member, Research Advisory Committee, Centre of Environmental Health, The Public Health Foundation of India and the Tata Institute of Social Sciences, New Delhi, India
2019- Reviewer, draft report on trace metals levels in pregnancy women, Agency for Toxic Substances and Disease Registry, Centers for Disease Control and Prevention, Atlanta

Regional

1988-1990 Health Facilities Appeals Board, Member, Dept. Public Health, Comm. Of Mass.
1988-2006 Advisory Board, Massachusetts Department of Public Health, Sentinel Event Notification System for Occupational Risks (SENSOR) Project
1989-1995 Advisory Board, Massachusetts Division of Occupational Hygiene, Lead Registry Project
1990-1992 Board of Directors, Member, Health Care for All, Boston, Massachussetts
1993-1995 Faculty Council, Member, Harvard School of Public Health
1995-2006 Faculty Advisory Committee, Public Health Practice Program, Harvard School of Public Health

1996-2006 Advisory Board, Boston VA Environmental Hazards Center, Boston
1997-2001 Faculty Steering Committee, Center for Children's Health, Harvard School of Public Health
1996-2006 Senior Epidemiology Consultant, Massachusetts Veterans Epidemiology Research and Information Center, Boston.
1996-2006 Associate, Center for Health and the Global Environment, Harvard Medical School
1997-2002 Faculty Advisory Committee on Continuing Professional Education, Harvard School of Public Health
1998-2006 Faculty Steering Committee, Masters of Public Health program, Harvard School of Public Health
2001-2003 Board of Directors, New England College of Occupational and Environmental Medicine
2001-2006 Associate Director, Harvard NIEHS Environmental Sciences Center, Harvard School of Public Health
2001-2006 Senior Advisory Council Member, Lowell Center for Sustainable Production, University of Massachusetts, Lowell, MA
2003-2006 Member, Human Subjects Committee, Harvard School of Public Health
2003-2006 Advisory Committee, Occupational Health Services Research Program, Harvard School of Public Health
2006 Study Section Review Committee, Pilot Project Program, Graham Environmental Sustainability Institute, School of Natural Resources and Environment, University of Michigan
2006-2007 Chair, Planning Committee, Health Sector, May 8-10, 2007 National Summit on Coping with Climate Change, University of Michigan
2007-2009 Member, Advisory Committee, SPH Practice Committee, University of Michigan School of Public Health
2007-2012 Member, Residency Advisory Committee, General Preventive Medicine Residency, University of Michigan School of Public Health
2008-2009 Member, Steering Committee, NIA T32 Training Grant on Aging Research (PI: Mary Haan), University of Michigan School of Public Health
2008-2013 Member, Advisory Committee, Outstanding New Environmental Scientist Awardee (Marie O'Neill), NIEHS
2008-2009 Member, Search Committee for Director of the Risk Science Center, University of Michigan School of Public Health
2009 Co-Chair, Planning Committee, Workshop on Predicting and Preventing Climate Change Impacts on Public Health, Goa, India (Collaboration with the UM Center for Global Health, the US Centers for Disease Control and Prevention, and the Indian Council for Medical Research)
2009-2011 Director and PI, NIA T32 Training Grant on Aging Research, University of Michigan School of Public Health
2009-2010 Member, Planning Committee, University Research Corridor (U of M, Michigan State, Wayne State) symposium on environmental health sciences in January 2010

2009-2012 Faculty Associate, Center for Global Health, University of Michigan
2009-2012 Member, Internal Advisory Board, Cancer Epidemiology Education in Special Populations Program, University of Michigan School of Public Health
2009-2011 Chair, Steering Committee on Global Health, University of Michigan School of Public Health
2010-2012 Member, Executive Committee, Graham Environmental Sustainability Institute, University Of Michigan
2010-2012 Member, Committee on Diversity, University of Michigan School of Public Health
2012-2017 Chair, Executive Committee, Dalla Lana School of Public Health, University of Toronto
2012-2017 Chair, Tenure Committee, Dalla Lana School of Public Health, University of Toronto
2012-2017 Chair, Decanal Promotions Committee, Dalla Lana School of Public Health, University of Toronto
2012-2017 Chair, Executive Advisory Committee, Institute for Global Health Equity & Innovation, Dalla Lana School of Public Health, University of Toronto
2013-2015 Interim Director, Institute for Global Health Equity & Innovation, Dalla Lana School of Public Health, University of Toronto
2013-2014 Co-Chair, Research Committee, Dalla Lana School of Public Health, University of Toronto
2014-2017 Chair, Executive Advisory Committee, Institute for Health Policy Management and Evaluation, University of Toronto
2014 Chair, Ad-hoc Committee to create an Institute for Indigenous Health (based on a $10 million endowment gift made to DLSPH), Dalla Lana School of Public Health, University of Toronto; Chair, Executive Advisory Committee beginning 2015
2015-2017 Chair, Executive Advisory Committee, Joint Centre for Bioethics, University of Toronto
2015- Chair (2015-2017); Member (2017-present), Taskforce on Environmental Health, Ministry of Health and Longterm Care, Province of Ontario
2016-2017 Chair, Executive Advisory Committee, Centre for Critical Qualitative Health Research, University of Toronto
2017-2018 Executive Co-Chair, Workshop on the Global Burden of Disease-Pollution and Health Initiative (a collaboration between the Global Alliance on Health and Pollution and the Institute for Health Metrics), Seattle, WA

Hospital

1982-1985 Occupational Safety and Health Committee, Member, Boston City Hospital, Boston
1983-1984 House Officers Association, Treasurer, Boston City Hospital
1984-1985 House Officers Association, Co-President, Boston City Hospital

OTHER PUBLIC SERVICE

1987 Member, Fact-finding tour on "The Health Effects of Massive Exposure to Tear Gas", Seoul, South Korea, July 11-18 (Sponsored by Physicians for Human Rights, American College of Physicians)

1988 Member, Fact-finding tour on "Chemical Weapons and the Iraqi Kurdish refugees", Turkey Oct 6-16 (Sponsored by Physician for Human Rights and the MacArthur Foundation)

1990 Leader, Fact-finding tour on "Health and Human Rights in Burma (Myanmar)", Thailand-Burma Dec. 26-Jan 6 (Sponsored by Physician for Human Rights and the MacArthur Foundation)

2009 Consultant and senior advisor, Fact-finding tour on “Mining and Potential Exposures and Health Effects in Guatemala”, August 2009 (Sponsored by Physicians for Human Rights)

CONSULTING POSITIONS

1987-1989 Consultant, "In-Vivo Total Body Lead Analysis by X-Ray Fluorescence", NIH/SBIR Grant 2R44ES03918-02

1988-1989 Consultant, "Boston Area Health Coalition Demonstration Project", DHHS/MP000003-A1

1993-1995 Consultant, Employee Health Services, Brigham and Women’s Hospital

1994 Consultant, Public Welfare Foundation, Washington, DC (review of Environmental Programs)

1997-2006 Consultant, Pediatric Environmental Health Center, Children’s Hospital, Boston, MA

2000 Consultant, Doris Duke Foundation, New York, NY (review of potential Environment and Medicine programs)

2009-2010 Consultant and Member, Academic Program Review Site Visit Committee, Department of Environmental and Occupationa Health Sciences, University of Washington School of Public Health, Seattle, WA

2011 Consultant, JPB Foundation, New York, NY (review of Environmental Health programs)

2014- Advisor, Hearing Health Sciences, Ann Arbor MI and Amsterdam, Netherlands

VISITING PROFESSORSHIPS

1997 Alice Hamilton Visiting Professor, University of California at San Francisco

2000-2001 Visiting Professor, Sri Ramachandra Medical College & Research Institute, Chennai, India

2004 Visiting Professor, Department of Environmental Medicine, University of Rochester

2013 Visiting Professor, Shanghai Key Laboratory, Shanghai Jiao-Tung University

SEMINARS AND EXTRAMURAL INVITED PRESENTATIONS (last 15 years, since 2003; prior presentations upon request)

2003 Guest lecturer, “Lead as a trans-generational toxin”, Seminar series, Department of OB/GYN, Brigham and Women’s Hospital
2003 Plenary speaker, “Clinical opportunities in environmental health”, Annual Leadership Retreat, National Institute for Environmental Health Sciences, Greensboro, NC.
2003 Discussant, “Uncontrolled Hypertension in a Painter”, Grand Rounds in Occupational/Environmental Medicine, Harvard School of Public Health
2003 Discussant, “A 53-Year Old Teacher with Chemical Sensitivities”, Grand Rounds in Occupational/Environmental Medicine, Harvard School of Public Health
2003 Lecturer, “Pestilence and Progress: The Future of Public Health through the Lens of Blood”, Center for Blood Research Symposium, Museum of Science, Boston, MA.
2003 Speaker, “Bones, Genes, Plasma, and Lead: New Frontiers in Understanding the Toxicity of an Old Hazard”, Distinguished Lecture Series. National Center for Environmental Health/Agency for Toxic Substances and Disease Registry, Centers for Disease Control and Prevention, Atlanta GA.
2003 Plenary speaker, “Biomarkers, Genes, Interactions and Lead: New Insights from Research on an Old Hazard", Superfund Basic Research Program Annual Meeting, Dartmouth University, Hanover, NH.
2003 Special Lecturer, “Lead Exposure and Chronic Disease: Recent Research on Susceptible Sub-populations", Florida Agricultural and Mechanical University, Tallahassee, FL.
2004 Speaker, “New Frontiers in Understanding the Toxicity of Lead”, Department of Environmental Medicine, University of Rochester, Rochester, NY.
2004 Presenter, “Lead Exposure During Pregnancy: Mobilization of Maternal Bone Lead Stores and Their Threat to the Fetus”, Semi-annual meeting of the Childhood Lead Poisoning Prevention Branch, Centers for Disease Control and Prevention, Baltimore, MD
2004 Presenter, “Environmental Medicine”, Annual meeting of the Editorial Board, *Environmental Health Perspectives*, Baltimore MD
2004 Plenary speaker, “Metals, Genes, and Neurodegeneration: the Approach of the Metals Epidemiology Research Group at the Harvard School of Public Health”, National Institute for Environmental Health Sciences Conference on Neurodegeneration.
2004 Discussant, “Suspected Lead Toxicity” Grand Rounds in Occupational/Environmental Medicine, Harvard School of Public Health
2004 Discussant, “Mercury Exposure in a Metal Worker”, Grand Rounds in Occupational/Environmental Medicine, Harvard School of Public Health
2004 Presenter, “Effects of Our Environment on Intellect, Behavior, Life and Death,” Leadership Council meeting, Harvard School of Public Health
2004 Guest Speaker, “Biomarkers, Genes, Interactions and Lead: New Insights from Research on an Old Hazard”, Department of Environmental Health, University of Michigan School of Public Health
2004 Guest Speaker, "Medicine, Public Health, and the Great American Melting Pot: A Second-Generation Chinese-American Reflects on His Personal Odyssey", Sponsored by the Asian Student Association, Harvard School of Public Health
2004 Speaker, “Aging, the Environment and Genetics: Recent Insights from

Epidemiologic Studies of Environmental Lead Exposure", Annual Leadership Retreat, National Institute for Environmental Health Sciences, Pinehurst, NC.

2004 Plenary Speaker, "Guidelines for the Management of Lead-Exposed Adults: Recommendations by a National Expert Panel Based on Recent Research", New England College of Occupational and Environmental Medicine Annual Meeting

2005 Lecturer, "Biomarkers, Genes, Interactions and Lead: New Insights from Research on an Old Hazard", Sri Ramachandra Medical College and Research Institute, Chennai, Tamil Nadu, India

2005 Lecturer, "Your Child's IQ, Behavior and Neuropathology: Genes or Environment?", the Harvard Club of Boston, Boston, MA

2005 Guest Speaker, "Metals, Neurodevelopment, and Neurodegeneration: The Work of the Metals Epidemiology Research Group at HSPH", Neurostatistics Working Group, Harvard School of Public Health, Boston, MA.

2005 Plenary Speaker, "Aging, the Environment and Genetics: Recent Insights from Epidemiologic Cohort Studies of Environmental Lead Exposure", NIEHS Symposium on Aging and the Environment, Duke University, Durham, NC.

2005 Plenary Speaker, "SPECT Imaging and Chemical Intolerance", NIEHS/NIAA symposium on "Chemical Intolerance and Addiction: a Shared Etiology?", Research Triangle Park, NC

2005 Workshop Presenter, "Social and Environmental Threats: the Unnecessary Epidemics", Harvard School of Public Health Leadership Council Annual Conference, Boston, MA

2005 Keynote Speaker, "Our Food, Our Water, Our Homes: Toxic Metals", The Boston Foundation, Boston, MA.

2006 Invited Speaker (invited by David Schwartz, NIEHS Director), "Goal IV: Improve and Expand Community-Linked Research", Roundtable on Environmental Health Sciences, Research, and Medicine; Institute of Medicine, National Academy of Sciences, Wash DC.

2006 Speaker, "The Future of Environmental Health Sciences at the University of Michigan", Dean's Advisory Board, University of Michigan School of Public Health, Ann Arbor, MI

2006 Keynote Speaker and Harriett Hardy Annual Lecturer, "The 'E' in Occupational/ Environmental Medicine: the Present and the Future", New England College of Occupational Medicine Annual Meeting, New Bedford, MA

2007 Speaker, "The Future of Environmental Health Sciences at the University of Michigan", Meetings of the UMSPH Alumni Council and the EHS Emeritus Faculty, Ann Arbor, MI

2007 Moderator and Speaker, "The Normative Aging Study: Health Effects of Lead", Symposium on the Health Effects of Lead, 2007 Annual Meeting of the International Society for Environmental Epidemiology, Mexico City, Sept 8, 2007

2007 Guest Lecture, "Uncovering the Impact of the Environment on Disease: Big Opportunities for Physician-Scientists", Medical Scientist Training Program, University of Michigan Medical School

2007 Guest Lecture, "Industrialization, Pollution and Public Health in India: Can India Survive Modernization?", Osher Institute, Ann Arbor, MI

2007 Plenary Speaker, "Environmental Equity: Local and Global Challenges and the Balance Between Research and Advocacy", Michigan's Premier Public Health Conference, October 16, 2007, Dearborn, MI

2007 Board Member Lecture, "Metals, Genes, Health and Human Rights: from the Molecular to the Global", Fall Meeting of the Board of Population Health and Public Health Practice, Institute of Medicine, National Academies of Science, Washington DC, Dec 13, 2007.

2008 Speaker, "MDs as Leaders for Change in Environmentalism", 2008 Annual Regional Political Leadership Institute, American Medical Student Association, University of Michigan Medical School, February 16, 2008

2008 Speaker, Grand Rounds, "The Impact of Environmental Pollutants on Disease: New Insights and Implications for Research and Medical Practice" Department of Medicine, University of Michigan Health System.

2008 Guest Lecture, "Emerging Insights into the Pervasive Influence of Environment Toxicants on Reproductive Outcomes and Offspring Development: Lead as a Case Study", Reproductive Sciences Program, University of Michigan

2008 Panelist, "Environmental Health in China", Public Health Grand Rounds, Division of Health Practice, University of Michigan School of Public Health

2008 Keynote Speaker, "Human Health and the Role of Water", Symposium on Water, Health & The Environment, Graham Environmental Sustainability Institute, University of Michigan

2008 Guest Speaker, "Lead Exposure and Toxicity: New Insights Using Molecular Epidemiology" Wadswoth Laboratories and SUNY-Albany

2008 Speaker: "Impact of Climate Change on Human Health: Vulnerability" 5th AKKA World Kannada Conference, Chicago IL

2008 Speaker, "The 'E' in Occupational/Environmental Medicine: the Present and the Future", Michigan Occupational/Environmental Medicine Annual Meeting, Mackinac Island, MI

2008 Speaker, "Impact of Climate Change on Human Health", University of Michigan Chapter of the American Medical Student Association, Ann Arbor, MI

2008 Speaker, "Early Life Origins of Adult Chronic Disease: Environmental Health and Toxicology at a Crossroads" Michigan Chapter fo the Society for Toxicology, Ann Arbor, MI

2009 Speaker, "Evidence for Lead as an Environmental Stressor of Alzheimer's Disease and the Role of Epigenetics", Symposium Panel, Annual Meeting of the Society for Toxicology, Baltimore, MD

2009 Keynote Speaker, "Lead, Late-Life and Early Life Effects, and the Emerging Field of Environmental Epigenetics: Looking Ahead", Annual Meeting of the American College for the Advancement of Medicine, San Diego, CA

2009 Speaker, "Lead Toxicity and Mechanistically-Oriented Molecular Epidemiology: Targeting the Epigenetics of Alzheimer's Disease", Seminar Series, Institute for Environmental Health Sciences, Wayne State University, Detroit, MI

2009 Speaker, "Climate Change Impacts on Health in the Developing World", Research Discussion Series, University of Michigan Center for Global Health

2009 Speaker, "Autism, Aggressive Behavior, Anxiety, and Alzheimer's: are Environmental Toxicants Playing a Major Etiologic Role?", Department of Psychology, University of Michigan

2009 Speaker, "Early Life Exposures and Endocrine Disruption: Evidence from Molecular

Epidemiology", Pediatric Endocrine Seminar, University of Michigan Medical School
2009 Distinguished Speaker, "Lead Toxicity: Twenty Years of Research On The Poison That Keeps on Poisoning" 10th Anniversary of the Department of Microbiology and Environmental Toxicology, University of California at Santa Cruz
2010 Speaker, "The Centers for Disease Control and Prevention & the Environmental Protection Agency: Potential Funding Opportunities for Regional Collaboration in Michigan", University Research Corridor Symposium on Environmental Health, Detroit, MI.
2010 Speaker, "The Future of Public Health", University of Washington School of Public Health
2010 Speaker, "The Environment Meets the Epigenome: Is This Where Autoimmunity Begins?" Symposium on Autoimmunity and Epigenetics, University of Michigan
2010 Keynote Speaker, "A New Twist to an Old Story: The Evidence for Early Life Lead Exposure as a Risk Factor for Alzheimer's Disease through Epigenetic Programming", NIEHS Environmental Health Sciences Center and Toxicology Training Program Retreat, University of Rochester, NY
2010 Speaker, "Lead Toxicity: Twenty Years of Research on The Poison That Keeps on Poisoning" and "Environmental Health Sciences at the University of Michigan", Tianjin Centers for Disease Control, Tianjin, China
2010 Speaker, "Pediatric Lead Toxicity", Xinhua Hospital and the Shanghai Jiao-Tung Medical University Department of Pediatrics, Shanghai, China
2010 Speaker, "Environmental Health Sciences at the University of Michigan", Fudan University, Shanghai, China
2010 Speaker, "Alzheimer's Disease, Epigenetics and the Environment", Symposium Update, Alzheimer's Disease Association, Ann Arbor, MI
2010 Speaker, "Environmental Justice, Progress (and the Lack Thereof) and the Role of Research", Roundtable on Environmental Health Sciences, Research and Medicine, Institute of Medicine, National Academies, Washington DC.
2010 Speaker, "White Coats, Population Science and Poison Gas: A Life Spent at the Intersection of Academic Medicine, Global Health & Human Rights", Robert Wood Johnson Clinical Fellows Program, University of Michigan Medical School, Ann Arbor, MI
2011 Speaker, "The Three Most Difficult Challenges to Molecular Epidemiologic Research on Gene-Environment Interactions: Lead Toxicity as a Case Study." Department of Human Genetics, University of Michigan Medical School, Ann Arbor, MI
2011 Speaker, "The Integration of Data on Environmental Carcinogens with Population and Genetic Resources", "Opportunities & Challenges for Translational Research on Cancer Prevention", Translational Cancer Prevention & Biomarkers Workshop, Mazamdur-Shaw Cancer Center, Bangalore, India.
2011 Speaker, "Success in the Academy", Faculty Panel, Students of Color of Rackham, Rackham Graduate School, University of Michigan
2011 Speaker, "White Coats, Population Science and Poison Gas: Fact-Finding Missions by Health Professionals for Human Rights" , Sujal Parikh Memorial Symposium, University of Michigan Medical School.
2011 Speaker, "The Analysis of Biomarker Data to Ascertain the Contribution of Environmental

Exposures to the Etiology of Disease: Lead Exposure and Toxicity as a Case Study", Department of Computational Medicine and Bioinformatics, University of Michigan Medical School.

2012 Speaker, "Research and Analysis Linking Upstream and Downstream Disparities Work", Webinar hosted by the Health & Environmental Funders Network, Bethesda, MD, with 52 Foundations related Health.

2012 Keynote Speaker, "The Future of Public Health & Medicine in a Crowded, Diverse, Stratified, Hot, Urbanized, Polluted, Thirsty, Hungry and Debt-Ridden World". E.J. Van Liere Memorial Convocation and Health Sciences Center Research Day, West Virginia University, Morgantown, West Virginia

2012 Plenary Speaker, "Transgenerational Impacts of Pollutants on Offspring: Recent Insights and Case Studies", Connaught Global Challenge International Symposium, University of Toronto.

2012 Speaker, "Environmental Impacts on Aging (+ an update on the Dalla Lana School of Public Health)", Community Medicine Rounds, University of Toronto

2012 Speaker, "The Environment & Public Health in a Research-Intensive University: Opportunities for Scholarship in a Crowded, Diverse, Stratified, Hot, Urbanized, Polluted, Thirsty, Hungry and Debt-Ridden World", School for the Environment, University of Toronto

2012 Speaker, "Big Public Health Challenges (& Opportunities) in a Crowded, Diverse, Aging, Stratified, Urbanized, Polluted, Hot, Thirsty, Hungry, Debt-Ridden World", External Advisory Meeting, Public Health Ontario, Toronto

2012 Speaker, "Canadian Public Health Schools (in a Crowded, Diverse, Aging, Stratified, Urbanized, Polluted, Hot, Thirsty, Hungry, Debt-Ridden World): The View from Toronto, External Advisory Board Meeting, Institute for Population and Public Health, Canadian Institutes for Health Research, Toronto

2012 Speaker, "Sustainable Development and Health: The Global Mining Industry", Canadian Society for International Health Annual Meeting, Ottawa

2012 Speaker, "Big Public Health Challenges (& Opportunities) in a Crowded, Diverse, Aging, Stratified, Urbanized, Polluted, Hot, Thirsty, Hungry, Debt-Ridden World", Xinhua Hospital/Shanghai Jiao-Tung University, Shanghai, China.

2012 Speaker, "The Impact of Population-Wide Lead Exposure and Gene-Lead Interactions on Chronic Disease", Genetic Grand Rounds, Sick Kids Hospital, Toronto.

2012 Speaker, "Looking behind the curtain: Lead Toxicity as a Case Study of Methodologic Challenges in Gene-Environment Interactions Research", Strategic Training in Advanced Genetic Epidemiology (STAGE), Dalla Lana School of Public Health, University of Toronto.

2012 Keynote speaker: "Public Health—the Next Frontier in Health Professions Education". Council of Health Sciences annual retreat, University of Toronto.

2013 Speaker, "White Coats, Population Science and Poison Gas: Lessons from a Life Spent at the Intersection of Academic Medicine, Global Health & Human Rights", Joint Center for Bioethics, University of Toronto

2013 Speaker, "Gauging environmental impact on the development of chronic inflammation", Connaught Global Challenge Workshop, University of Toronto.

2013 Speaker, "The Future of Public Health & Medicine in a Crowded, Diverse, Aging, Stratified, Urbanized, Polluted, Hot, Thirsty, Hungry, Debt-Ridden World", Grand Rounds, Department of Medicine, University of Toronto.

2013 Speaker, "Metals, Mega-trends, and Me: Reflections on Research and the Vision for the Dalla Lana SPH", Occupational and Environmental Medicine Grand Rounds, St. Michael's Hospital, Toronto, ON.

2013 Speaker, "Air pollution and Cardiovascular Disease: Health Impacts, Mechanisms, and Research Opportunities", UniversityofToronto & FMUSP-InCor SymposiumonCardiology, Sao Paolo, Brazil.

2013 Speaker: "Lead Exposure's Impact on Health and Policy: A History of Neglect and Missed Opportunities", Public Health Policy Rounds, CIHR Strategic Training Program in Public Health Policy, University of Toronto.

2013 Speaker: "Lead Toxicity: The Long Tail of Health Impacts (and On-going Research Opportunities!) From an Historical Environmental Air Pollutant", Southern Ontario Centre for Air Pollution and Aerosol Research, University of Toronto.

2013 Speaker: "Water and Sanitation", Water, Sanitation and Hygiene (WASH) Canada, Toronto, Ontario, Canada

2014 Speaker: "Conflict and Public Health", Ontario Medical Association, Toronto, Canada

2014 Panelist: "Judging Evidence: Finding a Place for Variation in an Evidence-Based World", Health Quality Ontario, Toronto, Canada

2014 Speaker: "The Grand Convergence: Creating Health in a Globalized World", Special meeting of the Canadian Chamber of Commerce in Shanghai

2014 Speaker: "The Grand Convergence: Creating Health in a Globalized World", Jockey School of Public Health and Primary Care, Chinese University of Hong Kong, Hong Kong, China

2015 Speaker: "The Grand Convergence: Creating Health in a Globalized World", School of Public Health and the ASEAN Institute, Mahidol University, Bangkok, Thailand

2015 Speaker: "Gene-environment Interactions and the Role of Big Data in Environmental Health" Seminar series, School of the Environment, University of Toronto, Toronto, Canada

2015 Speaker: "Global Health Security", Ill with Illness—Economic, Social & Security Barriers to the Provision of Global Health, Munk School of Global Affairs, University of Toronto, Toronto, Canada

2015 Speaker: "The Dalla Lana School of Public Health: Big Ideas and Initiatives for Creating Health in a Globalized World", Speaker Series, University of Toronto Alumni of Toronto.

2015 Speaker: "Unique Scientific Opportunities for the Precision Medicine Initiative National Research Cohort: Exposomics, Data Linkage, and Global Collaborations". Working group on President Obama's Precision Medicine Initiative (Chaired by Francis Collins, Director, NIH)

2015 Speaker: "What is the Role of Schools of Public Health in the 21st Century?" 50th Anniversary Celebration of the Department of Epidemiology, Biostatistics and Occupational Health, McGill University, Montreal, Quebec.

2015 Welcoming Address: "Global Public Health and Mental Health", Going Glo-cal for Mental Health conference, Centre for Addictions and Mental Health/Department of Psychiatry/Dalla

Lana School of Public Health, Toronto, ON

2015 John Goldsmith Memorial Lecture: “Big Data, Environmental (and Social) Epidemiology, Power and Politics”, Opening Plenary Session, International Society for Environmental Epidemiology Annual Meeting, Sao Paulo, Brazil

2015 Inaugural Speaker: “The Future of Public Health and Medicine in a Crowded and Complex World”, Global Health Leadership Series, PSG Medical School & the Shanti Ashram Foundation, Coimbature, Tamil Nadu, India

2016 Speaker “The Future of Public Health & Medicine in a Crowded, Diverse, Aging, Stratified, Urbanized, Polluted, Hot, Thirsty, Hungry, Debt-Ridden World”, Indian Institutes of Public Health—Hyderabad, Hyderabad, India

2016 Speaker: “Integration of Public Health & Health Care: The Unmet Agenda for a Truly Sustainable Health System”, Board of Directors Retreat, Toronto East General Hospital, Toronto

2016 Plenary speaker: “Health Promotion, Prevention and Health Protection: Innovative Initiatives”, 6th Asia-Pacific Conference on Public Health | 1st ASEAN Health Promotion Conference Bangkok, August

2016 Speaker: “Big Data, Environmental (and Social) Epidemiology, Power and Politics”, Mount Sinai School of Medicine, New York, NY

2016 Plenary Speaker: “The Impact of Environmental Toxicants on Health: Recent Epidemiologic Approaches & Advances”, International College of Integrative Medicine Annual Meeting, Toronto, ON

2016 Plenary Speaker: “Big Data and Implications for Environmental Health”, 15th Anniversary Conference, Jockey Club School of Public Health & Primary Care, Chinese University of Hong Kong, Hong Kong

2016 Plenary Speaker: “Innovations in Assessing Lead Poisoning and Child Health: Policy & Clinical Implications”, Chinese University of Hong Kong-Fudan-Oxford International Symposium on Health Impacts of Environmental Exposures”, Hong Kong

2016 Speaker: “Addressing a Changing Environment (and Impacts on Health, AKA Can India Survive Modernization?”, Indian Institutes of Technology Alumni, Canada, International Conference 2016, Toronto.

2016 Plenary Speaker, “Hidradenitis Suppurativa: Research Directions from a Population Health Perspective”, Symposium on Hidradenitis Suppurativa Advances, Toronto.

2016 Plenary Speaker, “Children’s Environmental Health”, The 2016 Annual National Conference on Children’s Healthcare, Shanghai, China

2016 Special Guest Speaker, “Big Data, Environmental (and Social) Epidemiology, Power and Politics”, Shanghai Municipal Center for Disease Control, Shanghai, China

2016 Lecturer, “Lead and Human Health: Recent Research and Associated Lessons for Science & Policy”, Fudan University School of Public Health, Shanghai, China

2017 Lecturer, “The Impact of Environmental Toxicants on Health: Recent Epidemiologic Approaches & Advances”, Saw Swee Hock School of Public Health, National University of Singapore, Singapore

2017 Lecturer, “The Future of Academic Public Health”, Saw Swee Hock School of Public Health, National University of Singapore, Singapore

2017 Lecturer, "Recent Advances in Understanding, Preventing, and Reversing the Impact of Environmental Factors on Health", Society of Chinese Bioscientists in America, Li Ka Shing Knowledge Institute, St. Michael's Hospital, Toronto, ON

2017 Lecturer, "Environmental Epidemiology in the Era of Exposomics, Lifecourse Epidemiology, Big Data and Big Science", Department of Environmental Health, Harvard School of Public Health, Boston, MA

2017 Speaker, "The Role of a Re-emergent Canadian School of Public Health in a Hot, Hungry, Polluted, Aging, Polarized World Prone to Pandemics, Chronic Disease, and Unsustainable Health Systems", Royal Canadian Institute for Science, Toronto, ON

2017 Speaker, "The Early Life Exposures in Mexico to Environmental Toxicants (ELEMENT) Birth Cohort Study: Current Research on Fluoride and Neurodevelopment", Seminar Series in Environmental Epidemiology, University of Washington School of Public Health, Seattle, WA

2017 Plenary Speaker: "New realities arising from the extractive industries and agri-business: the Pollution and health perspective," Hong Kong Summit of Global Health Leaders. University of Hong Kong, Hong Kong

2018 Plenary Speaker: "The GBD-Pollution and Health Initiative: Challenges & Opportunities", Workshop on the Global Burden of Disease-Pollution and Health Initiative, Institute for Health Metrics, University of Washington, Seattle, WA

2018 Guest Lecturer: "Partnerships, Local Responsiveness, National and Global Impacts", University of Iowa College of Public Health, Iowa City, IA

2018 Plenary Speaker: "Current Research on Fluoride and Neurodevelopment: The Early Life Exposures in Mexico to Environmental Toxicants (ELEMENT) Birth Cohort Study", Annual meeting of the International Academy of Oral Medicine and Toxicology, Denver, CO

2018 Speaker, "Recent Epidemiologic Research on Lead Toxicity: New Surprises regarding an Old Global Pollutant", Department of Environmental and Occupational Health Sciences Seminar Series, University of Washington School of Public Health, Seattle, WA

2018 Speaker: "The Early Life Exposures in Mexico to Environmental Toxicants (ELEMENT) Birth Cohort Study: Current Research on Fluoride and Neurodevelopment", Symposium on Fluoride research, Annual meeting of the International Society for Environmental Epidemiology/International Society for Exposure Science, Ottawa, ON

2018 Panelist, "The Fluoridation Decision: Considering the Evidence for Benefits, Possible Risks as well as Ethical World Views", Annual meeting of the International Society for Environmental Epidemiology/International Society for Exposure Science, Ottawa, ON

2018 Speaker: "Grand Opportunities", The UC-Irvine School of Population Health and the Samueli College of Health Sciences, Irvine, CA

2018 Speaker, "The Global Burden of Disease-Pollution and Health Initiative", Office of the Director and the Global Environmental Health Program, U.S. National Institute for Environmental Health Sciences, Research Triangle Park, NC

2019 Speaker, "Evaluating, treating and managing disabilities of patients with chemical intolerance", Symposium on Chemical Intolerance—A Way Forward, Marilyn Brachman

Hoffman Foundation and the Hoffman Program on Chemicals and Health at the Harvard T.H. Chan School of Public Health, Dallas, TX

2019 Guest Lecturer: "The Global Burden of Disease-Pollution and Health Initiative", Center for Population Health Sciences, Stanford University, Palo Alto, CA

INVENTIONS/PATENTS: n/a

BIBLIOGRAPHY: (H-index, as of April, 2019, Google Scholar: 83)

Peer-reviewed journals

1. Hu H, Markowitz SB. A case-study of industrial bladder cancer. Einstein Quarterly Review of Biology and Medicine 1982;1:29-35.

2. Hu H. Benzene and myelofibrosis. Annals of Internal Medicine 1987;106:171-172

3. Hu H, Milder FL, Burger DE. X-Ray Fluorescence: Issues surrounding the application of a new tool for measuring burden of lead. Environmental Research 1989;49:295-317.

4. Hu H, Fine J, Epstein P, Kelsey K, Reynolds P, Walker B. Tear Gas: Harrassing agent or toxic chemical weapon? JAMA 1989;262:660-663.

5. Hu H, Cook-Deegan R, Shukri A. The use of chemical weapons: Conducting an investigation using survey epidemiology. JAMA 1989;262:640-643.

6. Hu H, Tosteson T, Aufderheide AC, Wittmers L, Burger DE, Milder FL, Schidlovsky G, Jones KW. Distribution of lead in human bone: I. Atomic absorption measurements. Basic Life Sci 1990;55:267-274.

7. Burger DE, Milder FL, Morsillo PR, Adams BB, Hu H. Automated bone lead analysis by k-x-ray fluorescence for the clinical environment. Basic Life Sci 1990;55:287-292.

8. Schidlovsky G, Jones KW, Burger DE, Milder FL, Hu H. Distribution of lead in human bone: II. Proton microprobe measurements. Basic Life Sci 1990;55:275-280.

9. Jones KW, Schidlovsky G, Burger DE, Milder FL, Hu H. Distribution of lead in human bone: III. Synchrotron x-ray microscope measurements. Basic Life Sci 1990;55:281-286.

10. Hu H, Milder FL, Burger DE. X-ray fluorescence measurements of lead burden in subjects with low-level community lead exposure. Arch Environ Health 1990;45:335-341.

11. Hu H, Win KU, W, Arnison ND. Burma: Health and human rights. Lancet 1991;337:1335.

12. Hu H. A 50-year follow-up of childhood plumbism: hypertension, renal function, and hemoglobin levels among survivors. Am J Dis Child 1991;145:681-687.

13. Hu H. Knowledge of diagnosis and reproductive history among survivors of childhood plumbism. Am J Publ Health 1991;81:1070-1072.

14. Hu H, Milder FL, Burger DE. The use of K-X-Ray Fluorescence for measuring lead burden in epidemiological studies: high and low lead burdens and measurement uncertainty. Environ Health Perspect 1991;94:107-110.

15. Hu H, Pepper L, Goldman R. Effect of repeated occupational exposure to lead, cessation of exposure, and chelation on levels of lead in bone. Am J Ind Med 1991;20:723-735.

16. Hu H. Toxic weapons, epidemiology, and human rights. Polit Politics and Life Sci 1992;February:3-4.

17. Hu H, Sparrow D, Weiss S. Association of serum albumin with blood pressure in the Normative Aging Study. Am J Epidemiol 1992;136:1465-1473.

18. Hu H, Christiani D. Reactive airways dysfunction after exposure to tear gas. Lancet 1992;339:1535.

19. Hu H. Physicians, IPPNW, and the Environment. PSR Quarterly 1993;3:79-87.

20. White RF, Diamond R, Proctor S, Morey C, Hu H. Residual cognitive deficits 50 years after lead poisoning during childhood. Br J Industr Med 1993;50:613-622.

21. Hu H, Beckett L, Kelsey K, Christiani D. The left-sided predominance of asbestos-related pleural disease. Am Rev Resp Dis 1993;148:981-984.

22. Payton M, Hu H, Sparrow D, Young JB, Landsberg L, Weiss ST. Relation between blood lead and urinary biogenic amines in community-exposed men. Am J Epidemiol 1993;138:815-825.

23. Hu H, Kotha S. Ethics and epidemiology: International Guidelines. Polit Life Sci 1993;February:29-30.

24. Goldman RH, White R, Kales SN, Hu H. Lead poisoning from mobilization of bone stores during thyrotoxicosis. Am J Industr Med 1994;25:417-424.

25. Bellinger D, Hu H, Titlebaum L, Needleman HL. Attentional correlates of dentin and bone lead levels in adolescents. Arch Environ Health 1994;49:98-105.

26. Watanabe H, Hu H, Rotnitzky A. Correlates of bone and blood lead levels in carpenters. Am J Industr Med 1994;26:255-264.

27. Hu, H. Decision-making in human health impact assessments: a clinician's perspective. Environ Impact Assess Rev 1994;14:439-450.

28. Hu H, Watanabe H, Payton M, Korrick S, Rotnitzky A. The relationship between bone lead and hemoglobin. JAMA 1994;272:1512-1517.

29. Payton M, Hu H, Sparrow D, Weiss ST. Low-level lead exposure and renal function in the Normative Aging Study. Am J Epidemiol 1994;140:821-829.

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31. Guo H-R, Chiang H-S, Hu H, Lipsitz SR, Monson RR. Arsenic in drinking water and urinary cancers: a preliminary report. Environ Geochem Health 1994;s16:119-128.

32. Hoppin JA, Aro ACA, Williams PL, Hu H, Ryan PB. Validation of K-xrf bone lead measurements in young adults. Environ Health Perspect 1995;103:78-83.

33. Smith CM, Wang X, Hu H, Kelsey KT. A polymorphism in the δ-Aminolevulinic acid dehydratase gene may modify the pharmacokinetics and toxicity of lead. Environ Health Perspect 1995;103:248-253.

34. Hu H, Aro A, Rotnitzky A. Bone lead measured by X-ray fluorescence: Epidemiological methods. Environ Health Perspect 1995;103(Suppl 1):105-110.

35. Kim R, Aro A, Rotnitzky A, Amarasiriwadena C, Hu H. K x-ray fluorescence measurements of bone lead concentration: the analysis of low-level data. Phys Med Biol 1995;40:1475-1485.

36. Kim R, Hu H, Rotnitzky A, Bellinger D, Needleman H. A longitudinal study of chronic lead exposure and physical growth in Boston children. Environ Health Perspect 1995;103:952-957.

37. Hu H, Kotha S, Brennan T. The role of nutrition in mitigating environmental insults: policy and ethical issues. Environ Health Perspect 1995;103(Suppl 6):185-190.

38. Smith CM, Hu H, Wang X, Kelsey K. Delta-aminolevulinic acid dehydratase genotype is not associated with hematocrit or hemoglobin levels among construction trade workers exposed to low

levels of lead. Medicine Lavoro 1995;86:229-235.

39. Makhijani A, Hu H, Yih K. Nuclear wastelands: Nuclear weapons production worldwide and its environmental and health effects. Med Global Surv 1995;2:26-34.

40. Potula V, Hu H. Occupational and lifestyle determinants of blood lead levels among men in Madras, India. Int J Occup Environ Health 1996;2:1-4.

41. Hu H, Hashimoto D, Besser M. Levels of lead in blood and bone of women giving birth in a Boston hospital. Arch Environ Health 1996;51:52-58.

42. Hu H, Aro A, Payton M, Korrick S, Sparrow D, Weiss ST, Rotnitzky A. The relationship of bone and blood lead to hypertension: The Normative Aging Study. JAMA 1996;275:1171-1176.

43. Kim R, Rotnitzky A, Sparrow D, Weiss ST, Wager C, Hu H. A longitudinal study of low-level lead exposure and impairment of renal function: The Normative Aging Study. JAMA 1996;275:1177-1181.

44. Proctor SP, Rotnitzky A, Sparrow D, Weiss ST, Hu H. The relationship of blood lead and dietary calcium to blood pressure in the Normative Aging Study. International Journal of Epidemiology 1996;25:528-536.

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46. Hu H, Payton M, Korrick S, Aro A, Sparrow D, Weiss ST, Rotnitzky A. Determinants of bone and blood lead levels among community-exposed middle-aged to elderly men: the Normative Aging Study. Am J Epidemiol 1996;144:749-759.

47. Hernandez-Avila M, Gonzalez-Cossio T, Palazuelos E, Romieu I, Aro A, Fishbein E, Peterson K, Hu H. Dietary and environmental determinants of blood and bone lead levels in lactating postpartum women living in Mexico City. Environ Health Perspect 1996;104:1076-1082.

48. Potula V, Hu H. Relationship of hemoglobin to occupational exposure to motor vehicle exhaust. Tox Industr Health 1996;12:629-637.

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53. González-Cossío T, Peterson KE, Sanín L, Fishbein SE, Palazuelos E, Aro A, Hernández-Avila M, Hu H. Decrease in birth weight in relation to maternal bone lead burden. Pediatrics 1997;100:856-862.

54. Hu H, Rabinowitz M, Smith D. Bone lead as a biological marker in epidemiologic studies of chronic toxicity: Conceptual paradigms. Environ Health Persp 1998;106:1-8.

55. Payton M, Riggs KM, Spiro A, Weiss ST, Hu H. Relations of bone and blood lead to cognitive function: The VA Normative Aging Study. Neurotox Teratol 1998;20:19-27.

56. Amarasiriwardena C, Lupoli N, Potula V, Korrick S, Hu H. Determination of total arsenic concentration in human urine by inductively coupled plasma mass spectrometry: a comparison of the accuracy of three analytical methods. Analyst 1998;123:441-445.

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62. González-Cossío T, Sanín LH, Hernández-Avila M, Rivera J, Hu H. Longitud y peso al nacer: El papel de la nutrición materna. [Length and weight at birth: The role of maternal nutritional status.]

Salud Pública Mex [Public Health in Mexico] 40:119-126, 1998.

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Abstracts of Work (Upon request)

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UNITED STATES DISTRICT COURT
FOR THE NORTHERN DISTRICT OF CALIFORNIA
AT SAN FRANCISCO

FOOD & WATER WATCH, et al., Plaintiffs, vs. U.S. ENVIRONMENTAL PROTECTION AGENCY, et al. Defendants.	Civ. No. 17-CV-02162-EMC **DECLARATION OF BRUCE LANPHEAR, MD, MPH**

TABLE OF CONTENTS

I, Bruce Lanphear, MD, MPH, declare that:

1. I am a Clinical Investigator at the BC Children's Hospital Research Institute, BC Children's Hospital, and Professor in the Faculty of Health Sciences at Simon Fraser University in Vancouver, British Columbia.

2. I am also the Co-Principal Investigator of an ongoing study to examine the impact of early-life fluoride exposures on intellectual abilities in a cohort of mothers and offspring from Canada known as the MIREC Study. Our study of fluoride and IQ in the MIREC cohort was funded by a grant from the U.S. National Institutes of Health (NIH).

I. SUMMARY OF QUALIFICATIONS

3. A complete summary of my qualifications and publications can be found in my Curriculum Vitae, which has been marked as Plaintiffs' Exhibit 6 and attached herein.

4. I have studied the impact of toxic chemicals, including lead and pesticides, on children's brain development for over 20 years. My research has been almost exclusively funded by federal agencies, including the Environmental Protection Agency (EPA), Centers for Disease Control and Prevention, the Department of Housing and Urban Development, Health Canada, National Institute of Allergy and Infectious Diseases, National Institute for Child Health and Human Development, National Institute of Environmental Health Sciences, National Institute of Neurologic Diseases, and the National Heart, Lung and Blood Institute.

5. My research has been published in leading medical and scientific journals, including *Journal of the American Medical Association*, *New England Journal of Medicine*, and *Pediatrics*, and has been extensively relied upon by environmental and public health agencies, including the EPA. My pooled analysis of blood lead and IQ (Lanphear 2005) was cited by the EPA as the critical study upon which the Agency based the current national air standard for lead.

6. I have served on the editorial boards of seven academic journals, including *Public Health Reports* (the official journal of the U.S. Surgeon General), *PLoS Medicine* (a peer-reviewed medical journal published by the Public Library of Science), and *Environmental Health Perspectives* (a journal funded by the National Institutes of Environmental Health Sciences).

7. I have served on numerous scientific committees on environmental health issues impacting children, including multiple scientific advisory boards for the EPA and the Executive Council on Environmental Health for the American Academy of Pediatrics. My work with the EPA has included invited expert advisory roles on EPA's (i) Science and Research Work Group of the Children's Health Protection Advisory Committee (1998-2001); (ii) Workshop on Assessing Environmental Exposures to Children (2000-2002); (iii) Clean Air Scientific Advisory Committee (2006-2008); (iv) Science Advisory Board for Evaluating Dust Lead Standards (2010-2012); and (v) Science Advisory Board for Evaluating Hazards of Partial Water Line Replacement (2011-2012).

8. My research has earned various awards and honors, including the Research Integrity Award from the International Society for Environmental Epidemiology in 2012, the Public Policy and Advocacy Award from the Academic Pediatric Association in 2013, and the Research Award from the Academic Pediatric Association in 2015.

9. I have been involved with the MIREC Study for over 10 years. I was a Co-Principal Investigator for the neurobehavioral assessments conducted when the children were 3 to 4 years old and I oversaw the neurodevelopmental assessments in Vancouver. I have been a coauthor of twelve publications from the MIREC Study, including three publications on fluoride described below.

10. In light of the consistent association between elevated fluoride and IQ reported in cross-sectional studies (Choi, et al. 2012), we received a grant from the NIH to study the association between prenatal and early-life measures of fluoride and IQ in children in the MIREC cohort. To date, we have

published three peer-reviewed studies, including the most extensive assessment of fluoride exposure during pregnancy ever conducted and prospective studies on early life fluoride exposure on IQ. These studies have been published in *Environmental Health Perspectives*, *JAMA Pediatrics*, and *Environment International* (Till 2018, Green 2019, Till 2020). Our study on prenatal fluoride and IQ was the highest scoring study in *JAMA Pediatrics* in 2019 (Christakis 2020).

11. I agreed to participate as a non-retained expert in this case because I believe it is a public duty to present the results of studies that suggest substantial risk to public health. I have asked *not* to be compensated for this work.

II. SUMMARY OF OPINIONS

12. Our study of prenatal fluoride and IQ in the MIREC cohort (Green 2019) further enhances the quality of data related to the neurotoxicity of fluoride. As with the ELEMENT cohort, we employed a prospective cohort design, had extensive control for potential confounders, and had multiple measures of fluoride exposure during pregnancy, including three types of urinary fluoride measurements for each trimester of pregnancy.

13. The maternal urinary fluoride levels in the MIREC cohort were significantly associated with lower intellectual abilities in 3-4-year-old children. These associations remain large and significant when controlling for relevant covariates.

14. Converging results from the MIREC and ELEMENT cohorts indicate that exposure to "optimal" levels of fluoride during fetal development is associated with diminished intelligence in childhood.

15. In the MIREC cohort, exposure to fluoridated water in infancy, particularly among formula-fed infants, was also associated with diminished intelligence (Till 2020). This association remains significant after controlling for fetal fluoride exposure and other relevant covariates, suggesting that

susceptibility to fluoride's adverse neurological effects may extend into infancy.

III. BASIS FOR OPINIONS

A. The Growing Problem with Brain-Based Disorders

16. As I have discussed elsewhere, the causes of death and disability in children have shifted over the past century (Lanphear 2015). Concerted public health efforts to control tuberculosis, cholera, typhoid, and other infectious agents in the early twentieth century led to a dramatic reduction in child mortality, followed by a rise in life expectancy. By the end of the twentieth century, the 'new morbidities of childhood' had emerged: attention deficit hyperactivity disorder (ADHD), autism, asthma, obesity, and preterm birth. Learning disabilities and neurodevelopmental disorders are now two of the most prevalent morbidities in children. About 7.6% of US children are estimated to have a learning disability, and 13% are estimated to have a neurodevelopmental disorder, including anxiety, autism, conduct disorder, depression, or ADHD (Lanphear 2015). These data indicate that we are in the midst of an epidemic of brain-based disorders.

17. Neurotoxicants can have a lifelong impact on brain function. Children who have higher blood lead concentrations, for example, may never meet the same peak cognitive ability in adulthood as that in less exposed children. At the other end of the age spectrum, cognitive decline is accelerated in adults who have higher bone lead concentrations and some evidence has shown that lead exposure is a risk factor for the development of late-onset Alzheimer's disease. Few birth cohorts have been studied into adulthood; however, it would be surprising if the effects of other neurotoxicants observed in school-aged children do not persist into adulthood (Lanphear 2015). The cumulative impact of exposures to various toxins that only modestly impact intellectual abilities can be substantial (Lanphear 2015).

18. The high reported prevalence of learning disabilities and neurodevelopmental disorders has fueled research to better understand the role of environmental chemicals, including the use of prospective

cohort studies that collect individualized biomarkers of exposure to environmental toxins. Biologic markers, or biomarkers, of exposure, which can enhance our ability to quantify an individual's internal dose of a contaminant, are revolutionizing the study of environmental toxins in the same way genetic tests are revolutionizing the study of heritability (Lanphear 2015).

B. The MIREC Cohort Is a Comprehensively Characterized Birth Cohort

19. The MIREC[1] cohort in Canada was developed to obtain biomonitoring data for pregnant women and their infants to examine potential adverse health effects of early-life exposure to environmental chemicals.

20. The MIREC cohort is a geographically diverse and comprehensively characterized birth cohort. Women were recruited during the first trimester of pregnancy from 10 cities across Canada, including cities that add fluoride to water for caries prevention purposes (e.g., Toronto), and cities that do not (e.g., Vancouver). Women were followed through delivery and their offspring have undergone periodic neurodevelopmental tests, including IQ testing.

21. We administered questionnaires during pregnancy and early childhood to collect information on demographics, occupation, lifestyle, medical history, environmental exposures and diet. Dietary questions included whether the mother drank tap water during pregnancy, how many glasses of water and other beverages she consumed, and duration of breastfeeding.

22. Information on the pregnancy and the infant was abstracted from medical charts. Maternal urine was collected at multiple points throughout pregnancy, as was blood, urine, hair, breast milk, cord blood and infant meconium. These samples have been archived in a biobank.

23. Study staff from each participating study site completed a 3-day training session that was

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[1] MIREC stands for Maternal-Infant Research on Environmental Chemicals. It is an interdisciplinary collaboration between Health Canada scientists and clinical and academic researchers, and was funded by Health Canada, the Ontario Ministry of the Environment, and a grant from the Canadian Institutes of Health Research.

led by a PhD-level psychologist and focused on specialized training of the neurodevelopmental tests. The training emphasized the importance of providing an ideal and standardized environment in the home by ensuring that the test area was well-lit, quiet, and free from distractions and interruptions.

C. Urinary Fluoride Study (Till 2018)

24. In 2018, we published the most comprehensive study of urinary fluoride during pregnancy that has ever been conducted (Till 2018). Our study included 1,566 pregnant women from the MIREC cohort who had urine samples for each trimester of pregnancy. It was the first study of its kind in water-fluoridated areas of North America. A similar study has recently been published of a smaller pregnancy cohort in California (Uyghurturk 2020), but our study remains the largest and most thorough.

1. Methodological Strengths

25. Our study of urinary fluoride was conducted in accordance with sound and objective science practices. Important strengths of the study include: (1) a large study size, with over 1,500 women and over 5,000 urine samples; (2) collection of urine samples from each trimester for each mother; (3) empirical data on the actual measured water fluoride levels for each mother's water treatment plant boundary (WTP) during the course of the pregnancy; (4) control for other factors that have potential to influence urinary fluoride excretion, including tea consumption, alcohol use, pre-pregnancy BMI, maternal age, maternal education, annual household income, and race; (5) control for fluctuations that can occur in urine fluoride during the day by adjusting for dilution using two methods (specific gravity and creatinine) and controlling for time of void and time since last void; and (6) measurement of fluoride in urine using the same scientist (Dr. Martinez-Mier), method (microdiffusion), and laboratory (University of Indiana) as the ELEMENT cohort, thereby enhancing the comparability of the data.

26. Dr. Martinez-Mier's lab at the University of Indiana is considered a gold-standard lab for the testing of fluoride in urine and blood. EPA appears to recognize Dr. Martinez-Mier's expertise as she

was approached by EPA to serve as an expert in this case.

2. Fluoridated Water Has a Large and Significant Effect on Urinary Fluoride

27. In our study, fluoride in water had the strongest correlation with urine fluoride of all the factors that we measured, thus confirming that fluoridated water remains a major source of fluoride intake (Till, 2018).

28. The average urinary fluoride level among pregnant women in fluoridated areas is almost two times higher than the average levels in nonfluoridated areas (Till 2018).

29. The average creatinine-adjusted[2] maternal urinary fluoride level in the fluoridated areas is 0.87 mg/L, versus 0.46 mg/L in the non-fluoridated areas.[3]

30. Our data suggests that, for every 0.5 mg/L increase in water fluoride level, urinary fluoride levels will increase by 74-82%.

31. Our findings are consistent with prior studies showing that, among adults, fluoride levels in urine are closely correlated with the concentration of fluoride in water.

32. As part of our study, we attached a table showing the full distribution of urinary fluoride levels, including the 75th and 95th percentile exposures for each trimester (Till et al. 2018, Table S4). At the second trimester, 95th percentile values in the fluoridated areas were 2 mg/L (adjusted for creatinine), and 1.63 mg/L (adjusted for specific gravity).

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2 Creatinine-adjustment of spot urine samples adjusts for dilution and has been found to have good agreement with 24-hour fluoride values (WHO 2014, Zohouri 2006). We used the same method for creatinine adjustment as Bashash (2017). We also adjusted for dilution by correcting for specific gravity. Our adjustments for creatinine and specific gravity produced results that were highly correlated ($r = 0.91$) and interchangeable in our analyses of the factors that increase urinary fluoride.

3 The average specific gravity-adjusted concentration in the fluoridated areas was 0.71 mg/L (SD 0.38 mg/L), with a range of 0.10 to 3.12 mg/L.

3. Pregnant Women in Water-Fluoridated Areas of the MIREC Cohort Have Similar Urinary Fluoride Levels as the ELEMENT Cohort

33. One of the key findings from our 2018 study is that pregnant women who live in water-fluoridated areas of Canada have urinary fluoride levels that are essentially the same as the urinary fluoride levels documented in the ELEMENT cohort.

34. The similarity in maternal urinary fluoride levels between the MIREC and ELEMENT cohorts can be appreciated when comparing the respective mean values (0.87 vs. 0.91 mg/L), standard deviations (0.50 vs. 0.40 mg/L), and ranges (0.14-3.80 vs. 0.02-3.67 mg/L) of the study participants in the water fluoridated areas of the MIREC cohort vs. the ELEMENT cohort.

35. The similarity in maternal urinary fluoride levels between the MIREC and ELEMENT cohorts is of scientific and public health relevance given the findings of Bashash (2017) showing an inverse association between maternal urinary fluoride and offspring IQ.

D. Prenatal Fluoride/IQ Study (Green 2019)

36. In 2019, we published our findings on the relationship between prenatal fluoride exposure and IQ in the MIREC cohort. The study, which was published in *JAMA Pediatrics*, provides reliable and unbiased results and was conducted in accordance with sound and objective science practices.

1. Methodological Strengths

37. ***Prospective Birth Cohort Study Design***: A key strength with our study is that we used a prospective cohort study design, also known as a longitudinal study. Prospective studies are the best available method for investigating the impact of environmental chemicals and, as noted earlier, have helped to revolutionize our understanding of how chemicals impact childhood health.

38. ***Extensive Control for Potential Confounders***: The MIREC cohort is one of the most comprehensively characterized birth cohorts, with abundant individualized data on factors that may influence neurodevelopment. We took full advantage of this data. We excluded study participants if there

was a known fetal abnormality, if they had any medical complications (i.e., cancer, renal disease, cardiovascular disease), or if there was known maternal alcohol or drug abuse during pregnancy. For analyses using water fluoride, we excluded women if they did not drink tap water during their pregnancy. For those not excluded, we controlled for the following factors: maternal education; maternal age; quality of the child's home environment (HOME); child sex; mother's race; city of residence; secondhand smoke; maternal blood or urinary concentrations of other neurotoxicants, such as lead, arsenic, mercury, manganese, and PFOA. Additionally, we controlled for the diurnal fluctuations that may occur in urinary fluoride levels by including time of day that the urine sample was collected and time since last void.

39. ***Individualized Measures of Fluoride Exposure***: Another important strength of our study is that we had multiple individualized measures of prenatal fluoride exposure, including: (1) urinary fluoride samples averaged across each trimester of pregnancy and corrected for urinary dilution; (2) measured water fluoride levels from within each participant's water treatment plant boundary during the course of their pregnancy;[4] and (3) questionnaire data about how much water each woman drank from tap water and water-based beverages.

40. ***Large, Multi-Center Cohort***: Our study included 512 mother-offspring pairs which is a robust sample size for statistical analyses. Further, the mother-offspring pairs came from 6 cities across Canada, some of which fluoridate their water (average F = 0.59 mg/L), and some of which do not (average F = 0.13 mg/L).[5] Our study thus permitted us to examine the impact of fluoride exposures on IQ in communities with water fluoridation, thus addressing what some have perceived to be a limitation of prior

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[4] Water treatment plants measured fluoride levels daily if fluoride was added to municipal drinking water and weekly or monthly if fluoride was not added to water. We matched participants' postal codes with water treatment plant zones, allowing an estimation of water fluoride concentration for each woman by averaging water fluoride concentrations (in milligrams per liter) during the duration of pregnancy.

[5] Pregnant women in the fluoridated areas tended to have higher incomes and more education, which may help explain the lack of difference in average *unadjusted* IQs between fluoridated and non-fluoridated areas (Green 2019, Table 1).

studies of fluoride neurotoxicity.

41. ***Reliable and Objective IQ Test***: We assessed children's intellectual abilities with the Wechsler Preschool and Primary Scale of Intelligence, Third Edition (WPPSI-III). This is a validated IQ test with excellent internal reliability (r = 0.96), and good test-retest reliability (r = 0.86). We used Full Scale IQ (FSIQ), which is a measure of global intellectual functioning, as the primary outcome. The FSIQ score is comprised of two composite scores: Verbal IQ (VIQ)—representing verbal reasoning and comprehension—and Performance IQ (PIQ)—representing nonverbal reasoning, spatial processing, and visual-motor skills.

42. ***Blinded Assessments***: As with the ELEMENT cohort, our study was "blinded," meaning the examiners were not aware of the mother's fluoride exposure status at the time of the examination. This eliminates the potential for examiner bias.

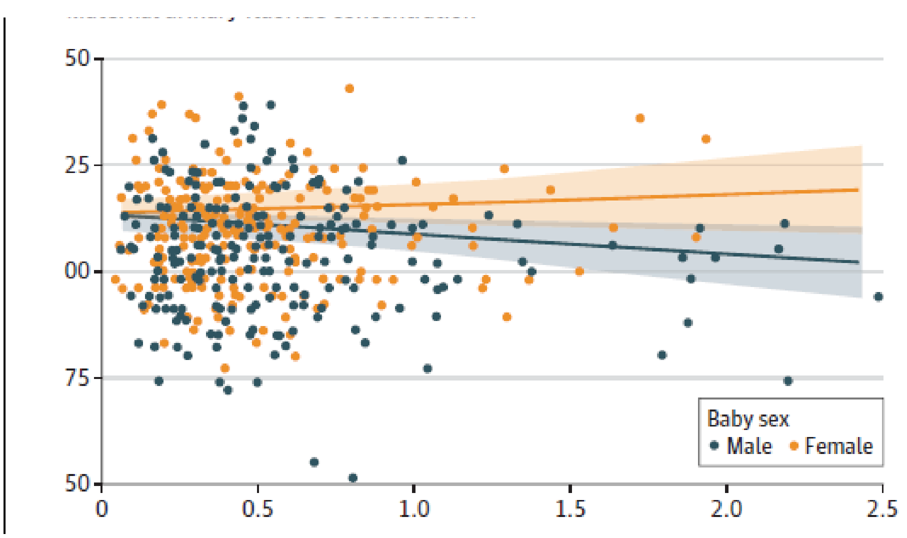
43. ***Statistical Analyses that Did Not Assume Linearity***: We conducted sound, objective, and rigorous statistical analyses that: (i) controlled for the impact of the large number of measured covariates; (ii) examined the data for collinearity, outliers, and influential points; and (iii) scrutinized the shape of the relationship between fluoride and IQ. With respect to linearity and possible threshold effects, we conducted sensitivity analyses that used quadratic and natural log effect models as well as ran spline analyses that examined the relationship below 0.5 mg/L, 0.8 mg/L, and 1.0 mg/L in urine (and below 0.4 mg/day and 0.8 mg/day in fluoride intake). We did *not* assume that statistical associations between fluoride and IQ were linear and without threshold.

44. Short of intentionally dosing pregnant mothers with fluoride, we maximized the power of environmental epidemiology to investigate whether prenatal fluoride exposure is associated with neurocognitive deficits in a prospective, observational cohort study.

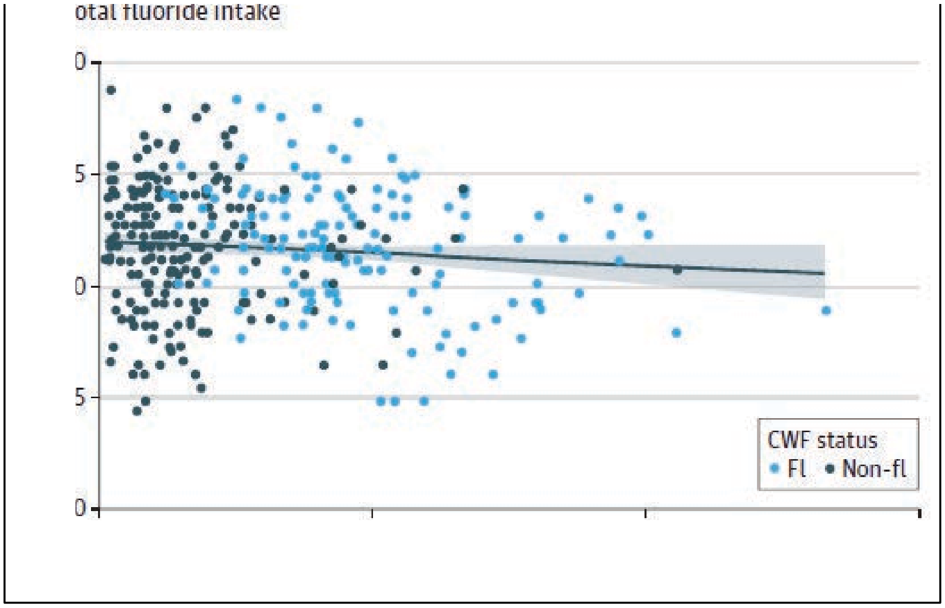
2. Prenatal Fluoride Exposure Is Associated with Large and Significant Reductions in IQ in the MIREC Cohort

45. All three measures of prenatal fluoride exposure—(i) maternal urinary fluoride, (ii) maternal fluoride intake from beverages, and (iii) water fluoride concentration during pregnancy—were associated with large, statistically significant decreases in IQ among the 3 to 4-year old children in the MIREC cohort.

46. After controlling for covariates, a 1-mg/L increase in maternal urinary fluoride[6] was associated with a **4.49 lower IQ** score (in boys); a 1-mg higher daily intake of fluoride from beverages was associated with a **3.66 lower IQ** score (in boys and girls); and a 1 mg/L higher water fluoride concentration was associated with a **5.29 lower IQ** score (in boys and girls). These fluoride-associated reductions in IQ that we found showed a linear, dose-response relationship with no apparent indication of a threshold (see figures below).



[6] We used specific gravity (SG) as the primary adjustment method for this study. The average SG-adjusted concentration among women from the fluoridated areas was 0.69 mg/L (SD = 0.42), which is consistent with the concentration in fluoridated areas from the larger cohort (0.71 mg/L, SD = 0.38) (Till 2018). Sensitivity analyses using the creatinine-adjusted urinary fluoride values did not change the results between fluoride and IQ (Green 2019, eTable 2).



47. The effect sizes that we found are large and rival the impact of a population blood lead concentration of 5 µg/dL.[7] Fourteen percent of the women had urinary fluoride concentrations exceeding 1.0 mg/L, and thus the impact for some children may exceed the ranges identified above.

48. Although maternal urinary fluoride was associated with significant reduction in full-scale IQ in boys, it was not associated with diminished full-scale IQ in girls. As is often the case, the reason for this discrepancy is not currently known. It is known, however, that boys have a higher prevalence of neurodevelopmental disorders such as ADHD, learning disabilities, and intellectual disabilities than girls. It is also known that boys and girls may respond differently to some neurotoxicants. In our studies of the MIREC cohort, for example, we found that low levels of blood lead correlate with a loss of IQ in boys, but not girls (Desrochers-Couture 2018).

49. The possibility that boys are more sensitive to prenatal fluoride exposure than girls is

[7] My pooled analysis of blood lead and IQ found that an increase in childhood blood lead from <1 to 10 µg/dL was associated with a 6.9 IQ point decrement (Lanphear 2015, p. 814). General population exposures to lead today are now on the low-to-mid range of this spectrum, with relatively few children having blood lead levels as high as 10 µg/dL.

supported by at least one animal study, in which males had greater deficits from prenatal exposure and females had greater deficits from postnatal exposure (Mullenix et al 1995). While we did not find sex-specific differences with our other two exposure measures (water F concentration and water F intake), these other measures may better correlate with chronic or postnatal exposure and thereby reflect distinct risks.

3. Convergent Findings of ELEMENT and MIREC Cohorts.

50. The significant associations we observed between prenatal fluoride and IQ in the MIREC cohort are consistent with the findings from the ELEMENT cohort (Bashash, 2017).

51. In the ELEMENT study, an increase of 1 mg/L in creatinine-adjusted maternal urine was associated with a loss of 6.3 IQ points among 4-year olds, as measured by General Cognitive Index (GCI) of the McCarthy Scales of Children's Abilities. In our analysis, an increase of 1 mg/L in creatinine-adjusted maternal urine was associated with a loss of 4.96 IQ points among 3- to 4-year old boys, as measured by the WPPSI-III test (Green 2019, eTable 2). These effect sizes are generally consistent with each other.

52. The consistency of results in both population cohorts adds further confidence that the association is real, particularly when viewed in the context of other studies that have reported inverse associations between fluoride and IQ in many different locations in other countries, as well as general knowledge about the vulnerability of the developing brain.

E. Infant Fluoride/IQ Study (Till 2020)

53. We recently completed and published a study that examined whether fluoride exposures during infancy have an influence on IQ at 3-4 years (Till 2020).

54. Concerns have been raised about the use of fluoridated water in baby formula due to the high intake of water by bodyweight during infancy. These high intakes have been associated with

significant increases in dental fluorosis, including in the permanent teeth.

55. In our study, we obtained information about fluoride intake of infants through questionnaires that the mothers completed when the children were 30 to 48 months of age.[8] The questionnaire included the question, "How old was your baby when you ceased breastfeeding exclusively?" Women who breastfed exclusively for six months or longer were included in the breastfeeding (BF) group; those who reported introducing formula within the first six months (never breastfed or partial breastfeeding) were included in the formula-feeding (FF) group.

56. As a separate measure of infant fluoride exposure, we estimated fluoride intake by obtaining the measured water fluoride levels within the water treatment plant boundary during infancy. We took the average of these levels, multiplied by the amount of time that that the infant was not exclusively breast-fed during the first year,[9] and divided it by the estimated average water intake among Canadian formula-fed infants (0.8 L). We excluded any mother-offspring pair if the mother reported not drinking tap water.

1. Methodological Strengths

57. Our study of infants shares many of the same methodological strengths as our prenatal study, including: (i) prospective cohort design; (2) extensive control for potential confounders (discussed below); (3) blinded assessment; (4) relatively large cohort (398 mother-offspring pairs) from both fluoridated and non-fluoridated areas; (5) the same validated and standardized IQ test that we used for the prenatal study (i.e., Wechsler Primary and Preschool Scale of Intelligence-III); and (6) rigorous statistical

8 The answers to these questions correlated well with the contemporaneous infant feeding practices that were reported among 11% of the cohort. Among women with breastfeeding questionnaire data and infant feeding observation data, the median difference for when breast-feeding ceased was 0 months, with two-thirds being within 1.5 months of each other.

9 The mean duration of exclusive breastfeeding was 4.98 months; 13.6% of women reported never breastfeeding, 8% reported discontinuing breastfeeding after the first three months, and 50.2% reported continuing to breastfeed at six months or longer. Average water fluoride concentration did not significantly differ between the BF (mean=0.32 mg/L) and FF groups (mean=0.29 mg/L; p=.18).

analyses that tested for sex-specific effects and scrutinized the impact of outliers and influential points.

58. We adjusted for potential confounding by selecting covariates *a priori* that have been associated with fluoride, breastfeeding, and children's intellectual abilities. Final covariates[10] included child's sex and age at testing, maternal education, maternal race, second-hand smoke in the home, and quality of the child's home environment (measured at time of testing using the Home Observation for Measurement of the Environment (HOME). We also controlled for fetal fluoride exposure, using the previously measured maternal urinary fluoride concentrations averaged across each trimester of pregnancy.

2. Fluoride Exposure During Infancy Is Associated with Significant Reductions in Non-Verbal IQ in the MIREC Cohort

59. We found that fluoride exposure during infancy is associated with significant reductions in non-verbal IQ in the MIREC cohort.

60. For each 0.5 mg/L increase in water fluoride concentration, we found a decrease of 4.4 Full-Scale IQ (FSIQ) points among preschool children who were formula-fed in the first six months of life; 0.5 mg/L is the approximate difference in mean water fluoride level between fluoridated (0.59 mg/L) and non-fluoridated (0.13 mg/L) regions. In contrast, we did not find a significant association between water fluoride concentration and FSIQ among children who were exclusively breastfed in the first 6 months.

61. The association between water fluoride concentration and FSIQ must be interpreted with caution, however, because the association became nonsignificant when two outliers were removed.

62. We observed an even stronger association between water fluoride and PIQ (non-verbal intelligence). A 0.5 mg/L increase in water fluoride level predicted a decrement in non-verbal IQ in both

[10] For each analysis, a covariate was retained in the final model if its p-value was <0.20 or its inclusion changed the regression coefficient of water fluoride concentration or fluoride intake from formula by more than 10%.

the formula-fed (**9.3-points**) and the breastfed groups (**6.2-points**). Adjusting for fetal exposure or removing two extreme scores did not appreciably alter these results.

63. We observed converging results using fluoride intake from formula feeding, which is a continuous, time-weighted exposure estimate. For each 0.5 mg/day of fluoride intake, we found an **8.8-point** decrement in non-verbal IQ; adjusting for fetal exposure attenuated the association only slightly (**7.6-point** decrement).

64. The time-weighted fluoride intake estimate may reflect a more refined measure of exposure in infancy because it captures differences in both water fluoride level and the proportion of time each child was given formula over the first year of life. Yet, our binary classification of whether a child was exclusively breastfed for the first 6 months may distinguish infants who are exposed to higher amounts of fluoride during the early infancy period when the brain undergoes significant development because breast milk contains minutes amounts of fluoride.

65. Taken together, these findings suggest that using "optimally" fluoridated water (0.7 mg/L) to reconstitute infant formula may diminish the development of intellectual abilities in young children, particularly non-verbal abilities. The findings also suggest that both prenatal and early childhood fluoride exposure affect the development of non-verbal intelligence to a greater extent than verbal intelligence. Prior studies examining prenatal exposure to fluoride and IQ showed a similar pattern.

F. The Limitations of Our Studies Do Not Provide a Likely Explanation for the Results

66. As with all epidemiological studies, our fluoride studies have limitations. These limitations, however, are unlikely to explain the large and significant associations that we have found between early-life exposures and IQ.

67. Most of the limitations in our studies involve fluoride measurement. These limitations

include: (1) use of spot urine samples instead of 24-hour samples;[11] (2) lack of water fluoride measurements from the participant's home; (3) reliance on questionnaire data as to water consumption and breast-feeding; (4) lack of information on the fluoride content of the infant formula concentrate and other sources of fluoride exposure; and (5) use of non-validated methods[12] for estimating total beverage-based intake of fluoride by mothers and water fluoride intake by infants.

68. The limitations in our exposure estimates are non-differential, meaning they apply equally to study participants with low fluoride exposure and high fluoride exposure. Non-differential errors in exposure measurement will generally bias the results towards the null (i.e., attenuate, rather than inflate, an association between exposure and outcome). Because of this, the limitations in our exposure estimates do not provide a likely explanation for the significant IQ decrements we observed with fluoride exposures. If anything, these limitations likely attenuated the relationship.

69. Another limitation of our studies is that the MIREC cohort tends to be more affluent, more educated, and less ethnically diverse than the general population. Our results may thus not be representative of how fluoride may affect IQ in more disadvantaged populations. On the other hand. affluent populations tend to have less confounders (e.g., less exposure to other stressors and toxicants that can negatively affect neurodevelopment) (Lanphear 2015). We thus worry less about the role of confounders in the MIREC cohort than we would in other cohorts.

70. Finally, despite our comprehensive array of covariates included, our studies could not address the possibility of unmeasured residual confounding. This is a limitation in all observational

[11] A 24-hour sample of urine is considered the optimal dosimeter for measuring chronic fluoride exposure (WHO 2014). While we did not have 24-hour samples available to test, we partially controlled for this by adjusting samples by creatinine. Creatine-adjusted urine fluoride measurements have been found to have a strong correlation with 24-hour samples (Villa 2010; Zohouri 2006).

[12] These methods were not validated in the sense that they have not yet been replicated by other authors in other studies. Our method for assessing fluoride intake from beverages did show internal validity, however, by predicting maternal urinary fluoride levels. This can be seen in the highly significant correlations that we found ($p < 0.0001$) between maternal urinary fluoride and number of glasses of water consumed per day and black tea consumption (see Till 2018).

studies, and thus inherent to the field. One of the potential confounders for which we lacked data was maternal IQ. We did, however, control for maternal education which is highly correlated with maternal IQ. Moreover, a greater proportion of women living in fluoridated communities (76%) had a university-level degree compared with women living in nonfluoridated communities (66%), and thus it seems unlikely that controlling for maternal IQ would affect our results, particularly since there is no reason to believe that maternal IQ would be correlated with maternal urinary fluoride.

71. I understand that the EPA has suggested that the location of our study in Canada somehow reduces the relevance of our findings to populations in the US. I disagree. From a biologic standpoint, there is no credible basis to believe that people in the U.S. will respond differently to fluoride than people in Canada. Nor am I aware of any credible reason to conclude, let alone suspect, that people in water-fluoridated areas of the U.S. are exposed to materially less fluoride than people in water-fluoridated cities of Canada.

IV. CONCLUSION

72. The collective evidence from prospective cohort studies supports the conclusion that fluoride exposure during early brain development diminishes the intellectual abilities in young children, including at the purportedly "optimal" levels of exposure for caries prevention.

I declare under penalty of perjury, under the laws of the United States, that the foregoing is true and correct to the best of my knowledge and belief.

Executed on May 20, 2020, in Vancouver, British Columbia, Canada.

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BRUCE LANPHEAR, MD, MPH

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1992-1994	National Research Scholar Award in General Pediatric Research, University of Rochester School of Medicine and Dentistry.
1992-1997	Assistant Professor, Department of Pediatrics and of Community & Preventive Medicine, University of Rochester School of Medicine.
1997-2002	Associate Professor, Department of Pediatrics, Children's Hospital Medical Center and the University of Cincinnati, Cincinnati, Ohio.
1997-2008	Director, General Pediatric Research Fellowship Training Program, Children's Hospital Medical Center and the University of Cincinnati.
1997-2008	Director, Children's Environmental Health Center, Children's Hospital Medical Center and the University of Cincinnati.
1997-2006	Associate Professor (Adjunct), Departments of Pediatrics and of Environmental Medicine, University of Rochester School of Medicine & Dentistry, Rochester, NY.
1998-2003	Associate Director for Research, Division of General & Community Pediatrics, Children's Hospital Medical Center.
2001-2002	Associate Professor (tenured), Department of Pediatrics, University of Cincinnati, Cincinnati, Ohio.
2001-2004	Associate Professor (Adjunct), Department of Environmental Health Sciences, University of Michigan School of Public Health, Ann Arbor, Michigan.
2002-2008	Sloan Professor of Children's Environmental Health, Departments of Pediatrics and Environmental Health, University of Cincinnati, Cincinnati Children's Hospital Medical Center, Cincinnati, Ohio.

2008-2012	Adjunct Professor of Pediatrics, Department of Pediatrics, Cincinnati Children's Hospital Medical Center and the University of Cincinnati.
2008-	Professor of Children's Environmental Health, Faculty of Health Sciences, Simon Fraser University
2008-	Clinician Scientist, Child & Family Research Institute, BC Children's Hospital, University of British Columbia

Education

1980-1985	Bachelor of Arts in Biology
1980-1986	University of Missouri at Kansas City, Medical Degree (1986)
1986-1987	Internship, University of Arkansas for Medical Sciences, Little Rock, Arkansas
1987-1988	Tulane School of Public Health & Tropical Medicine Masters in Public Health & Tropical Medicine
1987-1989	General Preventive Medicine and Public Health Residency Tulane School of Public Health & Tropical Medicine
1992-1995	Postdoctoral Fellowship in General Academic Pediatric Research University of Rochester School of Medicine, Rochester, NY

Awards and Honors

2011	Sterling Prize in Controversy, Simon Fraser University
2012	Research Integrity Award, International Society for Environmental Epidemiology
2013	Public Policy and Advocacy Award, Academic Pediatric Association
2015	Research Award, Academic Pediatric Association
2015	Confederation of Union Faculty Associations of British Columbia (CUFA-BC) Academic of the Year Award
2018	Lumina Award from the Women for Healthy Environmental Health, Pittsburgh, PA

Teaching Experience

1992-1997 Course Instructor, "Public Health & the Environment", Department of Community & Preventive Medicine, The University of Rochester School of Medicine and Dentistry. A required course for MPH students taught annually.

1997-2008 Founding Director, NIH-funded, General Academic and Community Pediatric Research Fellowship Training Program, Cincinnati Children's Hospital Medical Center. This interdisciplinary, research training program, which included pediatricians, psychologists and epidemiologists, was the first training program in Children's Environmental Health.

1998-2008 Course Co-Instructor, "Children's Health & the Environment", Department of Environmental Health, The University of Cincinnati School of Medicine. A course taught every other year to MPH, PhD and postdoctoral trainees in medical subspecialties.

2008- Course Instructor, "Children's Health and the Environment". A 2-week intensive course taught annually to 4th year undergraduate students at Simon Fraser University.

2011- Course Instructor, "Plagues, Pollutants and Poverty: The Origins and Evolution of Public Health". An undergraduate course at Simon Fraser University.

Committee and Community Involvement

1993-1997	Lead Poisoning Prevention Task Force, Monroe County Health Department.
1994-1997	Investigational Review Board, Rochester General Hospital
1995-	Scientific Consultant, National Center for Healthy Housing, Columbia, Maryland.
1996-1997	Member, New York State Task Force on Environmental Neurotoxins, University of Rochester School of Medicine
1996-2001	Member, National Institute for Environmental Health Sciences Grant Review Committee for Community-Based Interventions (FG)
1996-1998	Chairman, U.S. Department of Housing and Urban Development Committee on Lead-Contaminated House Dust
1998	Member, Review Group for National Research Service Awards, Health Resources and Services Administration
1998-2000	Member, Cincinnati Board of Health, Cincinnati, Ohio.
1998-2001	Member, Science and Research Work Group, Office of Children's Health Protection Advisory Committee, U.S. EPA
1998-2000	Member, Cincinnati Lead Poisoning Prevention Advisory Task Force, Cincinnati, Ohio.
1999	Member, K23 Grant Review Committee, National Institute for Environmental Health Sciences, August 1999
1999	Member, Expert Panel on Soil Pica Behavior, Agency for Toxic Substance Disease Registry, June 7th-8th, Atlanta, Georgia
2000	Member, Panel on Health Disparities: Linking Biological and Behavioral Mechanisms with Social and Physical Environments, National Institute for Environmental Health Sciences, July 14-15th
2000-2002	Member, Workshop on Assessing Environmental Exposures to Children, U.S. Environmental Protection Agency, July 26-27th
2000-2004	Member, Children's Environmental Health Project, AAP's Child Health Research Center, Rochester, NY.
2001	Senate Testimony, "Ensuring that Children with Dangerous Levels of Lead in their Blood Receive Care as Early as Possible". Subcommittee on Housing and Transportation of the Committee on Banking, Housing and Urban Affairs, 107th U.S. Congress, November 13th, 2001.
2001	Reviewer, National Research Council, National Academy of Science Update of the 1999 Arsenic in Drinking Water Report
2001-2003	Member, Expert Panel on Children's Health and the Environment, North American Commission for Environmental Cooperation
2002-	Member, Scientific Advisory Board, Scientist Communication Network.
2003	Member, "Herculaneum Health Study Workshop" Agency for Toxic Substance Diseases Registry, May 22nd to 23rd, 2003
2003-2004	Panel Member, "Lead Poisoning in Pregnant Women", Mt. Sinai for Children's Health and the Environment, New York, NY
2003	Member, "Invitational Workshop on a proposed American Family Study" National Human Genome Research Institute, December 1st to 3rd, 2003.

2004-2006	Member, Committee on "Ethical Consideration for Research on Housing-Related Health-Hazards involving Children", National Research Council and the Institute of Medicine, The National Academies
2004	Congressional Testimony, "Tapped Out? Lead in the District of Columbia and the Providing of Safe Drinking Water", Subcommittee on Environment and Hazardous Materials of the Committee on Energy and Commerce, U.S. House of Representatives, 108th Congress, July 22nd, 2004
2005	Reviewer, "Superfund and Mining Megasites – Lessons from the Couer d" Alene River Basin", National Research Council, The National Academies.
2005	Ad Hoc Member, NIEHS Board of Scientific Counselors Review of the Epidemiology Branch, April 3rd to April 5th, 2005
2005	Senate Briefing, "The Connection of Environmental Chemicals and Learning Disabilities", U.S. Senate, May 10th, 2005
2006	Invited Participant, NIEHS Strategic Planning Forum, National Institute for Environmental Health Sciences, Chapel Hill, North Carolina, October 17-18th, 2006.
2006-2008	Member, U.S. EPA's Clean Air Scientific Advisory Committee Lead Review Panel.
2006-2008	Member, National Children's Study Steering Committee, NICHD
2006	Invited Participant, "How Does Housing Affect Health Outcomes of Children?", MacArthur Foundation, Chicago, Illinois, June 21st-22nd, 2006.
2006- 2010	Member, External Scientific Advisory Committee, Richmond Center for Excellence in Tobacco Research, American Academy of Pediatrics.
2007	Testimony, Vermont State Legislature, "The Lingering Legacy of Lead Toxicity", Montpelier, Vermont, February 1st, 2007
2007	Testimony, Connecticut State Legislature, "The Legacy of Lead Toxicity", Hartford, Connecticut, March 14th, 2007. (PG)
2007	Invited Testimony, United States Senate Hearing, "Lead and Children's Health". Committee on Environmental and Public Works, October 18th, 2007
2007-2008	Member, Committee on "Committee on Contaminated Drinking Water at Camp Lejeune", National Research Council, The National Academies.
2008	Member, Expert Panel on Health and the Environment, Statistics Canada, Ottawa,
2008-	Member, Alliance for the Global Elimination of Lead Paint, Intergovernmental Forum on Chemical Safety (IFCS), World Health Organization
2008-2009	Reviewer, Toxicological Review and Recommended Toxicological Reference Values for Environmental Lead Exposure in Canada, Health Canada
2009-2013	Scientific Advisor, Canada Lead Study funded by Health Canada (Patrick Levallois, Principal Investigator).
2009-2014	Board Member, Barro Sin Plomo
2009-2010	Member, Health and Environment Experts Advisory Group of the Canadian Longitudinal Study on Aging, Canadian Institutes of Health Research
2010-2012	Member, US Environmental Protection Agency Science Advisory Board for Evaluating Dust Lead Standards
2010-2013	Advisor, Canada Environmental Health Law and Canadian Partnership for Children's Health and Environment Retrofit Project
2010-2012	Member, Physicians Advisory Panel, Canada Health Measures Survey
2010	Invited Testimony, United States Senate Hearing, "Research on Environmental Health Factors with Autism and Neurodevelopmental Disorders", August 3rd, 2010
2010	Member, Joint FAO/WHO Expert Panel for Toxicological and Health Review of Bisphenol A
2010-2015	Board Member, Global Community Monitoring, Oakland, California
2010-	Chairman, Scientific Advisory Committee for Dartmouth University's Program in Children's Health and the Environment

2011-2016	Member, American Academy of Pediatrics Executive Council on Environmental Health
2011-2012	Member, US Environmental Protection Agency Science Advisory Board for Evaluating Hazards of Partial Water Line Replacement
2011	Invited Testimony, Special Committee on Cosmetic Pesticides, Legislative Assembly, Province of British Columbia, October 7th, 2011
2011-2012	Member, Panel on Health Effects of Low-level Lead, Office of Health Effects, National Toxicology Program of the National Institutes of Environmental Health Sciences,
2012-	Member, Expert Advisory Committee, Canada Health Measures Survey
2012-	Member, Environmental Defence Fund Science Advisory Committee on Toxics
2015	Reviewer, Review of Clinical Guidance for the Care of Health Conditions Identified by the Camp Lejune Legislation, Institute of Medicine, The National Academies
2016-	Member, The Lancet Commission on Pollution, Health & Development
2016-	Member, Targeting Environmental Neuro-Developmental Risks (TENDR)
2016	Member, Steering Committee, The National Lead Summit, United States
2017	Rockefeller Foundation Academic Writing Retreat, Bellagio, Italy
2017-	Member, Advisory Committee for the Flint (MI) Cohort Study
2017-	Pure Earth Leadership Council
2018-	Member Project TENDR Advisory Board
2018-	Member, Mercury Disability Board Committee, Health Canada

Editorial Boards

2000-2015	Assistant Editor, *Environmental Research*
2000-2008	Deputy Editor, *Public Health Reports*
2004	Associate Editor, *Pediatrics* supplement on Children's Environmental Health
2004-2017	Editorial Board Member, *PLoS Medicine*
2005-2014	Editorial Board Member, *Breastfeeding Medicine*
2007-	Editorial Board Member, *Environmental Health*
2008-2012	Editorial Review Board Member, *Environmental Health Perspectives*
2012-2015	Associate Editor, *Environmental Health Perspectives*
2016-	Advisor, *Environmental Health Perspectives* News Section

Societies and Organizations

1989-2008	American Public Health Association
1996-2015	Academic Pediatric Association
1997-2012	American Association for the Advancement of Science
2000-2008	Society for Pediatric Research
2001-2008	American Pediatric Society
2001-2016	Specialty Fellow, American Academy of Pediatrics
2006-	Fellow, Collegium Ramazzini
2006-	Member, International Society for Environmental Epidemiology
2008-	Founding Member, International Society for Children's Health & the Environment
2011-2017	Secretary and Treasurer, International Society for Children's Health & the Environment
2012-	Member, International Society for Exposure Science
2017-2018	Vice-President, International Society for Children's Health & the Environment
2019-2020	President, International Society for Children's Health & the Environment

Video and Website Production – www.littlethingsmatter.ca

1. Canadian Environmental Health Atlas: A Portal to Discover the Promise of Environmental Health.
2. Shifting the Curve: The Impact of Toxins on ADHD in U.S. Children (video)
3. Little Things Matter: The Impact of Toxins on the Developing Brain (video)
4. Little Things Matter: The Impact of Toxins on Preterm Birth (video)
5. Prevention Paradox: Why We are Failing to Prevent Disease (video)
6. Little Things Matter: The Deadly Impact of Airborne Particles (video)
7. Cause or Cure: A Plea for Prevention (video)
8. Crime of the Century: The Failure to Prevent the Lead Pandemic (video)

Original Research

1. Lanphear BP. Deaths in Custody. American Journal Forensic Medicine & Pathology 1987;8:299-301.
2. Lanphear BP, Snider DE. Myths of Tuberculosis. J Occ Med 1991;33:501-504.
3. Linnemann CC Jr, Cannon C, DeRonde M, Lanphear BP. Effect of educational programs, rigid sharps containers, and universal precautions on reported needle-stick injuries in healthcare workers. Infection Control Hospital Epidemiology 1991;12:214-20.
4. Lanphear BP, Buncher CR. Latent period for malignant mesothelioma of occupational origin. Journal Occupational Med 1992;34:718-721.
5. Lanphear BP, Linnemann CC Jr, Cannon CG, DeRonde MM. Decline of clinical hepatitis B in workers at a general hospital. Clinical Infectious Disease 1993:11:10-14.
6. Lanphear BP, Linnemann CC Jr, Cannon C, DeRonde MM, Pendy L, Kerly L. Hepatitis C virus infection in health care workers. Infection Control Hospital Epidemiology 1994;15:745-750.
7. Lanphear BP, Emond M, Jacobs DE, Weitzman M, Winter NL, Tanner M, Yakir B, Eberly S. A side-by-side comparison of dust collection methods for sampling lead-contaminated house-dust. Environmental Research 1995;68:114-123.
8. Lanphear BP, Winter NL, Apetz L, Eberly S, Weitzman M. A randomized trial of the effect of dust control on children's blood lead levels. Pediatrics 1996;98:35-40.
9. Christy C, Pulcino M, Lanphear BP, McConnochie K. Screening for tuberculosis infection in urban children. Arch Pediatrics Adolescent Med 1996;150:722-726.
10. Lanphear BP, Weitzman M, Eberly S. Racial differences in environmental exposures to lead. American Journal of Public Health 1996;86:1460-1463.
11. Lanphear BP, Weitzman M, Winter NL, Tanner M, Yakir B, Eberly S, Emond M, Matte TD. Lead-contaminated house dust and urban children's blood lead levels. American Journal of Public Health 1996;86:1416-1421.

12. Lanphear BP, Byrd RS, Auinger P, Hall CB. Increasing prevalence of recurrent otitis media among children in the United States. Pediatrics 1997. http//www.pediatrics.org/cgi/contents/full99/3/e1.
13. Emond MJ, Lanphear BP, Watts A, Eberly S for the Rochester Lead-in-Dust Study Group. Measurement error and its impact on the estimated relationship between dust lead and children's blood lead. Environmental Research 1997;72:82-92.
14. Lanphear BP, le Cessie S, Atkinson WL, Watelet L. Association of live births and the resurgence of measles. International Journal Epidemiology 1997;26:204-211.
15. Rust SW, Burgoon DA, Lanphear BP, Eberly S. Log-additive versus log-linear analysis of lead-contaminated house dust and children's blood lead levels: Implications for residential dust-lead standard. Environmental Research 1997;72:173-184.
16. Lanphear BP, Roghmann KJ. Pathways of lead exposure in urban children. Environmental Research 1997;74:67-73.
17. Lanphear BP, Byrd RS, Auinger P, Schaffer S. Community characteristics associated with elevated blood lead levels in children. Pediatrics 1998;101:264-271.
18. Lanphear BP, Rust SW, Burgoon DA, Eberly S, Galke W. Environmental exposures to lead and urban children's blood lead levels. Environmental Research 1998;76:120-130.
19. Lanphear BP. The paradox of lead poisoning prevention. Science 1998;281:1617-1618.
20. Lanphear BP, Hall CB, Black J, Auinger P. Risk factors for the early acquisition of HHV-6 and HHV-7 infection in children. Pediatric Infectious Disease Journal 1998;17:792-795.
21. Lanphear BP, Matte TD, Rogers J, Clickner R, Dietz B, Bornschein RL, Succop P, Mahaffey KR, Dixon S, Galke W, Rabinowitz M, Farfel M, Rohde C, Schwartz J, Ashley P and Jacobs DE. The contribution of lead-contaminated house dust and residential soil to children's blood lead levels: A pooled analysis of 12 epidemiologic studies. Environmental Research 1998;79:51-68.
22. Marron R, Lanphear BP, Kouides R, Dudman L, Manchester R, Christy C. Efficacy of informational letters on Hepatitis B immunization rates in university students. J Am College Health 1998;47:123-127.
23. Howard CR, Howard FM, Lanphear BP, deBlieck EA, Eberly S, Lawrence RA. Effect of early pacifier use on breastfeeding duration. Pediatrics 1999;103. http//www.pediatrics.org/cgi/contents/full103/3/e33.
24. Lanphear BP, Howard CR, Eberly S, Auinger P, Kolassa J, Weitzman M, Alexander K, Schaffer S. Primary prevention of childhood lead exposure: A randomized trial of dust control. Pediatrics 1999;103:772-777.

25. Moss ME, Lanphear BP, Auinger P. Association of dental caries and blood lead levels among the U.S. population. JAMA 1999;281:2294-2298.

26. Howard CR, de Blieck EA, ten Hoopen C, Howard F, Lanphear BP, Lawrence RA. Physiologic stability of newborns during cup and bottle-feeding. Pediatrics 1999;104:1204-1207.

27. Steiner JF, Curtis P, Lanphear BP, Vu KO, Reid A. Federally-funded fellowship training in primary care research: perspectives of program directors. Academic Medicine 2000;75:74-80.

28. Stone KE, Lanphear BP, Pomerantz WJ, Khoury J. Injuries and deaths in children due to window-falls. Journal of Urban Health 2000;77:26-33.

29. Mansour M, Lanphear BP, DeWitt TG. Barriers to asthma care among underserved, urban children. Pediatrics 2000;106:512-519.

30. Lanphear BP, Eberly S, Howard CR. Long-term effect of dust control on children's blood lead levels. Pediatrics 2000 http//www.pediatrics.org/cgi/contents/full106/4/e48.

31. Lanphear BP, Dietrich KN, Auinger P, Cox C. Cognitive deficits associated with blood lead levels <10 μg/dl in U.S. children and adolescents. Public Health Reports 2000;115:521-529.

32. Lanphear BP, Aligne C, Auinger P, Byrd R, Weitzman M. Residential exposures associated with asthma in U.S. children. Pediatrics 2001;107:505-511.

33. Lanphear BP, Kahn RS, Berger O, Auinger P, Bortnick S, Naahas R. Contribution of residential exposures to asthma in US children and adolescents. Pediatrics 2001. http://www.pediatrics.org/cgi/content/full/107/6/e98.

34. Phelan KJ, Kalkwarf H, Khoury J, Lanphear BP. Trends and patterns of playground injuries in U.S. children and adolescents. Ambulatory Pediatrics 2001;1:227-233.

35. Mansour M, Lanphear BP, Hornung R, Khoury J, Bernstein DI, Menrath W, Decolongon J. A side-by-side comparison of sampling methods for settled indoor allergens. Environ Res 2001;87:37-46.

36. Haynes E, Lanphear BP, Tohn E, Farr N, Rhoads GG. The effect of dust control on children's blood lead concentrations: A systematic review. Env Health Perspect 2002;110:103-107.

37. Lanphear BP, Hornung R, Ho M, Howard CR, Eberly S, Knauf K. Environmental lead exposure during early childhood. Journal of Pediatrics 2002;140:40-47.

38. Steiner JF, Lanphear BP, Curtis P, Vu KO. The training and career paths of fellows in the National Research Service Award (NRSA) program for research in primary medical care. Acad Med 2002;77:712-718.

39. Steiner JF, Lanphear BP, Curtis P, Vu KO. Indicators of early research productivity among primary care fellows. J Gen Intern Med 2002;17:845-851.

40. Kalkwarf HJ, Khoury JC, Lanphear BP. Milk intake in childhood and adolescence, adult bone density and osteoporotic fractures in U.S. women. Am J Clin Nut 2003;77:257-265.

41. Howard CR, Howard FM, Lanphear BP, Eberly S, deBlieck EA, Oakes D, Lawrence RA. A randomized trial of pacifier use and bottle-feeding and their effect on breastfeeding. Pediatrics 2003;111:511-518.

42. Curtis P, Dickinson P, Steiner J, Lanphear BP, Vu K. Building capacity for research in family medicine: Is the blueprint faulty? Family Medicine 2003;35:124-130.

43. Lanphear BP, Succop P, Roda S, Henningsen G. The effect of soil abatement on blood lead levels in children living near a former smelting and milling operation. Public Health Reports 2003;118:83-90.

44. Canfield RL, Henderson CR, Cory-Slechta DA, Cox C, Jusko TA, Lanphear BP. Intellectual impairment in children with blood lead concentrations below 10 micrograms per deciliter. N Engl J Med 2003;348:1517-1526.

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48. Steiner JF, Curtis P, Lanphear BP, Vu KO, Main DS. Assessing the role of influential mentors in the research development of primary care fellows. Academic Medicine 2004:79:865-872.

49. Yolton K, Auinger P, Dietrich KN, Lanphear BP, Hornung R. Exposure to environmental tobacco smoke and cognitive abilities among US children and adolescents. Environ Health Persp 2005:113:98-103.

50. Phelan KJ, Khoury J, Kalkwarf H, Lanphear BP. Residential injuries in US children and adolescents. Public Health Reports 2005;120:63-70.

51. Wilson SE, Kahn RS, Khoury J, Lanphear BP. Racial differences in exposure to environmental tobacco smoke among children. Environ Health Perspect 2005;113:362-367.

52. Lanphear BP, Hornung R, Ho M. Screening housing to prevent lead toxicity in children. Pub Health Rep 2005;120:305-310.

53. Rosner D, Markowitz G, Lanphear BP. J. Lockhart Gibson and the discovery of the impact of lead pigments on children's health: A review of a century of knowledge. Pub Health Rep 2005;120:296-300.

54. Lanphear BP, Hornung R, Khoury J, Yolton K, Baghurst P, Bellinger DC, Canfield RL, Dietrich KN, Bornschein R, Greene T, Rothenberg SJ, Needleman HL, Schnaas L, Wasserman G, Graziano J. Low-level environmental lead exposure and children's intellectual function: An international pooled analysis. Environ Health Perspect 2005;113:894-899.
55. Nagaraja J, Menkedick J, Phelan KJ, Ashley PJ, Zhang J, Lanphear BP. Deaths from residential injuries in US children and adolescents, 1985 to 1997. Pediatrics 2005;116:454-61.
56. Eskenazi B, Gladstone EA, Berkowitz GS, Drew CH, Faustman EM, Holland NT, Lanphear BP, Meisel SJ, Perera FP, Rauh VA, Sweeney A, Whyatt RM, Yolton K. Methodologic and logistic issues in conducting longitudinal birth cohort studies: Lessons learned from the Centers for Children's Environmental Health and Disease Prevention Research. Environ Health Persp 2005;113:1419-1429.
57. Eggleston PA, Diette G, Lipsett M, Lewis T, Tager I, McConnell R, Chrischilles E, Lanphear BP, Miller R, Krishnan J. Lessons learned for the study of childhood asthma from the Centers for Children's Environmental Health and Disease Prevention Research. Environ Health Persp 2005;113:1430-1436.
58. Spanier AJ, Hornung R, Lierl M, Lanphear BP. Environmental exposures and exhaled nitric oxide in children with asthma. J Pediatrics 2006;149:220-226.
59. Yuan W, Holland S, Cecil K, Dietrich K, Wessel SD, Altaye M, Hornung R, Ris M, Egelhoff J, Lanphear B. The impact of early childhood lead exposure on brain organization: A functional magnetic resonance imaging study of language function. Pediatrics 2006;118:971-977.
60. Howard CR, Lanphear N, Lanphear BP, Eberly S, Lawrence RA. Parental responses to infant crying and colic: the effect on breastfeeding duration. Breastfeeding Med 2006;1:146-155.
61. Brinkman W, Geraghty S, Lanphear B, Khoury J, Gonzalez-Del-Rey J, DeWitt T, Britto M. Evaluation of resident communication skills and professionalism: A matter of perspective? Pediatrics 2006;118:1371-1379.
62. Braun JM, Kahn RS, Froehlich T, Auinger P, Lanphear BP. Exposures to environmental toxicants and attention deficit hyperactivity disorder in U.S. children. Environ Health Perspect 2006;114:1904-1909.
63. Brinkman WB, Geraghty SR, Lanphear BP, Khoury JC, Gonzalez-Del-Rey JA, DeWitt TG, Britto MT. Effect of multisource feedback on resident communication skills and professionalism: a randomized controlled trial. Arch Pediatr Adolesc Med 2007;161:44-49.
64. Wilson SE, Kahn RS, Khoury J, Lanphear BP. The role of air nicotine in explaining racial differences in cotinine among tobacco-exposed children. Chest 2007;131:856-862.

65. Froehlich T, Lanphear BP, Dietrich KN, Cory-Slechta D, Wang N, Kahn RS. Interactive effects of a DRD4 polymorphism, lead, and sex on executive functions in children. Biol Psychiatry 2007:62:243-249.

66. Froehlich TE, Lanphear BP, Epstein JN, Barbaresi WJ, Katusic SK, Kahn RS. Prevalence, recognition and treatment of attention-deficit/hyperactivity disorder in a national sample of US children. Arch Pediatr Adolesc Med 2007;161:857-864.

67. Landrigan PJ, Woolf AD, Gitterman B, Lanphear B, Forman J, Karr C, Moshier EL, Godbold J, Crain E. The Ambulatory Pediatric Association Fellowship in Pediatric Environmental Health: A five-year assessment. EHP 2007;115:1383-1387.

68. Jusko TA, Henderson CR, Lanphear BP, Cory-Slechta DA, Parsons PJ, Canfield RL. Blood lead concentrations < 10 µg/dL and child intelligence at 6 years of age. Environ Health Perspect 2008;116:243-248.

69. Cecil KM, Brubaker CJ, Adler CM, Dietrich KN, Altaye M, Egelhoff JC, Wessel S, Elangovan I, Hornung R, Jarvis K, Lanphear BP. Decreased brain volume in adults with childhood lead exposure. PLoS Med 2008;5:e112.

70. Wright JP, Dietrich KN, Ris MD, Hornung RW, Wessel SD, Lanphear BP, Ho M, Rae MN. Association of prenatal and childhood blood lead concentrations with criminal arrests in early adulthood. PLoS Med 2008;5:e101.

71. Spanier AJ, Hornung RW, Kahn RS, Lierl MB, Lanphear BP. Seasonal variation and environmental predictors of exhaled nitric oxide in children with asthma. Pediatr Pulmonol 2008;43:576-583.

72. Braun JM, Froehlich TE, Daniels JL, Dietrich KN, Hornung R, Auinger P, Lanphear BP. Association of environmental toxicants and conduct disorder in U.S. children: NHANES 2001-2004. Environ Health Perspect 2008;116:956-962.

73. Yolton K, Khoury J, Hornung R, Dietrich KN, Succop P, Lanphear BP. Environmental tobacco smoke exposure and child behaviors. J Dev Behav Pediatr 2008;29:456-463.

74. Yeoh B, Woolfenden S, Wheeler D, Alperstein G, Lanphear B. Household interventions for prevention of domestic lead exposure in children. Cochrane Database Syst Rev. 2008;2:CD006047.

75. Geraghty SR, Khoury JC, Morrow AL, Lanphear BP. Reporting individual test results of environmental chemicals in breastmilk: potential for premature weaning. Breastfeed Med. 2008;3:207-213.

76. Spanier AJ, Kahn RS, Hornung RW, Wang N, Sun G, Lierl MB, Lanphear BP. Environmental exposures, nitric oxide synthase genes, and exhaled nitric oxide in asthmatic children. Pediatr Pulmonol. 2009;44:812-819.

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78. Phelan KJ, Khoury J, Xu Y, Lanphear BP. Validation of a HOME Injury Survey. Injury Prevention 2009:15:300-306.

79. Braun J, Yolton K, Dietrich K, Hornung R, Ye X, Calafat AM, Lanphear BP. Prenatal Bisphenol A exposure and early childhood behavior. Environ Health Persp 2009;117:1945-1952.

80. Yolton K, Khoury J, Xu Y, Succop P, Lanphear B, Bernert JT, Lester B. Low-level prenatal exposure to nicotine and infant neurobehavior. Neurotoxicol Teratol. 2009;31:356-363.

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2. “Common Misconceptions about Tuberculosis”. American Lung Association, St. Elizabeth’s Hospital, Belleville, IL, March 19, 1991.
3. “Prevention and Control of Infectious Disease in Health Care Workers”. Miami Valley Hospital, Dayton, OH, September 5, 1991.

4. “Transmission of Hepatitis B Virus Infection in Health Care Workers”. Ohio University, Athens, Ohio, March 21, 1992.
5. “Universal Immunization Against Hepatitis B Virus”. Grand Rounds, Dayton Children’s Hospital, May 1992, Dayton, Ohio.
6. "Correlation of Blood Lead Levels and Dust Lead Levels Using Three Dust Collection Methods. Environmental Protection Agency, Research Triangle, N.C., January 20,1994.
7. "Relation of Lead-Contaminated House Dust and Blood Lead Levels in Urban Children" Environmental Protection Agency, Washington, D.C., February, 1994.
8. "Lead-Contaminated House Dust and Blood Lead Concentrations in Children", Society for Pediatric Research, Seattle, Washington May 5, 1994.
9. "EPA Health-Based Standards for Soil and Dust". Alliance to End Childhood Lead Poisoning, Washington, D.C., May 17, 1994.
10. "Epidemiology of Tuberculosis in Health Care Settings". University of Cincinnati, Cincinnati, OH, August 19, 1994.
11. "A Side-by-Side Comparison of Sampling Methods for Lead-Contaminated House Dust". American Public Health Association, Washington, D.C., November 1, 1994.
12. “Trends in Childhood Exposure to Lead: Implications for Prevention”. University of Rochester, Pediatric Grand Rounds, February 15, 1995.
13. “Childhood Exposure to Lead”. Visiting Professor, Nazareth College, Rochester, New York, March 24, 1995.
14. “Transmission and Control of Infections in Health Care Workers”. (Moderator & Speaker) American College of Occupational Environmental Medicine, Las Vegas, Nevada, May 4, 1995.
15. “Lead Exposure Prevention Research at the University of Rochester”. New England Lead Conference, Kennebunkport, Maine, August 3, 1995.
16. “Prevention of Childhood Lead Exposure”. 1st Annual Midwest Conference on Childhood Lead Poisoning Prevention, Kansas City, MO, September 10-11, 1995.
17. “Childhood Lead Exposure: Implications for Occupational Health”. National Institute for Occupational Safety and Health, Cincinnati, OH, May 10, 1996.
18. “Community Characteristics and Children’s Blood Lead Concentrations”. American Public Health Association, New York City, NY, November 19, 1996.
19. “Evolution of a Disease: The Science of Childhood Lead Exposure Prevention.” American Public Health Association, New York City, NY, November 18, 1996.
20. “Childhood Lead Exposure: A Local and National Perspective.” Occupational Medicine Grand Rounds, University of Rochester, January 2, 1997.

21. “Prevention of Childhood Lead Exposure: The U.S. Experience”. (Keynote) University of the West Indies and Pan American Health Organization, Kingston, Jamaica, January 23, 1997
22. “Lead-Contaminated House Dust and Children’s Blood Lead Levels”. (Keynote Presentation) Look Out for Lead Conference, Madison, WI, May 22, 1997.
23. “Primary Prevention of Childhood Lead Exposure: A Randomized Trial of Dust Control”. American Public Health Association, Indianapolis, November 13, 1997.
24. “Evolution of a Disease: Prevention of Childhood Lead Exposure.” Pediatric Grand Rounds, Medical University of South Carolina, Charleston, SC, March 20, 1998.
25. “The Science of Childhood Lead Exposure Prevention.” Tulane/Xavier Center for Bioenvironmental Research, New Orleans, May 4-5th, 1998.
26. “Lead Hazard Control Research” Conference on Linking Health, Housing & Environment, Centers for Disease Control, Department of Housing and Urban Development, National Institutes of Health, Phoenix, Arizona, June 21-24, 1998.
27. “A Randomized Trial of Dust Control to Prevent Childhood Lead Exposure.” Presenter and Co-chairman, Section on Heavy Metals, 1st International Conference on Children’s Environmental Health, Amsterdam, The Netherlands, August 11-13th, 1998.
28. “Prevention of Childhood Lead Exposure: A Critique of the EPA’s Proposed Residential Lead Standard”. Office of Children’s Health Protection, U.S. Environmental Protection Agency, Washington, D.C., November 5, 1998.
29. “Science and Policy of Lead Poisoning Prevention in the United States”. Nicholas School of the Environment, Duke University, Durham, North Carolina, February 22, 1999.
30. “Behaviors in Early Childhood and Exposure to Environmental Toxins”. (invited) Pediatric Environmental Health Conference, San Francisco, CA May 4, 1999.
31. “Patterns of Lead Exposure in Early Childhood”. International Conference on Lead Exposure, Reproductive Toxicity and Carcinogenicity, Gargnano, Italy, May 7, 1999.
32. “Adverse Effects of Blood Lead Concentrations <10 μg/dL” (Invited), 17th International Conference Neurotoxicology Conference, Little Rock, Arkansas, October 17-20, 1999.
33. “Emerging Research and Implications for Prevention of Childhood Lead Exposure” (Invited), 2nd Annual Syracuse Lead Conference, Syracuse, New York October 27th, 1999.
34. “Prevention of Lead Poisoning in Children” Sierra Club, Omaha, NE, November 16th, 1999.
35. “Children’s Environmental Health: A Focus on Residential Hazards” Department of Pediatrics, University of Nebraska Hospital, November 17th, 1999.
36. “Effectiveness of Lead Hazard Controls”, New England Lead Conference, New Hampshire, Tufts University School of Medicine, April 25, 2000.

37. “Subclinical Lead Toxicity in U.S. Children and Adolescents”, Pediatric Academic Societies, Boston, MA, May 15, 2000.
38. “Contribution of Residential Exposures to Asthma in U.S. Children and Adolescents”, Pediatric Academic Societies, Boston, MA, May 16, 2000.
39. “The Effect of Soil Abatement on Blood Lead Concentration in Children living near a former Smelter and Milling Operation” (invited). Coeur d’Alene, Idaho, May 24, 2000.
40. “The Paradox of Lead Poisoning Prevention” (invited). National Institute of Justice, Washington, D.C., July 18th, 2000.
41. “Evolution of a Disease: Prevention of Childhood Lead Exposure.” Pediatric Grand Rounds, Children’s Hospital Medical Center, Cincinnati, Ohio, August 22, 2000.
42. “Children’s Environmental Health: A Focus on Residential Hazards” Pediatric Grand Rounds, Department of Pediatrics, University of Rochester School of Medicine, Rochester, NY, September 20th, 2000.
43. “Prevention of Lead Poisoning in Childhood” 7th Annual Childhood New York State Lead Poisoning Prevention Conference, Purchase College, NY, September 29, 2000.
44. “Excavating the Enigmas of Childhood Lead Exposure”. Department of Environmental and Occupational Medicine, Harvard University School of Public Health, Boston, MA, October 16th, 2000.
45. “Contribution of Residential Exposures to Asthma”. Eliminating Childhood Lead Poisoning: Our Challenge for the Decade, Centers for Disease Control and the U.S. Department of Housing & Urban Development, December 11th, 2000.
46. “Setting Research Priorities for the Decade”. (Moderator & Speaker) Eliminating Childhood Lead Poisoning: Our Challenge for the Decade, Centers for Disease Control and the U.S. Department of Housing & Urban Development, December 13th, 2000.
47. “Evolution of a Disease: Prevention of Childhood Lead Exposure.” (Keynote Presentation) Look Out for Lead Conference, Madison, WI, April 12, 2001.
48. “Environmental Lead Exposure and Children’s Intelligence at Blood Lead Concentrations below 10 μg/dl.” APA Presidential Plenary Session, Pediatric Academic Society Meeting, Baltimore, MD, April 30, 2001.
49. “Elimination of Childhood Lead Exposure: Obstacles & Opportunities” (Plenary). National Housing Conference and Exposition, New Orleans, LA, May 16th, 2001.
50. “Prevention of Childhood Lead Exposure: A Public Health Perspective” (Keynote Presentation). Philadelphia Health Department, Philadelphia, PA, May 23rd, 2001.

51. "Evolution of a Disease: Prevention of Childhood Lead Exposure." (Keynote Presentation), Charles Drew University, Los Angeles, California, October 22nd, 2001.
52. "Primary Prevention of Childhood Lead Exposure" (Keynote Presentation), Midwest Regional Lead Conference, Pittsburgh PA, October 29th, 2001.
53. "Prevention of Childhood Lead Exposure: Shifting to Primary Prevention" (Keynote Presentation), Indiana Department of Health, Lead-Safe Conference, November 7th, 2001.
54. "A Strategy for Primary Prevention of Childhood Lead Exposure" A testimony to Housing and Transportation Subcommittee, U.S. Senate, Washington, D.C., November 13, 2001.
55. "Ethical issues of Environmental Research involving Children" (moderator and speaker). Panelists were Jeffrey Kahn, Ph.D., and Leonard Glantz, J.D., Raleigh-Durham, North Carolina, NIEHS Conference of Children's Environmental Health Centers, January 23, 2001.
56. "Evolution of a Disease: Science and Prevention of Childhood Lead Exposure." Grand Rounds, Omaha Children's Hospital, Omaha, Nebraska, March 1, 2002.
57. "Racial Disparities in Children due to Environmental Hazards" Ohio Commission on Minority Health, Columbus, Ohio March 27, 2002.
58. "Prevention of Childhood Lead Exposure in a Former Mining Community" Tar Creek, Oklahoma, April 4, 2002.
59. "Evolution of a Disease: Science and Prevention of Childhood Lead Exposure." Grand Rounds, Hasbro Children's Hospital, Brown University, Providence Rhode Island, May 17, 2002.
60. "Evolution of a Disease: Science and Prevention of Childhood Lead Exposure." Grand Rounds, Dayton Children's Hospital, Wright University, Dayton, Ohio May 22, 2002.
61. "Evolution of a Disease: Science and Prevention of Childhood Lead Exposure." International Lead Congress, Washington, DC, June 3rd, 2002.
62. "Residential Hazards: A Neglected Health Problem" Agency for Toxic Substances Disease Registry, Centers for Disease Control and Prevention, Atlanta, Georgia, August 19th, 2002.
63. "Control of Residential Exposures to Environmental Neurotoxins" National Center for Healthy Homes (Moderator and Speaker), Annapolis, VA, November 7th, 2003.
64. "The Promises and Potential Pitfalls of Primary Lead Poisoning Prevention" Purchase College, 9th Annual Childhood New York State Lead Poisoning Prevention Conference, Purchase College, New York,, October 4th, 2002.
65. "Evolution of a Disease: the Science and Prevention of Childhood Lead Exposure." Pediatric Grand Rounds, Syracuse, NY, October 9th, 2002.
66. "Evolution of a Disease: the Science and Prevention of Childhood Lead Exposure." University of Texas at El Paso, El Paso, Texas January 29th, 2003.

67. "Childhood Lead Poisoning" Introduction to Children's Environmental Health, Seattle, Washington, Pediatric Academic Society, May 3rd, 2003.
68. "The Legacy of Lead: Childhood Lead Poisoning in the 21st Century". Chicago Lead Summit, Chicago, Illinois, May 28th, 2003.
69. "The Legacy of Lead: Childhood Lead Poisoning in the 21st Century". Case Western Reserve University, Cleveland, Ohio, June 3rd, 2003.
70. "Housing and Children's Health", Sprawl: The impact on vulnerable populations, University of Cincinnati College of Medicine, Cincinnati, Ohio, July 8th, 2003.
71. "Trials and Tribulations of Protecting Children from Environmental Toxins". Duke University, Nicholas School of the Environment, Durham, NC, November 6th, 2003.
72. "Adverse Effects of Fetal and Childhood Exposures to Prevalent Toxins" Midwest Critical Regional Neonatology Conference, Covington, KY, November 14th, 2003.
73. "Control of Residential Hazards in Children" American Public Health Association, San Francisco, CA, November 18th, 2003.
74. "Low-Level Exposure to Environmental Lead Exposure and Children's Intellectual Function: An International Pooled Analysis". 21st International Neurotoxicology Conference, Honolulu, Hawaii, February 11th, 2004.
75. "Trials and Tribulations of Protecting Children from Environmental Hazards" Workshop on Ethical Issues on Children's Environmental Health, Children's Environmental Health Network, Washington, D.C. March 5, 2004.
76. "Low-Level Exposure to Environmental Lead Exposure and Children's Intellectual Function: An International Pooled Analysis", Pediatric Academic Societies Annual Meeting. Pediatric Research 2004;55:163A.
77. "The Impact of the Environment on Children's Health" Bob Smith Endowed Lecture, Department of Pediatrics, First Gulf Coast Children's Environmental Health Symposium, Baylor University, Houston, Texas.
78. "The Search for Environmental Causes of Learning Disabilities, Learning Disabilities Initiative, Baltimore, MD, May 18th, 2004.
79. "Residential Hazards in Children: A Neglected Public Health Problem", Pediatric Grand Rounds, Boston Medical Center, Department of Pediatrics, Boston University Medical Center, Boston, MA, May 20th, 2004.
80. "Residential Hazards in Children" "Healthier Homes, Stronger Families: Public Policy Approaches to Healthy Housing", National Center for Healthy Housing, Washington, D.C., June 2nd, 2004.

81. "Fetal and Early Childhood Exposures to Prevalent Toxins" Pediatric Grand Rounds, Ste. Justine Children's Hospital, University of Montréal, Montreal, Canada, June 16th, 2004.
82. "Childhood Exposure to Lead-Contaminated Soil: A Problem of the Past or a Problem from the Past?" National Academy of Science Committee on Superfund Site Assessment and Remediation in Coeur d'Alene River Basin", June 17th, 2004, Coeur d'Alene, Idaho.
83. "The Legacy of Lead" (Keynote Speaker). Chicago Lead Summit, Region V EPA Headquarters, September 15th, 2004.
84. "A Tale of Two Toxins: Children's Exposure to Tobacco and Lead" (with Michael Weitzman), The American Academy of Pediatrics, San Francisco, CA, October 10th, 2004.
85. "A Legacy of Childhood Lead Poisoning" University of Washington, Seattle, Washington, October 30, 2004.
86. "Protecting Children from Environmental Toxins", Pediatric Grand Rounds, Seattle Children's Hospital, Seattle Washington, March 10th, 2005.
87. "The Science and Politics of Childhood Lead Poisoning", Northwest Pediatric Environmental Health Conference, University of Washington, Seattle, Washington, March 11th, 2005.
88. "The Effects of Low-level Exposure to Environmental Toxins during Fetal Development and Early Childhood", Children's' Hospital of Fudan University, Shanghai International Pediatric Forum, Shanghai, China, June 16th to 18th, 2005.
89. "The Role of Biomarkers in Revealing Genetic and Environmental Influences of Disease and Disability" Psychiatry Grand Rounds, University of Cincinnati, February 8th, 2006.
90. "Trials and Tribulations of Protecting Children from Environmental Hazards: Ethical Issues", Johns Hopkins University of Medicine, March 17th, 2006.
91. "Key Elements of a Primary Prevention Strategy for Lead Poisoning", Albany Law School, Union University, Albany, New York, March 16th, 2006.
92. "Low-Level Lead Toxicity: The Ongoing Search for a Threshold"", Case Western Reserve University, City Club of Cleveland, Cleveland, OH March 4th, 2006.
93. Integrating Genetic and Environmental Influences in Pediatric Research" (Moderator and Speaker), Pediatric Academic Societies, San Francisco, CA, April 30th 2006.

94. "Ethical Issues in Housing Health Hazard Research Involving Children" (Topic Symposia) Pediatric Academic Societies, San Francisco, CA, May 2nd 2006.
95. "Low-Level Lead Toxicity: The Ongoing Search for a Threshold"", International Workshop on Neurotoxic metals: from Research to Prevention, University of Brescia, Italy, June 17th, 2006.
96. "Efficacy of HEPA-CPZ Air Cleaners on Unscheduled Asthma Visits and Asthma Symptoms", International Society for Environmental Epidemiology, Paris France, September 6th, 2006.
97. "Protecting Children from Environmental Toxins", Region VIII Children's Environmental Health Summit, Vail, Colorado September 20th, 2006.
98. "Integrating Genetic and Environmental Biomarkers in Pediatric Epidemiology", Visiting Professor, Simon Fraser University and University of British Columbia, Vancouver, British Columbia, October 19th-20th, 2006.
99. "The Legacy of Lead", Indiana Lead Conference, Indianapolis, Indiana, October 24, 2006.
100. "Ethical dilemmas in Children's Environmental Health", Seminar Series in Ethics of Toxicology, University of Champagne-Urbana, Champagne, Illinois, November 19th, 2006.
101. "Low-Level Lead Toxicity: Implications for Prevention"", WHO Informal Workshop on Lead, University of Munich, Germany, November 30th, 2006.
102. "Low-Level Lead Toxicity: The Ongoing Search for a Threshold"", National Environmental Public Health Conference, National Centers for Disease Control, Atlanta, Georgia, December 4th, 2006.
103. "The Epidemiologic Conquest of Childhood Lead Toxicity: A Pyrrhic Victory". NIEHS Workshop on Children's Environmental Health Research: Past, Present and Future. January 22nd, 2007.
104. "Linking Low-level Exposures to Environmental Toxicants with ADHD". Duke Integrated Toxicology and Environmental Health Program Symposium on Developmental Neurobehavioral Disabilities and Toxic Exposures, March 23, 2007, Durham, North Carolina.
105. "Using Biomarkers to Link Environmental Influences with Disease and Disability", The Channing Laboratory, Harvard University, Boston, Massachusetts, April 4th, 2007.
106. "The Lingering Legacy of Lead Toxicity". Grand Rounds, Department of Pediatrics, St. Louis Children's Hospital, St. Louis University, St. Louis, Missouri, April 11th, 2007.
107. "Protecting Children from Environmental Toxicants", United States Council of Catholic Bishops, Washington, D.C., April 30th, 2007.
108. "Efficacy of HEPA-CPZ Air Cleaners on Unscheduled Asthma Visits and Asthma Symptoms", Pediatric Academic Societies, APA Presidential Platform Plenary Session, Toronto, Canada, May 7th, 2007.

109. “The Lingering Legacy of Lead Toxicity” Grand Rounds, Department of Pediatrics, Omaha Children’s Hospital, University of Nebraska, Omaha, Nebraska, April 11th, 2007.
110. “Linking Low-level Neurotoxicant Exposures of the Developing Brain to Learning and Behavioral Problems.” International Conference on Developmental Programming and Effects of Environmental Toxicants in Human Health and Disease, Faroe Islands, May 20th, 2007.
111. “Protecting Children from Environmental Toxicants: The Neglected Legacy of Rachel Carson”, National Policy Consultation Series on Children’s Health and Environment, Moncton, New Brunswick, Canada, May 31, 2007.
112. “Low-Level Toxicity of Environmental Toxicants: Much Ado about Nothing?” Occupational and Environmental Health Seminar Series, Health Canada, Ottawa, Canada, June 6th, 2007.
113. “Linking Low-Level Lead Exposure with Child and Adolescent Psychopathology”, 13th Annual International Society for Research in Child and Adolescent Psychopathology, London, England, June 19th, 2007.
114. “The Legacy of Lead Toxicity”. Pediatric Grand Rounds, New York Presbyterian Hospital-Weill Cornell Medical Center, September 18th, 2007.
115. “Protecting Children from Environmental Toxicants: The Neglected Legacy of Rachel Carson”. Pediatric Grand Rounds, Children’s Hospital at Dartmouth, Dartmouth Medical School, September 19th, 2007.
116. “The Legacy of Lead Toxicity: Effects of Childhood Lead Exposure in Children, Adolescents and Adults”. Mid-America Conference, Philadelphia, Pennsylvania, October 4th, 2007.
117. “Low-Level Toxicity of Environmental Toxicants: Much Ado about Nothing?” International Society for Exposure Analysis (invited plenary session), Raleigh-Durham, North Carolina, October 17th, 2007.
118. The Global Elimination of Lead Toxicity: A Focus on Housing.” National Institute of Public Health, Rennes, France, October 22nd, 2007.
119. Linkage of Environmental Lead Exposure with Psychopathology in Children and Adolescents” Ramazzini Collegium, Carpi, Italy, October 25th, 2007.
120. “Linking Exposures to Environmental Toxicants with Child and Adolescent Psychopathology”, Symposium on Environmental Toxicity and the Brain, University of Toronto, Toronto, Canada, December 7th, 2007.
121. “Linking Exposures to Environmental Toxicants with Child and Adolescent Psychopathology.” Pediatric Grand Rounds, Rochester General Hospital and Strong Memorial Hospital, Rochester, New York, April 1&2, 2008.

122. "Rochester's Role in the Ongoing Elimination of Childhood Lead Toxicity." Beaven Lecture, Rochester Academy of Medicine, Rochester, New York, April 1, 2008.
123. "The Lingering Legacy of Lead Toxicity: Lansing Legacy." Michigan's Conference for Lead Safe & Healthy Homes, East Lansing, MI, April 22, 2008.
124. First Annual Controversies in Pediatric Environmental Health, "Should the Centers for Disease Control Lower the Blood Lead Level of Concern". A debate by Bruce Lanphear and George G. Rhoads (James Sargent, Moderator). Pediatric Academic Societies Meeting, Honolulu, Hawaii, May 2nd, 2008.
125. "Linking Exposure to Environmental Toxicants with Psychopathology in Children and Youth". Visiting Professor, Alberta Child and Youth Network, Calgary Children's Hospital, Calgary, Alberta. May 13th-15th, 2008.
126. Lead Toxicity and the Teenage Brain", Youth Exploring Science Program, St. Louis Science Center, St. Louis, Missouri, June 30th, 2008.
127. "The Legacy of Childhood Lead Toxicity". Health Canada, Ottawa, Canada, October 6th, 2008.
128. "Protecting Children from Environmental Toxicants: The Neglected Legacy of Rachel Carson". The 2008 Rachel Carson Legacy Conference: Green Chemistry – Solutions for a Healthy Economy, Duquesne University, Pittsburgh, Pennsylvania, September 20th, 2008.
129. "Trials and Tribulations of Protecting Children from Environmental Hazards", Ethics in Toxicology Seminar Series, University of Champagne-Urbana, Champagne, Illinois, September 22nd, 2008.
130. "Industry's Influence on the Prevention of Childhood Lead Poisoning." In: Symposia on Insulating Environmental Health Research from Conflicting Interests. International Society for Environmental Epidemiology Annual Meeting, Pasadena, California, October 14th, 2008.
131. "The Lingering Legacy of Lead Toxicity: Implications for Research and Policy on Other Environmental Toxicants". (Keynote Presentation) BC Environmental and Occupational Health Research Network, Vancouver, BC, November 7th, 2008.
132. "Effects of Environmental Toxicants on Children's Development". DB-PREP Course, American Academy of Pediatrics, Atlanta, Georgia, December 5th, 2008.
133. "Linking Low-level Environmental Toxicants with New Morbidities of Childhood". BC Children's Grand Rounds, British Columbia, Vancouver, February 6th, 2009.
134. "Using Biomarkers to Link Exposures with Disease and Disability in Children". Workshop on Physical and Chemical Exposures in Canadian Cohort Studies, Canadian Institute of Health Research and Health Canada, February 8th-9th, 2009.

135. "How Dangerous Is Lead In Drinking Water?" An interview on "Around The Water Cooler" with Werner Troesken and Bruce Lanphear. February 18th, 2009.
136. "Linking Environmental Toxicants with ADHD in Children" (invited), Learning Disabilities Association Annual Meeting, February 25th, Salt Lake City, Utah.
137. "The Lingering Legacy of Lead Toxicity", Norfolk Children's Hospital, April 30th, 2009, Norfolk Virginia.
138. Second Annual Controversies in Pediatric Environmental Health Debate, "Should Pediatricians Advise Parents to Feed their Children Organic Foods?" A debate by Joel Forman and Janet Silverstein (Bruce Lanphear, Moderator and Organizer). Pediatric Academic Societies Meeting, Baltimore, MD, May 4th, 2009.
139. "A Pattern of Pathology: The Population Impact of Environmental Toxicants on Health". Workshop on Endocrine Disruptors, Endocrine Society, Washington, DC, June 9th, 2009.
140. "The Quandary of Environmental Contaminants in Human Milk", 25th Anniversary of US Surgeon General's Report on Breastfeeding, Washington, DC, June 13th, 2009.
141. "Linking Exposures to Environmental Toxicants with Learning Problems and Psychopathology in Children." Northwest Conference on Children's Health and Environment, Tukwila, Washington, October 1st, 2009.
142. "The Second Coming of the Sanitarians", Pediatric Grand Rounds, University of California at Davis Children's Hospital, Sacramento, California, October 9th, 2009.
143. "The Second Coming of the Sanitarians", National Institute of Public Health, Rennes, France, November 4th, 2009.
144. "Linking Exposure to Environmental Toxicants with ADHD in Children." Symposium on ADHD. Riyadh, Saudi Arabia, November 7th, 2009.
145. "The Interplay of Genetic and Environmental Influences in Common Conditions of Children." Macquarie University, Department of Geology, Sydney, Australia, November 18th, 2009.
146. "The Lingering Legacy of Lead Toxicity: A Call for the Global Elimination of Lead Exposure." Pacific Basin Consortium Symposium on Environment and Health, Perth, Australia, November 13th, 2009.
147. "The Second Coming of the Sanitarians", SFU President's Lecture, Simon Fraser University, Burnaby, BC, March 4th, 2010.
148. Third Annual Controversies in Pediatric Environmental Health Debate, "Should the American Academy of Pediatrics Sponsor a Ratings Board to Provide Evidence-based Ratings for Media?" A debate by James Sargent and Donald Shifrin (Bruce Lanphear, Moderator and Organizer). Pediatric Academic Societies Meeting, Vancouver, BC, May 2nd, 2010.

149. "Efficacy of Reducing Lead Hazards in Housing on Lead-Contaminated House Dust, Blood Lead Concentration and Intellectual Abilities in Children." Pediatric Academic Societies Meeting, Vancouver, BC May 1st, 2010.
150. "Protecting Children from Environmental Toxicants: The Neglected Legacy of Rachel Carson." Pediatric Grand Rounds, Cornell Weill Medical College, New York, New York. May 25th, 2010.
151. "Excavating the Enigmas of Childhood Lead Toxicity", Guest Lecturer, "Introduction to Toxicology, Harvard School of Public Health, Boston, Massachusetts, October 27th, 2010.
152. "The Conquest of Lead Poisoning: A Pyrrhic Victory", Lead Action Collaborative, New England Carpenters Center, Boston, Massachusetts, October 28th, 2010.
153. "Protecting Children from Environmental Toxicants: The Neglected Legacy of Rachel Carson." Academy of Breastfeeding Medicine, San Francisco, California, October 29th, 2010.
154. "Bisphenol A and Behavior Problems in Children". Eastern Perinatal Conference, Kingston, Ontario, November 10th, 2010.
155. "Low-Level Toxicity of Environmental Toxicants: Much Ado about Nothing?" UBC Statistics Department Seminar, November 18th, 2010.
156. "Protecting Children from Environmental Toxicants." Children's Hospital of Quebec, University of Laval, Quebec City, Quebec, December 17th, 2010.
157. "Low-level Toxicity: Implications for Research and Policy", Joint Talks by C. Arden Pope and Bruce Lanphear, SFU, UBC and UW Annual Occupational and Environmental Health Conference, Semiahmoo, WA January 7th, 2011.
158. "Crime of the Century: Lead Toxicity in the 20th Century", Panel Presentation and Discussion, UC Davis, Sacramento, California April 7th, 2011.
159. Fourth Annual Controversies in Pediatric Environmental Health Debate, "Should Parent Slather their Children with Sunscreen?" A debate with Russell Chesney, MD and Sophie Balk, MD, (Bruce Lanphear, Moderator and Organizer). Pediatric Academic Societies Meeting, Denver, Colorado, May 1st, 2011.
160. The Conquest of Lead Toxicity: A Pyrrhic Victory", Canadian Water Network, Ecole Polytechnique de Montreal, Montreal, Canada, June 9th, 2011.
161. The Contribution of Environmental Influences on Chronic Disease, Canadian Partnership for Health and Environment, Toronto, Canada, June 16th, 2011.
162. "The Second Coming of the Sanitarians", Environmental and Occupational Health Seminar, University of Washington School of Public Health, Seattle, WA, May 12th, 2011.
163. "Crime of the Century: The Failure to Prevent the Lead Pandemic". Sterling Prize in Controversy, Wosk Centre, Simon Fraser University, Vancouver, BC, October 19th, 2011.

164. “Measuring Exposure: The Benefits and Limits of Biomarkers”. Canadian Institute for Human Development, Child and Youth Research, Montreal, Canada, December 6th, 2011.
165. “Rachel Carson: Clarity of Vision”. SFU, UBC and UW Annual Occupational and Environmental Health Conference, Semiahmoo, WA, January 6th, 2012.
166. “The Truth About Toxins: What Parents and Health Professionals Should Know”. Environmental Influences on Neurodevelopment: Translating the Emerging Science into Public Health Policy”. UCLA School of Public Health, Los Angeles, California, January 12th, 2012.
167. “Protecting Children from Environmental Toxicants: The Neglected Legacy of Rachel Carson”. Mattel Children’s Hospital, Los Angeles, California, January 13th, 2012.
168. “Why Should We Share Data?”, Data Sharing Strategies for Environmental Health Workshop, National Institute of Environmental Health Sciences, Research Triangle Park, North Carolina, February 6th and 7th, 2012.
169. “The Science and Prevention of Lead Toxicity” (Keynote Presentation), Forum on Lead Toxicity: A Little is Still Too Much”, Macquarie University, Sydney, Australia, June 5th, 2012
170. “Canada Environmental Health Atlas Knowledge Translation Workshop”, Canadian Public Health Association, Edmonton, Alberta, June 13th, 2012.
171. “First Annual Controversies in Pediatric Environmental Health Debate: Should organophosphate pesticides be reduced or banned?” A debate with Brenda Eskenazi and Bruce Lanphear (Rob McConnell, Moderator). International Society for Environmental Epidemiology, Columbia, SC, August 28th, 2012.
172. “Supralinear Dose-Response Relationship of Environmental Toxicants: Research and Policy Implications.” Moderator and Speaker, with Arden Pope, Roel Vermeulen and Bruce Lanphear. International Society for Environmental Epidemiology, Columbia, SC, August 29th, 2012.
173. Tanya Froehlich and Bruce Lanphear, “ADHD and Environmental Toxicants: Time for Prevention?”, Society for Development and Behavioral Pediatrics, Phoenix, AZ, September 9th, 2012.
174. “The Epidemic of Childhood Disabilities: A Failure to Regulate”. Workshop on Children’s Rights and Corporate Responsibility, Green College, University of British Columbia, Vancouver, BC, October 19th, 2012.
175. “Low-level Toxicity: Much Ado About Nothing?”, Department of Preventive Medicine Seminar, University of Southern , California, Los Angeles, California, October 23rd, 2012.
176. “Reflections on Silent Spring”. (Invited Keynote). International Society for Exposure Sciences, Seattle, Washington, October 28th, 2012.

177. "Randomized Controlled Trials in Children's Environmental Health: Underutilized or Unethical?" The University of Washington Northwest Pediatric Environmental Health Specialty Unit and Center for Child Environmental Health, Seattle, Washington, February 26th, 2013.
178. "Crime of the Century: Our Failure to Prevent the Lead Pandemic". Dali Lana School of Public Health and of School Environment, University of Toronto, Toronto, Ontario, March 26th, 2013.
179. "The Ongoing Search for a Threshold". International Conference of Toxicology, Seoul, Korea, July 1, 2013.
180. "Blood Lead Concentrations and Cardiovascular Mortality in the United States: The NHANES Mortality Follow-up Cohort Study". International Society for Environmental Epidemiology, Basel, Switzerland, August 2, 2013.
181. "The Conquest of Lead Poisoning: A Pyrrhic Victory". Corporations and Global Health Governance. Simon Fraser University, Burnaby, British Columbia. September 17th, 2013.
182. "Striking at the Root: Changing the Narrative on the Causes of Disease". Corporations and Global Health Governance. Simon Fraser University, Burnaby, British Columbia. September 17th, 2013.
183. "Crime of the Century: The Failure to Prevent the Lead Pandemic". Pacific Basin Consortium, East-West Center, Honolulu, Hawaii. September 26, 2013.
184. "Low-level Toxicity: Policy Implications for the 21st Century". Symposium on Policy Implications of Environmental Exposures in the 21st Century. Pacific Basin Consortium, East-West Center, Honolulu, Hawaii. September 27, 2013.
185. "Excavating the Enigmas of Childhood Lead Toxicity". Network for Soil Contamination Research (INSCR), Delhi University, New Delhi, India. October 22nd, 2013.
186. "The Lingering Legacy of Lead Toxicity: A Call for the Global Elimination of Lead Exposure", World Health Organization, New Delhi, India. October 24th, 2013. "The Environmental Health Atlas: A Portal to Discover the Promises of Environmental Health." National Institute of Environmental Health Sciences, Raleigh-Durham, NC, November 10th, 2013.
187. "Protecting Children from Environmental Toxins". Japan Dioxin and Endocrine Disruptors Preventive Action, Tokyo, Japan, November 24th, 2013.
188. "ADHD: A Preventable Epidemic?" Alberta Children's Hospital, Calgary, Alberta, December 16th, 2013.
189. "Little Things Matter: The Impact of Toxins on the Developing Brain". Early Years Conference, Vancouver, British Columbia, January 30th, 2014.
190. "Little Things Matter: The Impact of Toxins on the Developing Brain". Dalhousie University, Halifax, Nova Scotia, March 6th, 2014.

191. "Low-level Toxicity of Environmental Toxins: Much Ado About Nothing?". Dalhousie University, Halifax, Nova Scotia, March 6th, 2014.
192. "The Canadian Environmental Health Atlas: A Portal to Discover the Promises of Environmental Health." School of Occupational and Environmental Health, University of British Columbia, March 28th, 2014.
193. "Little Things Matter: The Impact of Toxins on the Developing Brain". British Columbia Healthy Child Alliance, Vancouver, British Columbia, April 2nd, 2014.
194. "Sixth Annual Controversies in Pediatric Environmental Health Debate, E-Cigarettes: A weapon in the war against tobacco or a threat to tobacco control. (Moderator). Featuring Greg Connelly and James Sargent. Pediatric Academic Societies, Vancouver, May 4th, 2014.
195. "Striking at the Root Causes of Chronic Disease in Children" (Moderator). James Sargent, Joel Bakan and David Kessler, May 5th, 2014.
196. "Little Things Matter: The Impact of Toxins on the Developing Brain" (Keynote). OHKA Healthy Homes Alliance, Omaha, Nebraska, May 15th, 2014.
197. "Excavating environmental risk factors for autism: Suspects and strategies". A workshop on examining a multi-systems approach to autism and the environment: challenges and opportunities for research". Toronto, Ontario, June 23rd-24th, 2014.
198. "Lead Poisoning: Tackling a Global Problem" (Co-Moderator and Speaker). International Society for Environmental Epidemiology, Seattle, Washington, August 25th, 2014.
199. "Interventions to Reduce Exposures to Environmental Hazards in Pregnant Women and Children", (Moderator and Speaker). International Society for Environmental Epidemiology, Seattle, Washington, August 25th, 2014.
200. 3rd Annual ISCHE-Sponsored Debate: Should there be any restrictions on universities or academicians receiving payment from industry or other sources? (Moderator). International Society for Environmental Epidemiology, Seattle, Washington, August 25th, 2014.
201. "Crime of the Century: Our Failure to Prevent the Lead Pandemic", Tulane University School of Public Health and Tropical Medicine, New Orleans, Louisiana, September 5th, 2014.
202. "Environment Matters", Children's Environmental Health Panel. Society for Environmental Journalists, New Orleans, Louisiana, September 6th, 2014.
203. "Insidious Influence of Industry on Science: How Corporations Undermine Science", 5th Annual C. Everett Koop Distinguished Lecture, "Corporate Threats to Children's Health", with Joel Bakan and James Sargent, Dartmouth University, New Hampshire, October 6th, 2014.
204. "Crime of the Century: Our Failure to Prevent the Lead Pandemic", John Rosen Memorial Lecture, Montefiore Medical Center, New York, New York, October 8th, 2014.

205. "Little Things Matter: The Impact of Toxins on the Developing Brain" (Keynote). Prenatal Environmental Health Education (PEHE) Conference, University of Ottawa. Ottawa, Ontario, November 21st, 2014.
206. "Little Things Matter: The Impact of Toxins on the Developing Brain" (Keynote). ISEE Asian Regional Meeting, Shanghai, China, November 30th, 2014.
207. "Crime of the Century: Our Failure to Prevent the Lead Pandemic", John Rosen Memorial Lecture, ISEE Asian Regional Meeting, Shanghai, China, November 31st, 2014.
208. "Data Visualization", with Joe Braun and Allan Just, Pediatric Environmental Health Scholars Retreat, Reston, VA, December 6th, 2014.
209. "Victories in Public Health: Progress or Adaptation?" SFU, UBC and UW Annual Occupational and Environmental Health Conference, Semiahmoo, WA January 8th, 2015.
210. "Food in the Industrial Era: Is Backward the Way Forward?" Children's Environmental Health Network, Austin, Texas, February 4th, 2015.
211. "Excavating the enigmas of childhood lead toxicity". Broken Hill City Council and Lead Reference Group, Broken Hill, New South Wales, Australia, March 3rd, 2015.
212. "Prevention Paradox: Why a Little Lead is Too Much". Unequal Exposure Symposium, Climate Change Research Center, University of New South Wales, March 5th, 2015, Sydney, Australia.
213. "Crime of the Century: Our Failure to Prevent the Lead Pandemic". 10th Annual Break the Cycle Conference, Emory University, Atlanta, Georgia. April 23rd, 2015.
214. "The Staggering Cost of Lead Toxicity and the Unbelievable Benefit of Preventing It". 10th Annual Break the Cycle Conference, Emory University, Atlanta, Georgia. April 24th, 2015.
215. Seventh Annual Controversies in Pediatric Environmental Health Debate, "GMOs: A Hazard or Harvest of Health?" A debate with Joel Forman, MD and Daniel Goldstein, MD, (Bruce Lanphear, Moderator and Organizer). Pediatric Academic Societies Meeting, San Diego, California, April 27th, 2015.
216. "Impact of Dwellings on Child Health", Canadian Green Building Council Conference, Vancouver Convention Center, Vancouver, BC, April 28. 2015.
217. "Impact of Tobacco on the Developing Brain", Developmental Effects of Nicotine and Implications for Emerging Tobacco Products, Rockville, Maryland, May 5thth, 2015.
218. "Impact of Toxins on the Developing Brain" India Tour (Bengaluru, Trivandrum, Kolkata, and Chandigarh) Sponsored by PAN-India, September 4th-11th, 2015.
219. "Impact of Dwellings on Child Health", Green School Summit, Calgary, Alberta, September 25th. 2015.

220. "Prevention Paradox: Why a Little Lead is Too Much", A debate with George Rhoads, Montefiore Medical Center, Tarrytown, October 2nd, 2015.
221. "Crime of the Century: Our Failure to Prevent the Lead Pandemic" (Keynote Presentation), University of Cincinnati Department of Environmental Health 50th Anniversary Gala, Cincinnati, Ohio, October 9th, 2015.
222. "Impact of Toxins on the Developing Brain" (Keynote Presentation) Children's Environmental Health Centers Annual Meeting, Washington, DC, October 31, 2015.
223. "The Impact of Toxins on the Developing Brain: Our Failure to Prevent Brain-based Disorders in Children", National Core for Neuroethics, UBC November 12th, 2015.
224. "Impact of Dwellings on Child Health", Canada Green Building Council, Toronto, ON Green, December 1st, 2015.
225. "The Tortuous Road to Prevention: Are We There Yet", Air Quality and Impacts on Health: Beyond the Heart and the Lungs, The Lung Association of BC, February 28th, 2016.
226. "Lead's Long Shadow: What the Story of Flint, Michigan Means for All of Us", with Bruce Lanphear, Mona Hanna-Attisha and Marc Edwards. Collaborative on Health and the Environment Webinar, March 8th, 2016.
227. "Little Things Matter: The Impact of Toxins on the Developing Brain", Collaborative on Health and Environmental Alaska Working Group Webinar, March 9th, 2016.
228. "Victories in Public Health: Progress or Adaptation?", Symposium Against Indifference, Ashland University, Ashland, Ohio, April 5th, 2016.
229. "Little Things Matter: The Impact of Toxins on the Developing Brain" (Keynote), Children's Environmental Health: New Findings from California Research, Sacramento, California, April 7th, 2016.
230. "Crime of the Century: Our Failure to Prevent the Lead Pandemic", Distinguished Visiting Professor in Health Law, Loyola University, Chicago, Illinois April 21st, 2016.
231. "The Population Impact of Toxins on Intellectual Abilities: Implications for Policy and Prevention", in Symposia on Environmental Toxins and the Brain: Growing Evidence of Risk, Pediatric Academic Societies, Baltimore, MD, May 2nd, 2016.
232. "Data Visualization and Video Production for Public Consumption", in Symposia on Innovative Tools to Enhance Knowledge Translation of Environmental Health: Data Visualization, Videos and Message Mapping, (co-Moderated by Mark Miller and Bruce Lanphear), Pediatric Academic Societies, Baltimore, MD, April 30th, 2016.
233. "Crime of the Century: Our Failure to Prevent the Lead Epidemic", Michigan State University, Flint, MI, May 7th, 2016.

234. "Crime of the Century: Our Failure to Prevent the Lead Epidemic", Johns Hopkins University School of Public Health, Baltimore, MD, May 7th, 2016.
235. "Little Things Matter: The Impact of Toxins on the Developing Brain", Baltimore, MD, International Medical Federation Autism Research (IMFAR), May 8th, 2016.
236. "Public Health Matters: Videos on Toxic Chemicals, Air Pollutants and the Prevention Paradox", Mongolian National University of Medical Sciences, June 23, 2016.
237. "Little Things Matter: The Impact of Toxins on the Developing Brain", USC Annenberg Center for Health Journalism, July 18th, 2016.
238. "Preventing Lead Toxicity", California Environmental Protection Agency, Occupational Environmental Health Hazard Assessment, September 23rd, 2016.
239. "Unleashing the Power of Prevention: Creating Video to Re-Imagine our Approach to Disease," World Issues Forum, Fairhaven College, University of Western Washington, (with Bob Lanphear), November 2, 2016.
240. "Little Things Matter: The Impact of Toxic Chemicals on the Developing Brain", Pediatric Grand Rounds, Maimonides Hospital, November 15th, 2016.
241. "Little Things Matter: The Impact of Toxic Chemicals on the Child Health" (Keynote), Hudson Valley Perinatal Conference, November 16th, 2016.
242. "Little Things Matter: The Impact of Toxins on the Developing Brain", IPEN, San Francisco, CA, November 18th, 2016.
243. "Unleashing the Power of Prevention: Creating Video to Re-Imagine our Approach to Disease", SFU, UBC and UW Annual Occupational and Environmental Health Conference Semiahmoo, WA, January 5th, 2017.
244. "Unleashing the Power of Prevention: Creating Video to Re-Imagine our Approach to Disease", University of New Brunswick, January 25th, 2017.
245. "Little Things Matter: The Impact of Toxic Chemicals on the Developing Brain", New Brunswick Children's Environmental Health Collaborative, January 26th, 2017.
246. "Unleashing the Power of Prevention: Creating Video to Re-Imagine our Approach to Disease", Rockefeller Center, Bellagio, Italy, February 22nd, 2017.
247. "Little Things Matter: The Impact of Toxic Chemicals on the Developing Brain", The Science in Society Speaker Series, Okanagan College, Vernon, BC, April 6th, 2017.
248. "Little Things Matter: The Impact of Toxic Chemicals on the Developing Brain" (invited plenary), Vancouver, British Columbia, Canadian Pediatric Society, June 3rd, 2017.
249. "Unleashing the Power of Prevention: Creating Video to Re-Imagine our Approach to Disease", Macquarie University, Sydney, Australia, September 29th, 2017.

250. “Unleashing the Power of Prevention: Creating Video to Re-Imagine our Approach to Disease”, Brown University, Providence, Rhode Island, October 13th, 2017.
251. Cause or Cure: Does the Relentless Pursuit of a Cure Endanger our Health? University of Alaska, Alaska Tribal Health Consortium, Anchorage, Alaska, November 2nd, 2017.
252. “Little Things Matter: The Impact of Toxic Chemicals on the Developing Brain” (Keynote), All Alaska Pediatric Conference, Anchorage, Alaska, November 3rd, 2017.
253. “Little Things Matter: The Impact of Toxic Chemicals on the Developing Brain”, CINBIOSE 30th Anniversary, University of Quebec at Montreal, Montreal, November 9th, -10th, 2017.
254. “The Legacy of Lead Poisoning: Moving towards Prevention”. East Chicago Community Meeting, Illinois, November 26th, 2017.
255. “Cause or Cure”, NIEHS Environmental Health Seminar, University of Southern California, Los Angeles, California, December 1st, 2017.
256. “Little Things Matter: The Impact of Lead on Brain Development” (Keynote Presentation), Workshop on Lead-Free Schools, Pew Trust, Washington, DC, December 6th-7th, 2017.
257. “Low-level Toxicity of Chemicals: No Acceptable Threshold?” Risk Modeling, Mitigation and Modeling in Health Sciences, Centre de Recherches Mathematiques, Montreal, QC, December 11th, 2017.
258. “Little Things Matter: The Impact of Toxic Chemicals on the Developing Brain”, Department of Psychology and Neuroscience, York University, Toronto, ON, December 13th, 2017.
259. “The impact of Pollutants on Human Health: No Safe Levels?”, Center for Energy and Environmental Contaminants, Macquarie University, Sydney, Australia, February 13th, 2018.
260. “Cause or Cure: Does the Relentless Pursuit of a Cure Endanger our Children’s Health?”, Department of Pediatrics, University of Wisconsin at Madison School of Medicine, Madison, Wisconsin, March 1st, 2018.
261. “Little Things Matter: The Impact of Toxic Chemicals on the Developing Brain”, Wisconsin Environmental Health Network, Madison, Wisconsin, March 2nd, 2018.
262. “Little Things Matter: The Impact of Toxic Chemicals on the Developing Brain”, Biennial Atlantic Symposium on Learning Disabilities Association, Fredericton, NB.
263. “Crime of the Century: The Failure to Prevent the Lead Pandemic” (Keynote). 11th UK and Ireland Environmental and Occupational Epidemiology, John Snow Lecture Hall, London School of Hygiene and Tropical Medicine, April 27th, 2018.
264. “The Impact of Pollutants on Human Health: No Safe Levels?” From Toxicology to Planetary Health, London School of Hygiene and Tropical Medicine, April 27th, 2018.

265. Topic Symposium: “Toxic Chemicals and the Rise of Chronic Disease in Childhood: A Preventable Epidemic?” (chair and speaker), Pediatric Academic Societies, May 7th, 2018.
266. “Prevention Paradox; Why a Little Lead is Too Much”, Ontario Water Advisory, Toronto, CA, May 7th, 2018.
267. “How the Secrets of Body Care and Cleaning Products Impact your Health”, Panel with Bruce Lanphear, Muhannad Malas and Janie McConnell, Centre for Free Expression, Ryerson University, Toronto, ON, May 7th, 2018.
268. “Prevention Paradox; Why a Little Lead is Too Much” (Keynote), Pittsburgh, PA, Get the Lead Out Conference, May 9th, 2018.
269. “Low-level Lead Exposure and Mortality”, Global Health Forum, Miami, Fl, May 23rd, 2018.
270. “Unleashing the Power of Prevention: Targeting Toxic Chemicals and Pollutants”, Canadian Public Health Association, Montreal, QC, May 28th, 2018.
271. “The Impact of Pollutants on Human Health: No Safe Levels?” Chemicals Management Plan Stakeholder Advisory Council, Health Canada, May 30th, 2018.
272. “Little Things Matter: The Impact of Toxic Chemicals on the Developing Brain”, Pediatric Grand Rounds, University of California – Davis, Sacramento, CA, June 8th, 2018.
273. “Why a Little Lead is Too Much”, Health Canada, Ottawa, ON, August 29th, 2018.
274. “Unleashing the Power of Prevention: Mobilizing Science to Prevent Disease”, ISEE-ISES Workshop, Ottawa, ON, August 30th, 2018.
275. “The Lingering Legacy of Lead: Why a Little Lead is Too Much”, LA Lead Summit: A Strategy for Prevention, University of Southern California, September 14th, Los Angeles, CA.
276. “Little Things Matter: The Impact of Toxic Chemicals on the Developing Brain”, Children’s Hospital of Orange County, Orange County, CA, September 19th, 2018.
277. “The Lingering Legacy of Lead: Why a Little Lead is Too Much”, Hurley Medical Center, Flint, Michigan, October 3rd, 2018.
278. “Lead and The Mysterious Decline in Coronary Heart Disease”, National Institute of Occupational Safety and Health, Cincinnati, OH, October 11, 2018.
279. “Little Things Matter: The Impact of Toxic Chemicals on the Developing Brain”, Grand Rounds, Oregon State Health University, Portland, OR, October 23, 2018.
280. “The Impact of Pollutants on Human Health: No Safe Levels?” Oregon Environmental Council, Portland, OR, October 23, 2018.
281. “Little Things Matter: The Impact of Toxic Chemicals and Organic Food on Children’s Health”, HIPP Scientific Symposium on Organic Food, Kranzberg, Germany, October 30, 2018.

282. “The Mysterious Decline in Coronary Heart Disease”, Harvard University Lead Summit, Cambridge, MA, November 15th, 2018.
283. “The Impact of Pollutants on Human Health: No Safe Levels?” Department of Epidemiology, UMass, Amherst, MA, November 16th, 2018.
284. “Little Things Matter: The Impact of Toxic Chemicals on Human Health”, School of Public Health, Oregon State University, Corvallis, Oregon, April 12th, 2019.
285. Why A Little Lead is Too Much: An Intimate History”, “The Impact of Pollutants on Human Health: No Safe Levels?”, Graduate Course in Environmental Health, School of Public Health, University of California, Berkeley, April 17th, 2019.
286. “The Impact of Pollutants on Human Health: No Safe Levels?”, Department of Environmental Health, School of Public Health, University of California, Berkeley, April 17th, 2019.

Grants

Active Grant Awards

1. Consultant (Joseph Braun, PI). Early Life Perfluoroalkyl Substance Exposure and Obesity: Mechanisms and Phenotyping. 02/01/2016-01/31/2021. National Institutes of Health, $523,725 (5% effort). The purpose of this award is to study the impact of exposure to perfluoroalkyl chemicals on the development of child obesity, adverse cardiometabolic markers and gene regulation. (2.5% effort)
2. Co-Applicant (Linda Booij, Maryse Bouchard PI). In utero exposure to Bisphenol-A and the developing brain in humans: A longitudinal study of epigenetic mechanisms. 03/01-2016 – 03/31/2019. Canadian Institutes of Health Research (CIHR), $344,025. (2.5% effort).
3. Principal Investigator (Multiple PI Award with Christine Till). “Impact of early life fluoride exposure on cognitive and behavioural outcomes in children”. NIEHS, 09/30/16 – 05/01/19, $296,683 (10% effort).
4. Consultant (Aimin Chen, Principal Investigator). Developmental neurotoxicity of organophosphate and novel brominated flame retardants in children. National Institute for Environmental Health Sciences. 1RO1ES028277. 09/30/2017-06/30/22 (10% effort).
5. Mentor (Cynthia Curl, Principal Investigator). Measurement of Agricultural and Dietary Glyphosate Exposure among Pregnant Women. National Institutes of Environmental Health Sciences. 1KO1ES028745-01A1. 09/01/2018-08/31/2022 (5% effort).

Past Grant Awards

1. Principal Investigator, "Dust-Lead and Blood Lead Levels among Urban Children". The National Center for Lead-Safe Housing, $561,619, 06/15/93 to 08/31/94. Department of Housing and Urban Development Contract MDLPT0001-93. (25% effort).
2. Principal Investigator, "Determinants of Lead Exposure among Children in Monroe County, NY", NIEHS Pilot Grant, University of Rochester School of Medicine and Dentistry, Department of Environmental Medicine. $7,600, 06/15/93 to 12/31/95. (0% effort)
3. Principal Investigator, "The Effectiveness of Dust Control in Reducing Children's Blood Lead Levels" U.S. Department of Housing and Urban Development, $128,394, 04/01/94 to 05/30/95. (25% effort).
4. Principal Investigator, "Primary Prevention of Exposure to Lead". Centers for Disease Control and Prevention, $832,228, 09/30/94 to 10/01/98. (25% effort)
5. Principal Investigator, "Lead-Contaminated House Dust and Children's Blood Lead Levels". National Center for Lead-Safe Housing, $43,260, 10/01/96 to 03/30/96. (25% effort).
6. Co-investigator (Christy, PI), “Tuberculosis Screening in Children”. New York Department of Health, $15,000, 01/01/95 to 12/31/96. (0% effort)
7. Co-investigator (Weitzman, PI), “Fellowship Training in General Pediatrics” (Grant # D28PE50008). Bureau of Health Professions, HRSA, U.S. Public Health Service, $1,752,816, 06/01/96 to 05/30/97. (10% effort).
8. Principal Investigator, “Neurobehavioral Effects of Low-Level Childhood Lead Exposure”. University of Rochester School of Medicine & Dentistry, $8,560, 06/01/96 to 05/30/97. (0% effort)
9. Principal Investigator, “Neurobehavioral Effects of low-level Lead Exposure in Children”. NIEHS Pilot Grant, University of Rochester Department of Environmental Medicine, $20,035, 09/01/97 to 08/30/97. (0% effort).
10. Co-investigator (Howard, PI), “Effect on Breastfeeding of Pacifiers and Bottle Feeding”. Bureau of Maternal and Child Health, $420,333, 10/01/96 to 09/30/00. (2.5% effort)
11. Co-investigator (Canfield, PI) “Lead and Children’s Cognitive Functioning”, Research Grants Program, Cornell University. $17,000, 10/01/96 to 09/31/97 (0% effort).

12. Principal Investigator, “Neurobehavioral Effects of Low-Level Lead Exposure in Children” (RO1-ES 08338). National Institute of Environmental Health Sciences, 12/01/96 to 11/31/01, $1,946,848. (25% effort).
13. Co-investigator, (Aligne, PI). “Reduction in Passive Smoking among Children with Asthma: A Randomized Trial of HEPA Air Filtration.” 10/01/96 to 09/31/97, $6,000. KIDD Grant, Rochester General Hospital (0% effort).
14. Co-investigator, (DeWitt, PI). “Faculty Development in General Pediatrics”. Bureau of Health Professions, Health, Department of Health and Human Services 07/01/97 to 06/30/00, $338,000. (15% effort).
15. Principal Investigator, “A Side-by-Side Comparison of Allergen Sampling Methods”, U.S. Department of Housing and Urban Development, 01/02/98 to 12/31/98, $163,065. (15% effort).
16. Principal Investigator, “National Research Service Award - Fellowship Training in General Pediatrics and Adolescent Medicine” (1T32PE10027), Health Resources and Services Administration, DHHS. 07/01/98 to 06/30/03. $634,408. (0% effort).
17. Co-investigator, (Steiner, PI) “Survey of Directors and Graduates of NRSA Fellowship Training Programs”, Health Resources and Services Administration, Department of Health and Human Services. 06/01/98 to 06/30/99.
18. Principal Investigator, “Effect of Soil Remediation on Children’s Blood Lead Levels in Midvale, Utah”. U.S. Environmental Protection Agency, 08/01/98 to 07/30/99. $62,550. (15% effort).
19. Co-investigator, (Phelan, PI) Trends and Patterns in Playground Injuries among U.S. Children.” Ambulatory Pediatric Association, 05/05/99 to 05/04/00. $9,000 (0% effort).
20. Principal Investigator, “Risk Assessment for Residential Lead Hazards”. U.S. Department of Housing and Urban Development, 09/01/99 to 08/30/00. $102,435. (25% effort).
21. Principal Investigator, “Residential Exposures associated with Asthma in U.S. Children and Adolescents” U.S. Department of Housing and Urban Development, 07/16/99 to 03/15/00. $30,400. (20% effort).
22. Principal Investigator, “Effectiveness of Lead Hazard Control Interventions – A Systematic Review" National Center for Lead-Safe Housing, 10/01/99 to 06/01/00. $22,500 (10% effort).
23. Principal Investigator, “Racial Disparity in Blood Lead Levels due to Genetic Variation in Calcium Absorption”. NIEHS Pilot Grant, Center for Environmental Genetics, University of Cincinnati, 04/01/00 to 03/31/01. $28,130 (0% effort).
24. Principal Investigator, “International Pooled Analysis of Prospective, Lead-Exposed Cohorts”. National Institute of Environmental Health Sciences, National Institutes of Health, 08/15/00 to 09/14/01, $16,000. (2.5% effort).

25. Principal Investigator, “A Randomized Trial to Reduce ETS in Children with Asthma” (RO1-HL/ES65731). National Heart, Lung and Blood Institute, National Institutes of Health, 09/29/00 to 09/28/04, $1,546,848. (25% effort).
26. Co-investigator, (Geraghty, PI) “Breastfeeding Practices of Mothers of Multiples”. Ambulatory Pediatric Association, 05/01/01 to 04/30/02. $5,000 (0% effort).
27. Principal Investigator (Subcontract), “A Longitudinal Study of Lead Exposure and Dental Caries”. National Institute of Dental and Craniofacial Research, National Institutes of Health, 08/01/01 to 07/30/04. $300,000 (10% effort).
28. Co-investigator (Phelan, PI), “Fatal and Non-Fatal Residential Injuries in U.S. Children and Adolescents” U.S. Department of Housing and Urban Development, 03/01/01 to 11/31/01. $40,700. (5% effort).
29. Principal Investigator, “Prevalent Neurotoxicants in Children” (PO1-ES11261). National Institute for Environmental Health Sciences and U.S. Environmental Protection Agency, 09/01/01 to 09/31/06, $5,000,000. (30% effort).
30. Principal Investigator, “International Pooled Analysis of Lead-Exposed Cohorts”. Centers for Disease Control (RO1/CCR 521049). Centers for Disease Control, 09/15/01 to 09/14/02, $28,473. (3% effort).
31. Principal Investigator, supplement to “Prevalent Neurotoxicants in Children” (PO1-ES11261). NIEHS, 09/01/02 to 09/31/07, $1,800,000. (10% effort).
32. Co-Investigator, “ADHD Phenotype Network: Animal Model to Clinical Trial”. National Institute of Neurologic Diseases, 09/15/02 to 06/30/05 (15% effort).
33. Principal Investigator, “Linkage of ADHD and Lead Exposure”, Springfield, Ohio Department of Health, 02/01/03 to 06/01/04, $25,000. (0% effort).
34. Co-investigator (Yolton, PI) “Explorations of ETS Exposure on Child Behavior and Sleep” NIEHS, 04/01/04 to 03/30/06, $300,000. (5% effort).
35. Co-investigator (Haynes, PI) “MRI as a Biomarker of Manganese Exposure”. NIEHS, 09/01/04 to 08/30/06, $300,000. (5% effort).
36. Co-investigator (National Center for Healthy Housing, PI) “Development of a Standardized Housing Assessment for Asthma”, U.S. Department of Housing and Urban Development, 11/01/05 to10/31/07, $50,000. (5% effort).
37. Co-Investigator (Hershey, PI) “Epithelial Genes in Allergic Inflammation” National Institutes of Allergy and Infectious Diseases”, 07/01/06 to 06/30/07, $4,787.541. (3% effort).

38. Co-Investigator and Mentor (Wilson, PI), "Racial Difference in DNA Adducts in Tobacco-Exposed Children". Dean's Scholar Award, University of Cincinnati, 02/22/06 to 01/21/09, $150,000 (5% effort).
39. Principal Investigator, "National Research Service Award - Fellowship Training in Primary Care Research," (1T32PE10027), Health Resources and Services Administration, DHHS. 07/01/98 to 06/30/08. $1,600,000. (0% effort).
40. Co-Investigator and Mentor (Kahn, PI). "Childhood Asthma in an Era of Genomics: Will the Generalist's Role be Recast?" Robert Wood Johnson Generalist Physician Faculty Scholars Program" 06/01/04 to 05/30/08, $300,000.
41. Co-Investigator and Mentor (Spanier, PI), "Exhaled Nitric Oxide to Manage Childhood Asthma". National Heart, Lung and Blood Institute, 07/01/06 to 06/31/08, $200,000 (10% effort).
42. Co-investigator (Sub-Contract PI), BYPL Vanguard Center (Specker, Principal Investigator), "National Children's Study", National Institute for Child Health and Development, 11/01/05 to 10/31/10, $500,000. (20% effort). [Relinquished with relocation to SFU].
43. Associate Director and Co-Investigator, (Ho, PI). "Center for Environmental Genetics," NIEHS, 04/01/08 to 3/31/13, $1,000,000 (10% effort). [Relinquished with relocation to SFU.]
44. Co-Investigator (Yolton, PI). "Tobacco Smoke and Early Human Behavior". Clinical Innovator Award, Flight Attendant Medical Research Institute", 07/01/07 to 06/30/10, $300,000. (3% effort).
45. Co-Investigator (Spanier, PI). "Low Level Prenatal Tobacco Exposure and Infant Wheeze." Young Clinical Scientist Award, Flight Attendant Medical Research Institute, 07/01/07 to 06/30/12, $300,000. (5% effort).
46. Co-Investigator and Mentor (Spanier, PI). K23, "Prenatal Low Level Tobacco & Phthalate Exposure and Childhood Respiratory Health". National Institute for Environmental Health Sciences, 12/1/07 to 11/30/12, $623,679 (0% funded effort).
47. Co-investigator (Yolton, PI). "Neurobehavioral effects of insecticide exposure in pregnancy and early childhood." NIEHS, 09/01/09 to 08/31/12.
48. Principal Investigator (Bruce Lanphear, PI), "A Community-Based Trial to Prevent Lead Poisoning and Injuries," National Institute for Environmental Health Sciences, 04/01/07 to 03/30/13, $2,000,000. (25% effort).
49. Co-Investigator (Kim N. Dietrich, PI). "Early Lead Exposure, ADHD & Persistent Criminality: Role of Genes & Environment," National Institute for Environmental Health Sciences, 04/01/07 to 3/31/2013, $1,250,000. (2.5% funded effort).

50. Co-Investigator and Sub-Contract PI (Brenda Eskenazi, PI). This supplemental award was to conduct a pooled analysis of prenatal organophophate pesticide exposures with birth outcomes and neurodevelopment in children using 4 US birth cohorts. NIEHS, 09/01/2009 to 08/31/2013, $96,000 (0% effort).
51. Mentor and Supervisor (Glenys Webster, PI). Michael Smith Foundation for Health Research Postdoctoral Training Award, 03/01/12 to 02/28/15, $134,500 (5% effort).
52. Co-Principal Investigator (Tye Arbuckle, PI). Maternal-Infant Research on Environmental Chemicals: Effects on Child Development (MIREC-CD). 06/26/11 to /5/25/14, Health Canada Chemical Management Program, $283,000 (10% effort).
53. Co-Investigator (Patti Dods and Amanda Wheeler, co-PIs). Phthalate Exposure and the development of asthma in the CHILD Study. 06/01/11 to 05/30/14, Health Canada Chemical Management Program, $204,000 (5% effort). Consultant (Stephanie Engel, PI). A pooled investigation of prenatal phthalate exposure and childhood obesity. 11/01/2012 – 10/31/15, NIEHS. $275,000. (5% effort).
54. Co-Investigator (Ryan Allen, PI). A randomized air filter intervention study of air pollution and fetal growth in a highly polluted community. 06/08/2012 – 05/30/15, CIHR $348,000 (10% effort).
55. Co-Investigator (William Fraser and Tye Arbuckle, co-PIs). MIREC-CD Biomonitoring Study in Vancouver. 09/01/2013 – 08/30/2014. Health Canada, $120,138 (10% effort).
56. Principal Investigator. Knowledge translation tools for capacity building for an online Canadian Environmental Health Atlas. 03/01/12 – 02/28/13, Canadian Institutes of Health Research, $98,974 (10% effort).
57. Principal Investigator (with Lawrence McCandless). Prenatal exposure to environmental contaminants and fetal growth: How to account for multiplicity when testing multiple statistical hypotheses?. 07/01/2015-06/30/2016. Canadian Institutes of Health Research (CIHR), $12,000 (5% effort).
58. Principal Investigator, Canadian Environmental Health Atlas Knowledge Translation to produce videos and interactive tools. 06/01/2015-07/30/2016. Canadian Internet Registration Authority, $50,000 (10% effort).
59. Co-Investigator (Kieran Phelan, PI). “Injury Prevention in a Home Visitation Population”. NICHD, 09/28/10 to 07/31/16, $2,000,000 (total direct costs over 5 years) (10% effort).
60. Co-applicant (Timothy F. Oberlander, PI). Developmental origins of autism: A population level linked data study of prenatal antidepressant medication exposure. 09/01/2013 – 09/31/2016, Canadian Institutes of Health Research (CIHR), $285,768.

61. Principal Investigator (Multiple PI Award with Aimin Chen and Kimberly Yolton). "Longitudinal study of exposures to PBDEs and PFCs and child behavior". NIEHS, 04/30/11 – 05/01/17, $2,150,000 (total direct costs over 5 years) (20% effort).
62. Principal Applicants (McCandless and Lanphear). Biostatistical methods for estimating the cumulative impact of environmental contaminant exposures on preterm birth. Canadian Institute for Human Development, Child and Youth Health. 12/06/16-12/05/18, $200,000 (10% effort).
63. Co-investigator (Ryan Allen, PI). Randomized Interventions to Evaluate the Effects of Air Pollution Exposure on Children's Health and Development. 03/01/2015 – 03/31/2019, Canadian Institutes of Health Research (CIHR), $720,535. (10% effort)
64. Co-investigator (Joseph Braun, PI). Endocrine Disrupting Chemicals, Thyroid Hormones and Child Neurobehavior. 06/01/2015-03/31/2019. National Institutes of Health, $471,241 (5% effort). The purpose of this study is test if and when early life exposures to phthalates, triclosan, or bisphenol A adversely impacts children's cognition and behavior.
65.

Ethics Training for Research

CITI (Collaborative Institutional Training Initiative) (Reference# 7159023). Academic and Regional Health Centers Curriculum Course, completed on December 16th, 2011.

CITI (Collaborative Institutional Training Initiative) (Reference# 7160515), Canada GCP Curriculum Course, completed on December 16th, 2011.

CITI (Collaborative Institutional Training Initiative) (Reference# 8316270), Human Subjects Core Curriculum, completed on August 17th, 2012.

CITI (Collaborative Institutional Training Initiative) (Reference# 13561457), Academic and Regional Health Centers Core Curriculum, completed on September 1st, 2014.

CITI (Collaborative Institutional Training Initiative) (Reference# 16954900), Human Subjects Research Core Curriculum, completed on October 31st, 2015.

Expert Declaration

of

Kathleen M. Thiessen, Ph.D.

May 20, 2020

Food & Water Watch et al. v. Environmental Protection Agency
No. 17-cv—02162

Oak Ridge Center for Risk Analysis, Inc.
102 Donner Drive
Oak Ridge, TN 37830

TABLE OF CONTENTS

I, Kathleen Thiessen Ph.D., declare that:

1. I am a risk assessment scientist at Oak Ridge Center for Risk Analysis in Oak Ridge, Tennessee. For more than 30 years, I have been involved in the evaluation of exposures, doses, and risks to human health from trace levels of contaminants in the environment, including fluoride, and in the use of uncertainty analysis for environmental and health risk assessment.

2. I was asked to apply risk assessment frameworks used by the Environmental Protection Agency (EPA) to the current scientific literature on fluoride neurotoxicity to determine whether neurotoxicity is a hazard of fluoride exposure, and whether this hazard is a risk at the levels of fluoride added to drinking water for fluoridation (0.7 mg/L).

I. SUMMARY OF QUALIFICATIONS

3. A complete summary of my qualifications and publications can be found in my Curriculum Vitae, which has been marked as Plaintiffs' Exhibit 7 and attached herein.

4. In the course of my work as a risk assessment scientist, I have done work for the U.S. Environmental Protection Agency (EPA), the U.S. Department of Energy, the Centers for Disease Control and Prevention, the U.S. Nuclear Regulatory Commission, the National Cancer Institute, and the National Institute for Occupational Safety and Health, as well as a number of other government and private clients.

5. I have authored several reports for the EPA on the health effects of specific environmental contaminants, including Health Issue Assessments of fluorides (hydrogen fluoride and related compounds) and mercuric chloride.

6. More recently, I served on two subcommittees of the National Research Council, one which was asked by EPA to review the toxicologic literature on fluoride (which resulted in the 2006 publication *Fluoride in Drinking Water: A Scientific Review of EPA's Standards*), and one

dealing with guidance levels for air contaminants in submarines. For the latter review, published in 2009, the NRC asked me to write much of the chapter on hydrogen fluoride.

7. Recently, I led the Working Group on Assessment of Exposures and Countermeasures in Urban Environments for the International Atomic Energy Agency's (IAEA) program on Development, Testing and Harmonization of Models and Data for Radiological Impact Assessment. I was also involved in the preparation of an IAEA guidance document on implementation of remediation strategies following accidental releases of radioactivity.

8. Throughout my career, I have authored or contributed to a number of open literature publications in peer-reviewed journals such as *Environmental Science and Technology*, *Environmental Pollution*, *Atmospheric Environment*, *Journal of Environmental Radioactivity*, and the *International Journal of Occupational and Environmental Health*. I have also served as a peer reviewer for journals such as *American Journal of Preventive Medicine*, *Environment International*, *Environmental Pollution*, *Risk Analysis*, *Science of the Total Environment*, *Environmental Health Perspectives*, and *Journal of Environmental Radioactivity*, among others.

II. SUMMARY OF OPINIONS

9. Under EPA's *Guidelines for Neurotoxicity Risk Assessment*, there is sufficient evidence to conclude that neurotoxicity is a hazard of fluoride exposure.

10. The animal data on fluoride neurotoxicity are consistent with the epidemiological data in showing a risk of cognitive deficits at doses of fluoride ingested from fluoridated water.

11. Fluoridation chemicals present an "unreasonable risk" of neurotoxic effects, including IQ loss, if assessed under the same risk characterization and risk determination framework that EPA uses in its evaluations of other chemicals under TSCA.

III. SUMMARY OF METHODOLOGY

A. Risk Assessment

12. EPA has stated it "will follow" its *Guidelines on Neurotoxicity Risk Assessment* (hereafter, *Guidelines*) when "evaluating data on potential neurotoxicity associated with exposure to environmental toxicants."[1] I conducted a risk assessment in accordance with these *Guidelines*, including a Hazard Characterization, Quantitative Dose Response Analysis, Exposure Assessment, and Risk Characterization.

13. Hazard Characterization: Pursuant to the *Guidelines*, I conducted a Hazard Characterization, in which I considered: (1) the animal studies on neuroanatomical, neurochemical, and behavioral effects, including effects on learning and memory; (2) human case reports, including clinician observations of occupationally exposed workers; (3) human epidemiology studies of fluoride and cognitive deficits, including all prospective cohort studies; (4) the literature on fluoride's neuroendocrine effects; (5) animal and human research on possible modes of action (direct and indirect) by which fluoride affects the brain; (6) dose-response data on fluoride and neurotoxic outcomes in animal and epidemiological studies; (7) the toxicokinetics of fluoride, including data on placental transfer and uptake into the brain; and (8) *in vitro* studies investigating fluoride's effects on brain cells, including several that used low concentrations.

14. Quantitative Dose Response Analysis: Since the literature demonstrates with high confidence that neurotoxicity is a hazard of fluoride exposure, I turned to the second step of an EPA neurotoxicity risk assessment: Quantitative Dose Response. In a quantitative dose-response analysis, a "Point of Departure" (POD) is identified from the available animal and human data in order to derive a dose that will be without appreciable risk (i.e., a Reference Dose, or RfD). For

[1] EPA (1998a), p. 1.

my analysis, I focused on the animal data, as I understood that Dr. Grandjean had already calculated a POD (i.e., BMDL) from the human birth cohort data.

15. To increase confidence in the risk characterization (a later step in the analysis, discussed below), I did not identify just one POD from the animal data. Instead, I identified the full range of PODs that can be justified, including the *least protective*. After converting these PODs into Human Equivalent Doses (HED), I applied different combinations of uncertainty factors (from non-conservative to conservative) to derive the *full range of reference doses that can be justified from the animal literature*.

16. Exposure Assessment: Consistent with the *Guidelines*, I conducted an Exposure Assessment that focused solely on the condition of use at issue in this case: fluoridation of drinking water. For my initial assessment, I relied primarily, but not solely, on the National Research Council's estimates of fluoride intake from water from the 2006 report. In response to criticisms that NRC's data may no longer be representative of contemporary exposures, I considered EPA's 2019 assessment of water intake data, in which the Agency identified the most scientifically sound and up-to-date data to use for risk assessment. I compared these updated values from EPA with the values I initially used to see if they have any material effect on my risk estimates (they did not).

17. Risk Characterization: Consistent with the *Guidelines*, I integrated the information on hazards and exposures in a risk characterization by, among other things, conducting a "Margin of Exposure" analysis for each of the PODs identified through the Quantitative Dose Response analysis.

18. Risk Determination: For the risk determination, I considered the risk-related factors that EPA has identified as relevant for risk determinations under TSCA. At the time of my initial report, EPA had not yet issued any draft risk evaluations under Section 6 of TSCA, so I relied for

guidance on risk evaluations that EPA had completed under Section 5. In response to criticism on this point, I reviewed the draft risk evaluations that EPA has subsequently issued under Section 6 to assess whether the factors EPA considers under Section 6 affect my initial determination (they do not).

B. Materials Relied Upon

19. For my risk assessment, I have relied upon my background, training and expertise in risk assessment, as well as my existing familiarity with the scientific literature on fluoride, which I first developed through extensive literature reviews for both the EPA and NRC. I also considered the following materials:

20. EPA documents, including (i) the *Guidelines* and other guidance documents that EPA has issued on risk assessment; (ii) risk assessments that EPA has conducted pursuant to the *Guidelines*;[2] (iii) risk evaluations that EPA has conducted under TSCA; and (iv) EPA's water intake data.

21. The NRC's review of the toxicologic literature on fluoride (NRC 2006), which I co-authored.

22. Animal studies on fluoride neurotoxicity that have been published since the NRC's 2006 review, which I obtained through a search of the National Library of Medicine's online database PubMed, as described further below.

23. The NTP's systematic review of studies addressing fluoride's impact on learning and memory in animals (NTP 2015, NTP 2016).

24. All prospective cohort studies on fluoride and neurodevelopment in humans

[2] I obtained the complete list of risk assessments that EPA has conducted pursuant to the *Guidelines* via an interrogatory response produced by EPA, which was provided to me by counsel.

(Bashash et al. 2017; Bashash 2018; Broadbent et al. 2015; Green et al. 2019; Shannon et al. 1986; Till et al. 2020; and Valdez-Jiminez et al. 2017).

25. Meta-analyses of the cross-sectional studies on fluoride and IQ (Choi 2012, Duan 2018).

26. The deposition of Dr. Kristina Thayer, the Director of EPA's Integrated Risk Information System (IRIS) and principal author of the NTP's 2015 and 2016 systematic reviews.

27. The deposition of Casey Hannan, the Acting Director of the Oral Health Division of the Centers for Disease Control and Prevention.

28. The deposition of Dr. Tala Henry, the Deputy Director of EPA's Office of Pollution Prevention and Toxics.

29. Studies provided by counsel—much of which I was already familiar with—which I understand were also provided to EPA's experts as well, including Dr. Tsuji.

C. Literature Search for Animal Neurotoxicity Data

30. For the animal literature, I conducted a search of the National Library of Medicine's online database PubMed to identify studies published since the NRC's 2006 review. The search terms used were: "fluoride and brain," "fluoride and learning," and "fluoride and memory."

31. The titles of all studies published since 2006 were reviewed to identify potentially relevant primary studies, and, among potentially relevant studies, abstracts were reviewed to verify relevance. Reviews, studies in Chinese for which translations were not available, and *in vitro* studies were excluded. Full-text copies of all relevant studies were obtained. In total, the search identified 110 papers. Papers that appeared to be reporting effects from the same underlying rodent experiment were treated as one study, leaving 105 distinct studies.

32. The 105 studies I identified are not an exhaustive list of the studies published since

2006, as they do not include studies that were not indexed in PubMed (e.g., studies published in the journal *Fluoride* or in certain Chinese-language journals such as the *Chinese Journal of Endemiology*). In addition, the search terms probably did not identify all relevant studies available on PubMed.

Nevertheless, the studies obtained through this pre-defined search protocol should be a reasonably representative sample of the recent literature.

D. Systematic Review

33. I did not conduct a formal systematic review, but a risk assessment under the *Guidelines* has been considered the effective equivalent of a systematic review.

IV. HISTORIC CONTEXT: 1930s to 2006

34. The early epidemiological studies in the U.S. that claimed to establish the safety of waterborne fluoride (fluoride concentrations ranging from 1 to 8 mg/L in drinking water) did not address the potential for fluoride to cause neurological effects, including IQ loss.[3] The primary focus of these early studies was, instead, on skeletal health.

35. Although largely overlooked, some of the early studies of occupationally exposed workers,[4] as well as some of the early studies of fluoride-exposed animals,[5] reported central nervous system effects from fluoride exposure. In a 1953 study of monkeys, Wadhwani and Ramasway reported that monkeys with chronic fluorosis "did not conduct themselves with intelligence and agility of mind normally associated with them. There was a significant lack of co-ordination in their behaviour."[6] These early observations, some of which remained unpublished,

[3] Call et al. (1965); Leone et al. (1954; 1955a; 1955b); McCauley and McClure (1954); McClure (1944); Schlesinger et al. (1956a; 1956b); Stevenson and Watson (1957).

[4] Roholm (1937); Popov et al. (1974); Duan et al. (1995); Guo et al. (2001); Yazdi et al. (2011).

[5] Wadhwani and Ramasway (1953); Lu et al. (1961); Rice and Lu (1963); Sadilova et al. (1968).

[6] Wadhwani and Ramasway (1953).

were largely ignored at the time.

36. The first known study of fluoride and intelligence in humans was published in 1989 by Ren and colleagues in China.[7] A flurry of similar studies were published in China in the 1990s.[8] Most of these studies were published in Chinese, and they remained largely unknown outside of China until English translations started to become available after the NRC's report in 2006.

37. In 2006, the NRC concluded that "fluorides have the ability to interfere with the functions of the brain and the body by direct and indirect means."[9] The NRC reached this conclusion based on the histological, biochemical, and molecular findings from animal studies published in the 1990s and early 2000s.[10] The NRC also reviewed two studies that examined the impact of fluoride on learning and memory in animals, but the data were not yet sufficient to draw conclusions on cognitive effects.[11]

38. As part of its report, the NRC also reviewed the 4 studies on fluoride and intelligence that were then available in English.[12] Various methodological limitations were identified with these studies, but the NRC concluded that the consistency of the results (i.e., reduced intelligence among children exposed to elevated fluoride) warranted further epidemiological research into the potential of fluoride to lower IQ.

39. The NRC also reviewed the toxicologic literature on fluoride's effects on the endocrine system, including the thyroid gland. The NRC concluded that fluoride is an endocrine-disrupting chemical which can alter thyroid function at estimated average intakes as low as 0.01 to

[7] Ren et al. (1989).
[8] Qin et al. (1990); Chen et al. (1991); Guo et al. (1991); Lin et al. (1991); Sun et al. (1991); An et al. (1992); Li et al. (1994); Xu et al. (1994); Yang et al. (1994); Duan et al. (1995); Li et al. (1995); Wang et al. (1996); Yao et al. (1996; 1997); Zhao et al. (1996).
[9] NRC (2006), p. 222.
[10] NRC (2006), pp. 221-222.
[11] NRC (2006), pp. 215-216, 221.
[12] Li et al. (1995); Zhao et al. (1996); Lu et al. (2000); Xiang et al. (2003a; 2003b).

0.03 mg/kg/day in individuals with iodine deficiency.[13] The NRC recognized the potential relevance of fluoride's endocrine effects to neurotoxicity, noting that depressed thyroid function during pregnancy can lower the IQ of the offspring.[14]

40. The NRC's findings on the neurotoxic potential of fluoride have been accepted as an accurate summary of the hazard by the EPA and other federal agencies, including the CDC.

41. My risk assessment builds upon NRC's hazard determinations by considering the large volume of additional research that has been published since the NRC findings were released.

V. HAZARD CHARACTERIZATION

A. The "Sufficient Evidence" Standard

42. The focus of the Hazard Characterization is whether, at some level of exposure, the chemical has a credible potential to cause neurotoxic effects (i.e., whether neurotoxicity is a *hazard* of the chemical). The question of whether this hazard is a *risk* at environmentally relevant exposures is a separate question that is addressed in the Risk Characterization phase (as discussed below).

43. Under the *Guidelines*, hazard assessment is a qualitative determination in which the risk assessor must determine whether "sufficient evidence" of a neurotoxicity hazard exists.[15] A "sufficient evidence" finding "can be based on either human or animal data."[16] EPA has a preference for using human data if suitable data exist;[17] in practice, however, animal data are almost always used.[18]

13 NRC (2006), pp. 262-263, 266.
14 NRC (2006), p. 263.
15 EPA (1998a), pp. 11, 53, 55-56.
16 EPA (1998a), p. 11.
17 EPA (2018a), p. 2-1.
18 EPA (1998a), p. 20.

1. Sufficient Evidence from Human Data

44. For human data, "sufficient evidence" of a neurotoxic hazard exists if epidemiologic studies show that "some neurotoxic effect is *associated* with exposure."[19] EPA contrasted this requirement of an "association" with what the Agency recognized to be the "more stringent requirement" of "causality."[20] Under the *Guidelines*, there is no requirement to prove causality; evidence of an association is enough.

45. In assessing whether epidemiological studies demonstrate an association with neurotoxicity, EPA has stated that prospective cohort studies "should weigh heavily" in the assessment.[21] The *Guidelines* recognize that prospective studies are "invaluable for determining the time course for development of dysfunction" and permit "direct estimate of risks attributed to a particular exposure."[22] The only drawback of prospective studies that the *Guidelines* identify are that they "can be very time-consuming and costly."[23]

2. Sufficient Evidence from Animal Data

46. For animal data, "sufficient evidence" of a neurotoxic hazard exists if experimental studies demonstrate a potential neurotoxic hazard in humans.[24] The "minimum evidence" necessary to demonstrate a potential hazard is "a single appropriate, well-executed study in a single experimental animal species." If no individual study is sufficient to establish a hazard, "the total

[19] EPA (1998a), p. 53.
[20] EPA (1998a), pp. 53.
[21] EPA (1998a), pp. 18.
[22] EPA (1998a), pp. 17.
[23] EPA (1998a), pp. 17.
[24] EPA (1998a), p. 53.

available data may support such a conclusion" including data on toxicokinetics[25] and mechanisms of action.[26]

47. Neurotoxic endpoints in animal studies fall into several categories, including neuroanatomical, neurochemical, and behavioral.[27]

48. Neuroanatomical endpoints include changes to the brain, including damage to brain cells, that are detectable under a microscope (i.e., "histological").[28] The *Guidelines* consider neuroanatomical changes to be "of concern," and EPA has established reference doses for chemicals based on neuroanatomical effects.

49. Neurochemical effects include biochemical changes, such as alterations in neurotransmitter function and effects on enzymes. The Guidelines state that neurochemical changes "may be regarded as

adverse because of their known or presumed relation to neurophysiological and/or neurobehavioral consequences."[29]

50. Behavioral changes include alterations to motor activity, changes in sensory abilities or motor coordination, and impairments in learning, memory, and attention.[30] EPA has repeatedly based reference doses on behavioral alterations documented in animals, including learning and memory impairments.

51. In considering the relevance of the animal data to humans, the *Guidelines* provide four default assumptions. First, EPA assumes that "an agent that produces detectable adverse

[25] The *Guidelines* use the term pharmacokinetics. Both pharmacokinetics and toxicokinetics refer to the uptake, distribution, and retention of chemicals, with the former term being more frequently used in the context of pharmaceuticals, and the latter term more frequently used in the context of toxicants.

[26] EPA (1998a), p. 56.

[27] EPA (1998a), pp. 20-21.

[28] EPA (1998a), p. 21.

[29] EPA (1998a), p. 55.

[30] EPA (1998a), p. 21.

neurotoxic effects in experimental animal studies will pose a potential hazard to humans."[31] Second, EPA assumes that neuroanatomical, neurochemical, and behavioral changes "are of concern."[32] Third, EPA assumes that "the neurotoxic effects seen in animal studies may not always be the same as those produced in humans" due to "species-specific differences in maturation of the nervous system, differences in timing of exposure, metabolism, or mechanisms of action."[33] Fourth, EPA assumes that "humans are as sensitive as the most sensitive animal species tested."[34] These four assumptions are "plausibly conservative," meaning that "they are protective of public health and are also well founded in scientific knowledge about the effects of concern."[35]

3. Data that EPA Has Found Sufficient for Hazard Determination

52. EPA has conducted 10 risk assessments pursuant to the *Guidelines*. In 9 of these risk assessments, EPA found sufficient evidence to make a hazard determination and established Reference Doses (RfDs) or Reference Concentrations (RfCs)[36] to protect against the hazard.[37] In each of these 9 assessments, EPA based its hazard determination on animal data. For 6 of these 9 assessments, the chemicals had *no* human data on neurotoxicity (Table 1). For the 3 chemicals with some human data, no prospective cohort studies were available.

53. The principal studies[38] which EPA has used to establish RfDs have not been

[31] EPA (1998a), p. 6.
[32] EPA (1998a), p. 6.
[33] EPA (1998a), p. 7.
[34] EPA (1998a), p. 7.
[35] EPA (1998a), p. 7.
[36] Reference Doses refer to oral exposures, while Reference Concentrations refer to inhalational exposure. Eight of the 9 neurotoxicity risk assessments established RfDs, while 1 set an RfC. For purposes of simplicity, I will refer to Reference Doses for the remainder of this declaration when discussing these assessments.
[37] These 9 risk assessments were performed for BDE-47 (EPA 2008a), BDE-99 (EPA 2008b), BDE-153 (EPA 2008c), BDE-209 (EPA 2008d), Chlorine Dioxide and Chlorite (EPA 2000b), 2-Hexanone (EPA 2009b), Methanol (EPA 2013a), RDX (EPA 2018a), and Trimethylbenzenes (EPA 2016).
[38] A principal study is the study that contributes most significantly to the assessment of risk and is generally the basis for the Point of Departure from which a reference value is derived.

"perfect" studies. In fact, in most of the neurotoxicity risk assessments, EPA has identified a number of methodological limitations with the studies. Some of the principal studies did not conform to EPA's testing guidelines for animal studies; some used relatively small numbers of animals (e.g., 10 per group); and the principal studies that investigated effects from prenatal exposures did not always control for "litter effects," a methodological deficiency that can skew the effect size in developmental studies. In light of these limitations, EPA had "low confidence" for the studies it relied upon for several of its risk assessments (see Table 1). This did not stop EPA from establishing RfDs for these chemicals.

54. In several of EPA's neurotoxicity risk assessments, EPA established an RfD despite a relatively small number of animal studies. In the RDX risk assessment, for example, EPA identified 16 animal studies, only two of which had been published. EPA characterized these studies as showing "consistent evidence" of neurotoxicity because 11 of the 16 studies reported neurological effects[39] and the effects were generally dose-related (although inconsistencies existed across the studies in terms of the doses that produced effects).[40] EPA has thus recognized that "consistency" of the evidence is not synonymous with unanimity.

[39] EPA (2018a), p. 1-23.
[40] EPA (2018a), pp. 1-12, 1-18.

Table 1. Chemicals with oral RfDs based on neurological endpoints, assessed according to EPA's Guidelines for Neurotoxicity Risk Assessment.[a]

Name of Chemical	Human Neurotoxicity Data?[b]	Principal Study	Confidence in Principal Study	Known Mode of Action?	Effect	Reference
BDE-47	No	Animal	Not given[c]	Inadequate data	Changes in spontaneous motor activity and habituation	EPA (2008a)
BDE-99	No	Animal	Not given[d]	Inadequate data	Neurobehavioral developmental effects; changes in motor activity	EPA (2008b)
BDE-153	No	Animal	Not given[e]	Inadequate data	Spontaneous behavior, learning and memory	EPA (2008c)
BDE-209	No	Animal	Low	Inadequate data	Changes in spontaneous behavior and habituation	EPA (2008d)
Chlorine Dioxide and Chlorite	No	Animal	Medium	No	Neurodevelopmental delay; lowered auditory startle amplitude	EPA (2000b)
2-Hexanone	No	Animal	Medium	Yes	Axonal swelling in peripheral nerves	EPA (2009b)
RDX	One cross-sectional study, 16 case reports	Animal	High	Yes	Convulsions	EPA (2018a)
Trimethylbenzenes	Occupational studies of solvent mixtures, controlled experiments with healthy adults	Animal	Low to Medium	Tentative, based on structurally similar compounds	Decreased pain sensitivity	EPA (2016)

[a] EPA (1998a); Federal Register (1998). In addition, an inhalation RfC was derived for methanol based on animal data (EPA 2013a); the confidence in the RfC was considered medium to high.

[b] Human studies of neurotoxicity endpoints.

[c] Confidence in the principal study was not stated, but the "overall confidence in the RfD assessment of BDE-47 is low" (EPA 2008a, p. 48).

[d] Confidence in the principal study was not stated, but the "overall confidence in the RfD [for BDE-99] is low" (EPA 2008b, p. 67).

[e] Confidence in the principal study was not stated, but the "overall confidence in the RfD assessment for BDE-153 is low" (EPA 2008c, p. 37).

55. EPA has taken a similar approach to animal data in some of its draft risk evaluations under Section 6 of TSCA. In its NMP risk evaluation, for example, EPA based its risk calculations for chronic exposures in humans on animal data linking NMP to reduced fertility, despite the fact that there were only six animal studies available, three of which found no effect.[41] These contradictory findings were considered a source of uncertainty, but did not stop EPA from using these animal data to assess risk in humans. In fact, EPA made findings of unreasonable risk in humans exposed to lower doses of NMP based on this small body of contradictory data.

B. Human Studies on the Neurotoxicity of Fluoride

56. As noted earlier, the *Guidelines* state that prospective cohort studies "should weigh heavily in the risk assessment process."[42] The *Guidelines* also identify other types of human studies that can inform the assessment, including case reports and cross-sectional studies.[43]

57. In contrast to 9 chemicals for which EPA has established reference doses under the *Guidelines*, there are abundant human data on fluoride neurotoxicity, including 4 high-quality prospective cohort studies with individualized measurements of exposure during the prenatal period.[44]

58. I understand that Dr. Hu and Dr. Lanphear will be addressing the ELEMENT and MIREC birth cohort studies, and I understand that Dr. Philippe Grandjean will be addressing the other epidemiological studies, so I will forego repeating the details here.

[41] EPA (2019d), pp. 173-174.
[42] EPA (1998a), p. 17.
[43] EPA (1998a), pp. 15-16.
[44] Bashash et al. (2017; 2018); Green et al. (2019); Valdez-Jiménez et al. (2017).

59. As I described in my expert report, the human data on fluoride *strongly* support a hazard determination. Most importantly, each of the 4 prospective studies with measurements of *prenatal* exposure has found large and significant adverse associations with neurodevelopment, including IQ loss and inattention. An additional prospective study has found an association between IQ deficits and fluoride exposure during *infancy*.[45] These studies—which have consistently detected a significant association between early-life fluoride exposure and cognitive deficits using the most reliable study design identified by the *Guidelines*—are by themselves enough to constitute "sufficient evidence" of a hazard.

60. The consistency of the inverse association between fluoride and IQ in cross-sectional studies also adds important weight to the hazard assessment. Although cross-sectional studies are limited in their capacity to establish causal relationships, this limitation is lessened where the study examines stable populations and stable water fluoride levels.[46] In any event, the focus of the *Guidelines* is on assessing whether there is a reliable *association* with neurotoxicity, not on definitively proving causality.[47] As several meta-analyses have demonstrated, the cross-sectional studies show large and significant inverse *associations* between fluoride and IQ, with an average loss of about 7 IQ points.[48]

61. Finally, the case reports of neurological symptoms following fluoride exposure (e.g., general malaise, fatigue, headaches, and difficulties with concentration and memory) add

[45] Till et al. (2020).

[46] Under these conditions, measurement of current water fluoride levels may be a reasonable, albeit imperfect, proxy for exposure from the prenatal period onward, and thus the temporality requirement for a causal inference is partially met. A number of the cross-sectional studies on fluoride and IQ have, in fact, expressly limited the study population to children who lived in the same area since birth, which increases the basis for inferring causation. Chen et al. (1991); Choi et al. (2015); Ding et al. (2011), Karimzade et al. (2014a; 2014b); Khan et al. (2015); Lu et al. (2000); Nagarajappa et al. (2013); Rocha Amador et al. (2007); Seraj et al. (2012); Sudhir et al. (2009); Wang et al. (2007); Yao et al. (1996; 1997); Zhang et al. (2015b).

[47] EPA (1998a), p. 53.

[48] Choi et al. 2012; Duan et al. 2018.

additional support to the hazard determination. While case reports are generally not sufficient, by themselves, to establish a hazard, the *Guidelines* consider them "useful when corroborating epidemiological data are available."[49] Further, as the NRC noted, several of the case reports on fluoride can be characterized as "experimental studies," since they involved "individuals who underwent withdrawal from their source of fluoride exposure and subsequent re-exposures under 'blind' conditions. In most cases, the symptoms disappeared with the elimination of exposure to fluoride and returned when exposure was reinstated."[50] There is credible evidence, therefore, that for some sensitive individuals, fluoride exposure may cause overt neurological symptoms, although the NRC called for more research to better understand the issue.

C. Animal Studies on Fluoride Neurotoxicity

62. The animal research on fluoride neurotoxicity was sufficient to permit the NRC to conclude, in 2006, that fluoride interferes with the functions of the brain.[51] The NRC based this finding on the neuroanatomical and neurochemical changes produced by fluoride in laboratory animals. These changes include: reduced protein and phospholipid content; inhibition of acetylcholinesterase; interference with neurotransmitters; increased production of free radicals in the brain (i.e., oxidative stress); neuronal deformations; increased uptake of aluminum; and enhancement of reactive microglia.[52]

63. Many animal studies have been published since the NRC review which add further support to the hazard determination, as I will now discuss.

1. <u>Studies Indexed by the National Library of Medicine (PubMed)</u>

64. In my search of PubMed, I identified 105 studies that have been published since

[49] EPA (1998a), p. 15.
[50] NRC (2006), pp. 208-209.
[51] NRC (2006), p. 222.
[52] NRC (2006), pp. 221-222.

2006. Of these studies, all but 4 reported associations between fluoride exposure and neurotoxic outcomes.[53]

65. Table A-1 in Appendix A to this declaration provides data from the 88 animal studies which investigated neuroanatomical and neurochemical endpoints (i.e., "structural" effects), while Table A-2 provides data from the 36 animal studies which investigated learning and memory endpoints (i.e., "functional" effects).[54] Twenty-nine studies investigated both types of effects and are in both lists.[55]

66. As can be seen in Table A-1, rodent studies published since the NRC review have continued to document structural (e.g., neuroanatomical and neurochemical) changes in the brains of fluoride-treated rodents. These changes include oxidative stress, neuronal degeneration, mitochondrial disturbances, reductions in nicotinic receptors, impaired synaptic plasticity, and neuroinflammation.

67. Among the studies that have investigated both structural and functional effects of fluoride, the former have sometimes (but not always) occurred at lower exposures, suggesting that fluoride can cause cellular and biochemical changes in the brain prior to the manifestation of outwardly demonstrable deficits.[56] Put another way, deficits in learning and memory likely represent a relatively advanced stage of fluoride neurotoxicity. Nevertheless, both structural and

[53] Negative results were reported by Whitford et al. (2009), Pulangan et al. (2018), McPherson et al. (2018), and Jia et al. (2019, which I discuss later.

[54] For purposes of simplicity, I have used the term "structural" to refer to both neuroanatomical and neurochemical effects. While neurochemical effects are technically "functional" in nature, I use the word "functional" to refer solely to outward manifestations of neurotoxicity (i.e., learning/memory deficits). In this declaration, therefore, "structural" changes refer to all changes observed *in the brain*, while functional effects refer to all changes in *outward behavior* (e.g., learning and memory test performance, etc).

[55] To facilitate comparisons across these studies, Tables A-1 and A-2 exclude 2 studies of non-rodents as well as four studies in which the fluoride exposure was part of a mixture involving other potentially neurotoxic chemicals, one study involving exposure by a route other than ordinary ingestion, and two behavioral studies with endpoints that did not specifically involve learning and memory.

[56] See, for example, Agustina et al. (2018); Ma et al. (2015); Niu et al. (2018a); Sun et al. (2018); Wang et al. (2018a); Zhang et al. (2019); Zhao et al. (2019).

functional harms have repeatedly been observed in rodents at water fluoride concentrations between 5 mg/L and 23 mg/L.[57] As with the RDX literature,[58] there are some inconsistencies across the studies in the reported doses that can cause certain types of harm; these differences likely result, at least in part, from differences in study design, including differences in timing of exposure, duration of exposure, and strain and sex of animal.

68. Most of the animal studies to date have used subchronic exposure scenarios, which would tend to understate the effect from lifetime exposure. EPA's testing guidelines define a chronic exposure study in rodents as one that lasts at least 12 months.[59] None of the recent learning studies has lasted 12 months, and only 1 of the recent structural studies has lasted 12 months or more.[60] Among the studies that have tested animals at multiple points in time, the effects have tended to worsen with time, with some effects not appearing at all until 3 to 6 months of chronic exposure.[61] Since most of the studies on fluoride neurotoxicity have lasted no longer than 3 months, the studies are likely not detecting the full spectrum of fluoride's effects.

69. I understand that EPA is asserting that systemic toxicity, as reflected by reduced body weight, may explain fluoride's observed effect on learning/memory in animals. The *Guidelines* provide that, "If several neurological signs are affected, but only at the high dose and in conjunction with other overt signs of toxicity, including systemic toxicity, *large decreases in body weight*, decreases in body temperature, or debilitation, there is less persuasive evidence of a direct neurotoxic effect."[62] The *Guidelines* further provide that "At doses causing moderate

[57] These concentrations of fluoride *ion* correspond to a concentration of approximately 10 to 50 mg/L of *sodium* fluoride, as there are 2.2 parts sodium for each 1 part fluoride.

[58] EPA (2018a).

[59] EPA (1998b), p. 1.

[60] Teng et al. (2018).

[61] For example, Güner et al. (2016); Liu et al. (2011); Yang et al. (2018a); Zhang et al. (2015a).

[62] EPA (1998a), p. 38.

maternal toxicity (i.e., 20% or more reduction in weight gain during gestation and lactation), interpretation of developmental effects may be confounded."[63] The fact that there is some effect on body weight, therefore, does not, by itself, negate a direct neurotoxic effect; the effect on body weight must be relatively large (i.e., >20%). While some of the animal studies on fluoride do show some body weight reductions, many do *not*—particularly at the lowest doses causing the effects. Systemic toxicity is thus an unlikely explanation of the neurotoxic effects reported.

2. NTP Systematic Review for Australian Government (2015)

70. In 2015, the National Toxicology Program (NTP) completed a systematic review of the animal literature on fluoride neurotoxicity and submitted a report to the Australian government.[64] The NTP limited its review to studies that have measured learning, memory, and other behavioral effects.[65] In total, the NTP identified 44 studies of learning and memory, 14 of which were excluded due to risk of bias from lack of randomization, lack of blinding at outcome assessment, or other design deficiencies.[66] From the remaining 30 studies, NTP concluded that there was "a moderate level-of-evidence for a pattern of findings suggestive of an effect on learning and memory in rats treated during development or adulthood."[67] Moderate level of evidence is the second highest level of evidence under NTP's 5-grade classification criteria.[68]

[63] EPA (1998a), p. 46.
[64] NTP (2015a).
[65] NTP (2015a), pp. 1, 28.
[66] In addition to PubMed, NTP searched several additional databases. NTP (2015a), p. 1.
[67] NTP (2015a), p. 1.
[68] NTP (2015a), p. 11. Under NTP's Hazard Identification Scheme, a chemical that has a moderate level of evidence of neurotoxicity in animals and a moderate level of evidence of neurotoxicity in humans is a "presumed" neurotoxicant (NTP 2015b, p. 67, Figure 8).

3. NTP Systematic Review (2016)

71. In 2016, the NTP published an updated version of its systematic review.[69] In the updated review, NTP identified an additional four studies on learning and memory, two of which were excluded for bias, resulting in a total of 32 studies for its analysis.[70] NTP maintained its conclusion that the animal evidence is “suggestive” that fluoride impairs learning and memory, but downgraded its confidence in the developmental studies to “low.”[71] NTP had less confidence in the developmental studies due to their general failure to control for litter effects, as well as the relatively few developmental studies that used fluoride concentrations lower than 25 mg/L in drinking water.[72]

72. The NTP identified several common methodological limitations with the learning and memory studies, including failure to rule out fluoride-induced motor effects as the cause of the apparent cognitive deficits; failure to control for “litter effects” in the developmental studies; lack of blinding; and lack of reported information on the study conditions, including the purity of the fluoride added to the water and the concentrations of fluoride in the rodent chow.

73. In contrast to NTP’s 2015 report, the 2016 report considered the absence of animal studies using 0.7 mg/L (the current recommended fluoride concentration for human drinking water[73]) to be an important limitation in the research in terms of its relevance to human exposure levels.[74]

69 NTP (2016).
70 NTP (2016), p. vi.
71 NTP (2016), p. vii.
72 NTP (2016), p. 57.
73 USDHHS (2015).
74 NTP (2016), pp. 55, 58.

4. Assessment of NTP's Review

74. The suggestion by NTP that rodent studies should use fluoride concentrations of 0.7 mg/L in order to be relevant to human exposures is at odds with EPA's approach to risk assessment.[75] As I discuss later, humans are considered much more sensitive to toxicants than are rats and mice, and the EPA has developed procedures to account for this increased sensitivity. The net effect of EPA's procedures is that what might initially seem to be a "high" dose in animal studies may be very relevant to assessing risk in humans at lower doses.

75. By limiting its review to studies investigating learning and memory, the NTP did not consider the much larger number of studies that have investigated neuroanatomical and neurochemical effects, endpoints that are more sensitive and also potentially less susceptible to bias associated with outcome assessment.

76. The NTP correctly identified a number of methodological limitations in the learning/memory studies. The lack of blinding in some studies, for example, does create some uncertainty because lack of blinding can bias results in the direction of the anticipated effect.[76] Some of the limitations, however, would not be expected to skew the results in a consistent direction across laboratories (e.g., lack of information on the purity of the fluoride compounds). Similarly, litter effects can produce false negatives as well as false positives, and can both inflate and deflate the true effect size.[77] The impact of these limitations on the reported results is thus unclear, particularly when considering that the studies also have limitations that will make it harder

[75] The principal author of the NTP study, Kristina Thayer, testified at her deposition that she is no longer comfortable with the assumption of a 1-to-1 equivalence between fluoride exposures in animals and humans; Thayer testified that she would approach the issue differently today, with greater attention to interspecies differences in toxicokinetics and toxicodynamics. (Thayer Deposition at 151:9-152:3, 302:21-303:23). These issues are discussed further below.

[76] Holman et al. (2015).

[77] Zorrilla (1997), p. 144; Lazic and Essioux (2013), p. 3.

to detect effects, including the absence of chronic studies and the absence of studies investigating neonatal exposures that are comparable to formula-feeding exposures in human infants, as discussed further below.

77. Although the NTP expressed concern about the difficulty of distinguishing fluoride's effects on learning/memory from its effects on the motor/sensory system, each of these effects is neurotoxic and a matter of concern.

5. Developmental Studies Published Since the NTP Review

78. Subsequent to the NTP's review, 11 additional developmental studies have reported learning and memory outcomes.[78] Ten of these studies found deficits in the fluoride-treated groups. Notably, the Bartos et al. studies, which controlled for litter effects, found impairments in learning and memory at a fluoride concentration of just 5 mg/L. I will discuss these studies further in the Quantitative Dose Response section below.

6. McPherson (2018) and Other "No Effect" Studies

79. McPherson et al. (2018) is the one developmental study published since the NTP review that did not find clear adverse effects on learning and memory, although it did find a significant increase in pain sensitivity (a neurotoxic effect).

80. There are several features of the McPherson study that may help to explain the absence of a clear effect on learning and memory. First, unlike the overwhelming majority of previous studies on fluoride neurotoxicity, the McPherson study used Long Evans Hooded rats, which some have suggested may have lower sensitivity to fluoride than other strains.[79] To date,

[78] Bartos et al. (2018; 2019); Chen et al. (2018a); Cui et al. (2017); Ge et al. (2018); McPherson et al. (2018); Sun et al. (2018); Wang et al. (2018a); Zhao et al. (2019); Zhu et al. (2017); Zhou et al. (2019).
[79] Elliott (1967).

three studies[80] have examined the effect of fluoride on learning in Long Evans rats, and all three have failed to find an effect.[81]

81. Second, in contrast to most of the other developmental studies, McPherson et al. did not start the exposure until the 6th day of gestation.[82] As pregnancy in rats lasts approximately 21 days, any effects due to exposures early in, or preceding, the pregnancy may not have been detected by McPherson's study design.

82. Third, the offspring in the McPherson study had virtually no fluoride exposure during the neonatal period because the rat pups were breastfed during the pre-weaning period. This is important because the fluoride content of breast milk in rats (as with other mammals, including humans) is negligible, even when the mother is consuming large quantities of fluoride.[83] The rats in the McPherson study thus missed a potentially key period of vulnerability (early infancy)—an important limitation given the widespread use of infant formula among human neonates.[84]

83. In addition to the McPherson study, three other studies (with weaker study designs) reported no neurotoxic effects from fluoride exposure.[85] These three studies include Whitford et al. and Pulungan et al. which started with adult animals, and an unusual study by Jia et al. which started at gestational day 9. All or part of the gestational period was thus missed in each of these studies.

[80] Elliott (1967); Varner et al. (1994); McPherson et al. (2018).

[81] The plausibility of strain-specific differences between Long Evans and other rats is supported by other research which has found that Long Evans Hooded rats have different sensitivities to teratogenic substances in utero than Sprague-Dawley rats (Kang et al. 1986).

[82] McPherson et al. (2018).

[83] Fluoride concentrations in mammalian milk are very low in comparison to the mother's fluoride intake, even when the mother's fluoride intake is quite high (NRC 2006, pp. 33, 36; Drinkard et al. 1985).

[84] This limitation is not unique to the McPherson study, as all other developmental studies on fluoride have failed to supplement the pup's exposure during the breastfeeding stage.

[85] Whitford et al. (2009); Pulungan et al. (2018); Jia et al. (2019).

84. According to the *Guidelines*, "To judge that an agent is unlikely to pose a hazard for neurotoxicity, the minimum evidence would include data from a host of endpoints that revealed no neurotoxic effects."[86] This evidence does not exist for fluoride. To the contrary, almost all studies, including McPherson et al. (2018), have reported adverse effects on at least one of the endpoints measured.

D. Other Considerations

1. Dose Response

85. The *Guidelines* recognize that "determining a hazard often depends on whether a dose-response relationship is present,"[87] and thus "dose-response evaluation is a critical part of the qualitative characterization of a chemical's potential to produce neurotoxicity."[88] Because "human studies covering a range of exposures are rarely available," the *Guidelines* state that the dose-response evaluation will typically be limited to animal data.[89]

86. In contrast to the chemicals that EPA has evaluated under the *Guidelines*, there is abundant dose-response data for fluoride from *human* studies. Most importantly, the ELEMENT and MIREC birth cohort studies have found linear dose-response relationships between maternal urinary fluoride and IQ in the offspring.[90] The linearity of the dose-response relationships in these studies was not simply assumed—it was scrutinized through several methods, which I understand Drs. Hu and Lanphear will be explaining as part of their testimony.

87. Dose-response trends have also been observed in cross-sectional studies as a function of childhood urine and serum fluoride levels, although these are inherently less certain.[91]

[86] EPA (1998a), pp. 55-56.
[87] EPA (1998a), p. 2.
[88] EPA (1998a), p. 50.
[89] EPA (1998a), p. 50.
[90] Bashash et al. (2017; 2018); Green et al. (2019).
[91] Cui et al. (2018); Ding et al. (2011); Xiang et al. (2011); Zhang et al. (2015b).

An important limitation with dose-response data from cross-sectional studies is that the exposures are tested after the effect (reduction in IQ) has occurred. The data, however, are not without value, as current exposures can be reflective of developmental exposures in areas with stable populations and stable water fluoride concentrations. In the Zhang study, for example, most of the children had been living in the same household and drinking from the same wells since birth.[92]

88. In addition to dose-response data from human studies, there is also considerable dose-response data from animal studies. A prerequisite for dose-response analysis in animal studies is that there be multiple treatment groups with different exposures to the test substance. Many of the animal studies on fluoride have used multiple treatment doses, and thereby permit evaluation of dose response. Of the studies published since the NRC review (summarized in Table A-1), 1 used four treatment doses, 17 used three treatment doses, and 16 used two treatment doses (in addition to the control groups). Of these 34 studies, 30 show visually apparent dose-response trends for at least one of the effects being investigated.

2. Neuroendocrine Effects

89. EPA's *Guidelines* recognize the relevance of a chemical's ability to alter the function of the thyroid gland.[93] According to the *Guidelines*, "the development of the nervous system is intimately associated with the presence of circulating hormones such as thyroid hormone."[94] A thyroid disturbance during a specific developmental period may cause a "nervous system deficit, which could include cognitive dysfunction, altered neurological development, or visual deficits, [depending] on the severity of the thyroid disturbance and the specific developmental period when exposure to the chemical occurred."[95] Elsewhere, EPA has recognized

[92] Zhang et al. (2015b), p. 4.
[93] EPA (1998a), p. 50.
[94] EPA (1998a), p. 50.
[95] EPA (1998a), p. 50.

that "thyroid hormones are essential for normal brain development in humans and that hypothyroidism during fetal and early neonatal life may have profound adverse effects on the developing brain."[96]

90. Thyroid toxicity may be a significant mechanism by which fluoride affects neurodevelopment. In 2006, the NRC had enough information to conclude that fluoride is an "endocrine disrupter" which may lower thyroid function.[97] Sodium fluoride was once prescribed as a therapeutic agent for *lowering* thyroid activity in cases of *hyper*thyroidism.[98] The NRC reported that fluoride can lower thyroid function at estimated average intakes of 0.05-0.13 mg/kg/day in humans with adequate iodine intake, and at estimated average intakes as low as 0.01 to 0.03 mg/kg/day in individuals with iodine deficiency.[99] Put differently, fluoride affects thyroid function at lower doses in people with iodine deficiency than in those with optimal intake of iodine.

91. Epidemiological research published subsequent to the NRC's report is consistent with and further supports NRC's findings. In 2018, Malin et al. reported a relationship between urinary fluoride and elevated TSH (thyroid stimulating hormone) among iodine-deficient adults in Canada, but not in the general population as a whole (excluding those with known thyroid disease and excluding pregnant individuals).[100] Elevated TSH is indicative of a decrease in thyroid function. Ten percent of women of child-bearing age in the US are iodine deficient.[101]

[96] EPA (2008a), p. 40, citing Morreale de Escobar et al. (2000) and Haddow et al (1999). See also EPA (2008b), p. 54, citing Morreale de Escobar et al. (2000). EPA's Science Advisory Board in 2013 found that "the most sensitive life stages are the fetus, neonates and infants because these are the stages when thyroid-dependent brain development occurs" (EPA 2013b, cover letter, p. 2).

[97] NRC (2006), pp. 262-263.

[98] Galletti and Joyet (1958).Consistent with this thyroid use (i.e., lowering thyroid function), fluoride exposure has been associated with hypothyroidism in animal and human studies (Hillman et al. 1979; Peckham et al. 2015; Yang et al. 2019).

[99] NRC (2006), pp. 262-263.

[100] Malin et al. (2018). Barberio et al. (2017) found no association between fluoride exposure and thyroid status, but the iodine-deficient part of the population was not specifically addressed.

[101] CDC (2008), Chapter 4a, pp. 91-100; see also Pearce (2015); Caldwell et al. (2011).

92. In 2015, a nationwide study from England reported a significant association between water fluoridation and increased prevalence of hypothyroidism.[102]

3. Toxicokinetics

93. Under the *Guidelines*, consideration should be given to the toxicokinetics of the chemical with "particular importance" given to the chemical's capacity to get through the blood-brain barrier.[103] The permeability of the blood brain barrier is particularly important when a chemical, such as fluoride, is able to make it through the placenta. Studies in humans have repeatedly demonstrated that fluoride crosses the placenta and reaches the fetus,[104] and thus it is generally accepted that "fluoride readily crosses the placenta."[105] In general, measured concentrations of fluoride in umbilical cord blood and in blood of neonates are similar to concentrations in maternal blood.[106] In short, the fluoride that a mother ingests will cause exposure to the fetus.

94. Fluoride is also known to cross the blood-brain barrier,[107] and passage of fluoride into the brain can be expected to be higher during the fetal and neonatal life stages when the blood brain barrier is not yet fully developed.[108] As the EPA has recognized, "Because the blood-brain barrier limits the passage of substances from blood to brain, in its absence, toxic agents can freely enter the developing brain."[109] Consistent with EPA's observation, the recent rat study by

[102] Peckham et al. (2015).
[103] EPA (1998a), p. 47.
[104] See for example, Feltman and Kosel (1961); Gedalia et al. (1964); Blayney and Hill (1964); Armstrong et al. (1970); Hanhijärvi et al. (1974); Forsman (1974); Shen and Taves (1974); Ron et al. (1986); Malhotra et al. (1993); Gupta et al. (1993); Brambilla et al. (1994); Shimonovitz et al. (1995).
[105] NRC (2006), p. 193.
[106] Feltman and Kosel (1961); Gedalia et al. (1964); Hudson et al. (1967); Armstrong et al. (1970); Hanhijärvi et al. (1974); Ron et al. (1986); Malhotra et al. (1993); Gupta et al. (1993); Shimonovitz et al. (1995).
[107] Geeraerts et al. (1986); Mullenix et al. (1995); Zhang et al. (2013c); Niu et al. (2015b).
[108] EPA (2009b), p. 58.
[109] EPA (2009b), p. 58.

McPherson et al. found sharply elevated concentrations of fluoride in the brain following prenatal exposure.[110]

4. Mode of Action

95. EPA's *Guidelines* recognize that hazard identification is strengthened by, but not dependent upon, an identifiable mechanism by which the chemical can exert neurotoxic effects.[111] For most of the chemicals for which EPA has established RfDs pursuant to the *Guidelines*, the mode of action has not been known (see Table 1). As noted recently by the NAS, "solid conclusions about causality can be drawn without mechanistic information, for example, when there is strong and consistent evidence from animal or epidemiology studies."[112] The NAS added that "mechanistic frameworks today could probably be completed for only a few chemicals."[113]

96. Several plausible mechanisms—both indirect and direct—have been identified that could help explain the neurotoxicity of fluoride.

97. *Indirect Mechanisms*: Depression of thyroid function is likely a principal indirect mechanism and could account for some of the neurotoxic effects reported in the literature. A thyroid mechanism is particularly plausible as a cause of IQ loss among offspring born to women with suboptimal iodine intakes.

98. *Direct Mechanisms* A recent study by Zhao et al. provides *in vitro*, *in vivo*, and epidemiological data that, together, suggest that disturbances in hippocampal mitochondrial dynamics (marked by fission inhibition and fusion promotion) play an important role in fluoride-induced cognitive loss.[114] The hippocampus is an important region in the brain for learning and

[110] McPherson et al. (2018).
[111] EPA (1998a), pp. 10, 53.
[112] NAS (2018), p. 9.
[113] NAS (2018), p. 9.
[114] Zhao et al. 2019.

memory, and many of the studies investigating the neuroanatomical and neurochemical effects of fluoride exposure have identified adverse effects in this region (see Table A-1). Other potential modes of action have also been identified, including signaling disruption, oxidative stress, and selective reductions in nicotinic receptors.[115]

5. *In Vitro* Studies

99. EPA's *Guidelines* also call for consideration of *in vitro* data. While positive *in vitro* data are not sufficient, by themselves, to demonstrate a neurotoxic hazard in humans, the existence of such data helps enhance the reliability of *in vivo* data.[116]

100. Fluoride's ability to damage brain cells has been documented in *in vitro* experiments. While most of the *in vitro* studies have used high concentrations that are unlikely to be present in the human brain, several studies have examined environmentally realistic fluoride concentrations. Gao et al. found increased lipid peroxidation and reduced α7 nicotinic acetylcholine receptors in brain cells at fluoride concentrations (i.e., 9.5 parts per billion) that are commonly found in the blood of people living in fluoridated areas.[117] Increases in markers of neuroinflammation have also been found at low concentrations.[118] Under the *Guidelines*, these data do not demonstrate a hazard in humans, but they do enhance the reliability of the animal studies, as similar effects have been reported in fluoride-treated rodents.[119]

6. Validity of the Database

101. Under the *Guidelines*, the validity of the database should be evaluated by assessing

[115] Bartos et al. (2018); Chen et al. (2003; 2018a); Gao et al. (2008); Liu et al. (2010); Long et al. (2002); Shan et al. (2004); Zhang (2017b); Zhu et al. (2017).
[116] EPA (1998a), p. 49.
[117] Gao et al. (2008), Figures 1A, 3A.
[118] Goschorska et al. (2018).
[119] Bartos et al. (2018); Dong et al. (2015); Yang et al. (2018a); Yan et al. (2016); Zhao et al. (2019).

the content validity, construct validity, concurrent validity, and predictive validity of the data.[120]

102. *Content validity* addresses "whether the effects result from exposure."[121] This factor weighs decisively in favor of a neurotoxicity hazard determination for fluoride. The NRC concluded that fluoride interferes with the brain,[122] and the evidence has gotten stronger since. Kristina Thayer, the Director of EPA's IRIS Division, has explained that "experimental animal studies are designed to let you draw causal inferences," and that the animal studies show that fluoride damages the brain at some level of exposure.[123] Further, while the human cross-sectional studies are limited in their ability to produce causal inferences, the *Guidelines* provide that prospective cohort studies permit "direct estimates of risk attributable to a particular exposure."[124]

103. *Construct validity* addresses whether the neurologic effects that have been observed "are adverse or toxicologically significant."[125] This factor is satisfied in the fluoride database. Animal studies have linked fluoride to learning and memory deficits, which are an adverse effect upon which EPA has established reference doses for other neurotoxicants (e.g., BDE-153).[126] Further, the human epidemiological data have linked fluoride with IQ detriments, including an approximate 5 to 6 point drop in IQ as maternal urinary fluoride increased from 0 to 1 mg/L.[127] EPA has recognized that a loss of a single IQ point is associated with a loss in lifetime earnings,[128] and EPA's Clean Air Science Advisory Council has stated that "a population loss of 1-2 IQ points

[120] EPA (1998a), pp. 10-11.
[121] EPA (1998a), pp. 10-11.
[122] NRC (2006), p. 222.
[123] Thayer Deposition at 225:8-15, 226:13-16, 270:23-25.
[124] EPA (1998a), p. 17.
[125] EPA (1998a), pp. 10-11.
[126] EPA (2008c), p. 36. Effects on memory were also noted in the RfD determination for BDE-99 (EPA 2008b, p. 27).
[127] Bashash et al. (2017); Green et al. (2019).
[128] EPA (2008e), p. 5-28.

is highly significant from a public health perspective" and should be prevented in 99.5% of the population.[129]

104. *Concurrent Validity* addresses "whether there are correlative measures among behavioral, physiological, neurochemical, and morphological endpoints.[130] Studies have correlated fluoride's cognitive effects in animals with various neurochemical and neuroanatomical changes,[131] and a few studies have correlated fluoride-associated cognitive loss in humans with increased TSH and alterations in mitochondrial dynamics.[132] For example, Zhao et al.[133] reported lower circulating levels of a mitochondrial protein (fission-related protein-1, Fis1) in children from high fluoride areas (compared with children in low fluoride areas), and higher circulating levels of a second mitochondrial protein (mitofusin-2, Mfn2) in the same children. The levels of circulating Fis1 were positively associated with children's IQ scores, while the levels of circulating Mfn2 were negatively associated with the IQ scores. In addition, several plausible mechanisms of fluoride neurotoxicity have been described (discussed above).

105. *Predictive validity* addresses "whether the effects are predictive of what will happen under various conditions."[134] The condition of perhaps greatest interest with respect to prediction of fluoride neurotoxicity is exposure during the prenatal period. Studies in both animals and humans have, with one exception,[135] reported neurologic effects following prenatal exposure. The database, therefore, does have some degree of predictive validity, although further research remains necessary to determine to what extent other conditions (e.g., nutrition, genetics, neonatal

[129] Federal Register (2008), p. 67000.
[130] EPA (1998a), pp. 10-11.
[131] For example, see Bartos et al. (2018); Zhao et al. (2019); Zhou et al. (2019).
[132] For example, Zhang et al. (2015b); Zhao et al. (2019).
[133] Zhao et al. (2019).
[134] EPA (1998a), pp. 10-11.
[135] McPherson et al. (2018).

exposure, and kidney function) may modify or predict outcomes. Exposure during the early postnatal period also requires further research.

7. Data Gaps

106. EPA's *Guidelines* point to the need to address "significant data gaps."[136] One of the major data gaps for fluoride is the lack of research on the impact of fluoride during the neonatal and early infancy period. EPA has recognized that the neonatal period represents a critical window of vulnerability to neurotoxicants,[137] yet most developmental rodent studies do not address neonatal exposures to fluoride (due to exclusive breastfeeding of the rat or mouse pups and absence of gavage exposures). Other data gaps include the absence of long-term animal studies, and the scarcity of epidemiological research into fluoride's neurologic effects in the elderly. Data gaps also remain with respect to how the dose which causes neurologic effects varies across susceptible subsets of the population, including those with nutrient deficiencies, genetic polymorphisms, kidney disease, and the elderly.

E. Conclusion: There Is Sufficient Evidence that Neurotoxicity Is a Hazard of Fluoride

107. The large and substantial body of evidence that now exists for fluoride, from both animal and human studies, satisfies EPA's "sufficient evidence" standard for hazard determination.

108. The *Guidelines* provide that "the minimum evidence sufficient would be data on a single adverse endpoint from a well-conducted study."[138] The *Guidelines* also recognize that prospective cohort studies are the optimal type of epidemiological study that permit direct

[136] EPA (1998a), p. 12.
[137] See for example, EPA (2008a), p. 42.
[138] EPA (1998a), p. 55.

estimates of risk. The minimum evidence threshold is thus met for fluoride because there is not just one, but *four* high-quality prospective cohort studies that support the endpoint of IQ loss, and another high-quality prospective cohort study that supports the endpoint of inattention.[139]

109. EPA's *Guidelines* also permit consideration of the collective evidence when no study, by itself, is sufficient to permit a hazard determination. This, again, supports a hazard determination for fluoride because the prospective studies are most compelling when viewed in the context of (i) the toxicokinetic data showing that fluoride crosses the placenta and enters the fetal brain; (ii) animal data showing neurochemical and neuroanatomical damage following fluoride exposure; (iii) animal data finding impairments in learning and memory following prenatal exposure to fluoride; (iv) cross-sectional studies consistently finding reductions in IQ in communities with elevated fluoride exposure; (v) *in vitro* studies reporting effects on brain cells at concentrations of fluoride found in the blood of individuals living in fluoridated communities; and (vi) animal and human studies finding that fluoride can depress thyroid function, a known risk factor for neurodevelopmental harm.

110. Based on the collective data—which are far more robust than the data EPA has relied upon for prior hazard determinations—I conclude with a reasonably high degree of confidence that neurotoxicity is a hazard of fluoride exposure.

VI. QUANTITATIVE DOSE RESPONSE

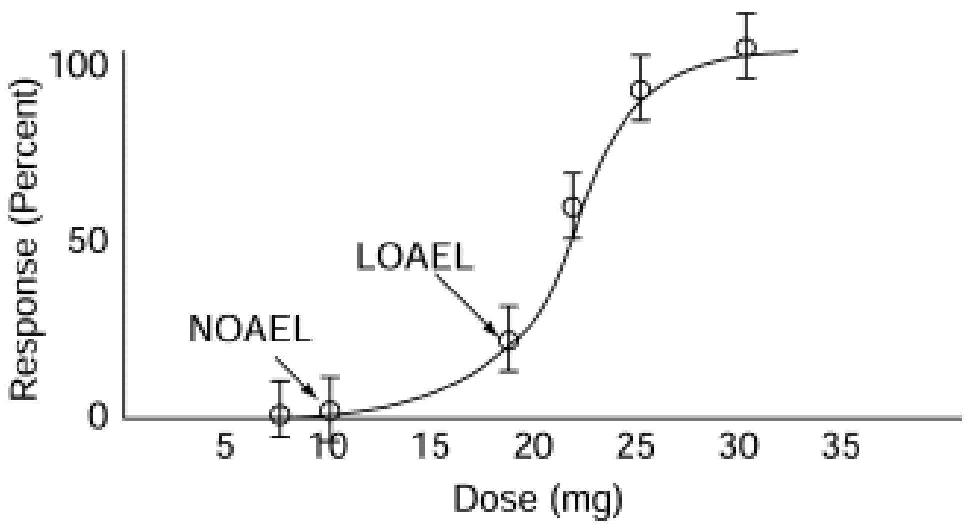
111. If a chemical is identified as posing a neurotoxic hazard, EPA's *Guidelines* call for a quantitative dose-response analysis to determine the reference dose (RfD). The RfD is "an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the

[139] Bashash et al. (2017; 2018); Green et al. (2019); Till et al. (2020); Valdez-Jiménez et al. (2017).

human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime."[140]

112. In the Quantitative Dose Response analysis, human or animal data are assessed to determine an appropriate "Point of Departure" (POD). As the name implies, the Point of Departure (POD) is the datapoint from which the RfD is ultimately derived. The POD can be one of three types of values: the No Observed Adverse Effect Level (NOAEL), the Lowest Observed Adverse Effect Level (LOAEL) or the Benchmark Dose Level (BMDL). While EPA now has a preference for using BMD, it still uses both the NOAEL and LOAEL approaches in its assessments.

113. The following figure provides a visual illustration of the difference between a NOAEL and LOAEL on a dose-response curve.



A. Basis for Using Animal Data

114. When human data are available, EPA's preference is to use human data for the Point of Departure.[141] In the case of fluoride, the recent prospective cohort studies[142] with individual-level biomonitoring data provide suitable data for this purpose. If one had to choose,

140 EPA (1998a), p. 57. See also EPA (2009a).
141 EPA (2018a), p. 2-1.
142 Bashash et al. (2017; 2018); Valdez-Jiménez et al. (2017); Green et al. (2019).

therefore, between deriving the POD for fluoride from the human or animal data, *the choice would clearly be to use the human data*. But this does not mean that the animal data are without value. In EPA's assessment of methylmercury, for example, the EPA derived its RfD from human prospective cohort data, but it also considered what the RfD would be if it were derived from the animal literature.[143] As the EPA noted, "[i]t is informative to compare RfDs derived from animal studies with those derived from the epidemiological literature."[144] In the case of methylmercury, the animal-based RfD supported the human-based RfD, and EPA cited this as a factor that increased its "confidence" in the assessment.[145]

115. In this case, Dr. Philippe Grandjean conducted a dose-response analysis of the prospective cohort data where he derived a BMDL. To avoid duplication of Dr. Grandjean's effort, and to determine whether Dr. Grandjean's BMDL is consistent with potential RfDs derived from the animal data, I focused my assessment on the animal literature.

116. In this assessment, I did not seek to select a single value for the RfD. Instead, I sought to identify the full range of RfDs that can be derived, including the *least* protective. If human exposures exceed RfDs that use non-protective assumptions, there would be greater confidence that a human risk does, in fact, exist.

117. There are several considerations that support the use of animal data to establish an RfD for fluoride. First, EPA has used animal studies as the principal studies for each of the neurotoxicity risk assessments it has thus far conducted under the *Guidelines*. Second, EPA has used impairment in learning and memory in rodents as the adverse effect upon which to base the RfD for other chemicals,[146] thus this is an accepted endpoint to use in deriving an RfD. Third, a

[143] EPA (2001), pp. 17-18.
[144] EPA (2001), pp. 17.
[145] EPA (2001), pp. 18-19.
[146] For example, BDE-153 (EPA 2008c, p. 36).

substantial number of animal studies of fluoride neurotoxicity have used 2 or 3 treatment groups (in addition to control groups), and EPA has found this to be sufficient for identifying Points of Departure,[147] including in animal studies with as few as 10 rats per group (2-Hexanone).[148]

B. Selecting Points of Departure

118. In the literature review discussed earlier, 37 rodent studies were identified that have investigated fluoride's impact on learning and memory since the NRC report (Table A-2). All but 3 of these studies found adverse effects in the fluoride-treated rodents, including 16 of the 17 studies that investigated prenatal fluoride exposures. Since the prenatal period represents a point of heightened vulnerability to neurotoxicants, the prenatal studies are a logical candidate for the point of departure.

119. To avoid studies at high risk of bias, the three studies that did not specifically mention using a randomization procedure were excluded from further consideration.[149] Further, in order to focus the analysis on those studies best suited for identifying a Point of Departure (POD), four studies that only used one treatment dose were excluded.[150]

120. Table 2 and the figures below summarize the 10 prenatal studies that remained for POD consideration. Most of the studies used a similar dosing regimen with 2 or 3 treatment groups and at least 10 rodents per group, which is consistent with several of the principal studies that EPA has used to establish an RfD. The figures show the lowest-observed and no-observed effect levels in each study and help to visually compare the data across studies.

121. Nine of the 10 studies found dose response trends for one or more effects, which

[147] EPA (2000b; 2008a; 2008c; 2008d; 2009b).
[148] EPA (2009b).
[149] Bera et al. (2007); Basha et al. (2011b); Ge et al. (2018).
[150] Niu et al. (2014); Banala and Karnati (2015); Dong et al. (2015); Zhu et al. (2017).

adds confidence to a causative role of the fluoride treatment.[151] Six of these studies also provide data on the body weights of the pups, and no bodyweight changes were seen in any of the studies at the lowest concentrations producing the effects.[152] Only one of the six studies found any bodyweight changes among pups in the higher-dose groups.[153] Of the two studies that reported maternal weight, neither found any changes.[154]

122. One limitation with these studies is that only three of them specifically mention controlling for litter effects,[155] which introduces some uncertainty since the failure to control for litter effects can result in false positives, as well as false negatives.[156] While a source of uncertainty, the failure to control for litter effects does not preclude use for risk assessment purposes. As noted earlier, EPA has used studies that do not control for litter effects as the principal studies upon which it has based RfDs for developmental neurotoxicity.[157]

[151] See for example, Jiang et al. (2014b), Table 3; Cui et al. (2017), Table 3; Chen et al. (2018a), Figure 1d,e; Sun et al. (2018), Tables 2 and 3; Wang et al. (2018a), Figure 4b,c; Zhao et al. (2019), Figure 5e.

[152] Bartos (2018; 2019); Cui et al. (2017); Jiang et al. (2014b); Wang et al. (2018a). The study by McPherson (2018) also showed no changes in bodyweight, although it did not find effects on learning/memory.

[153] Jiang (2014b) found reduced body weight gain among the pups in the 23 mg/L and 45 mg/L groups.

[154] Bartos (2018; 2019).

[155] Bartos et al. (2018; 2019); McPherson et al. (2018).

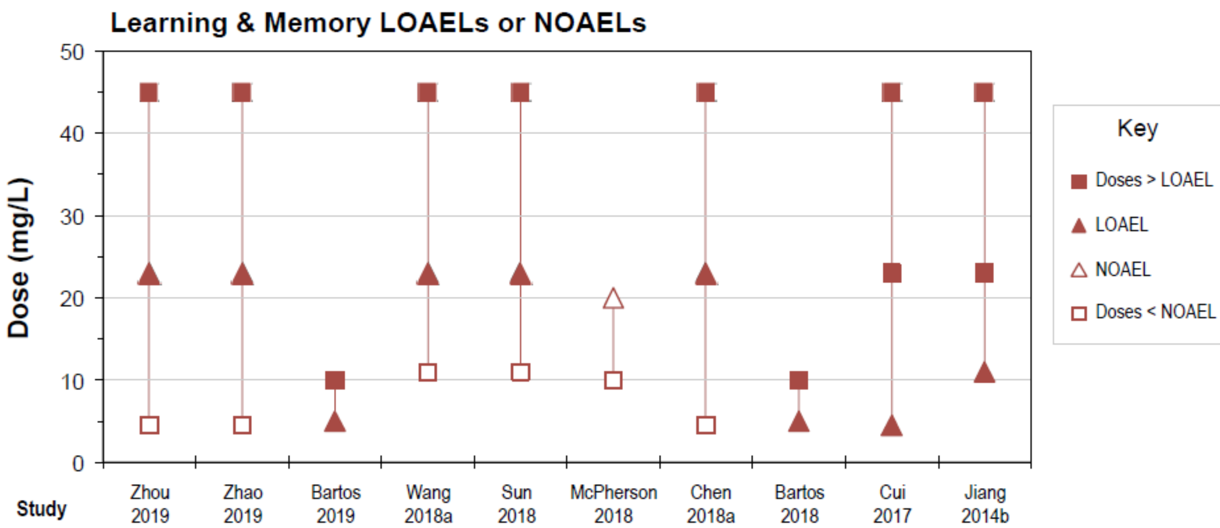
[156] Zorrilla (1997), p. 144; Lazic and Essioux (2013), p. 3.

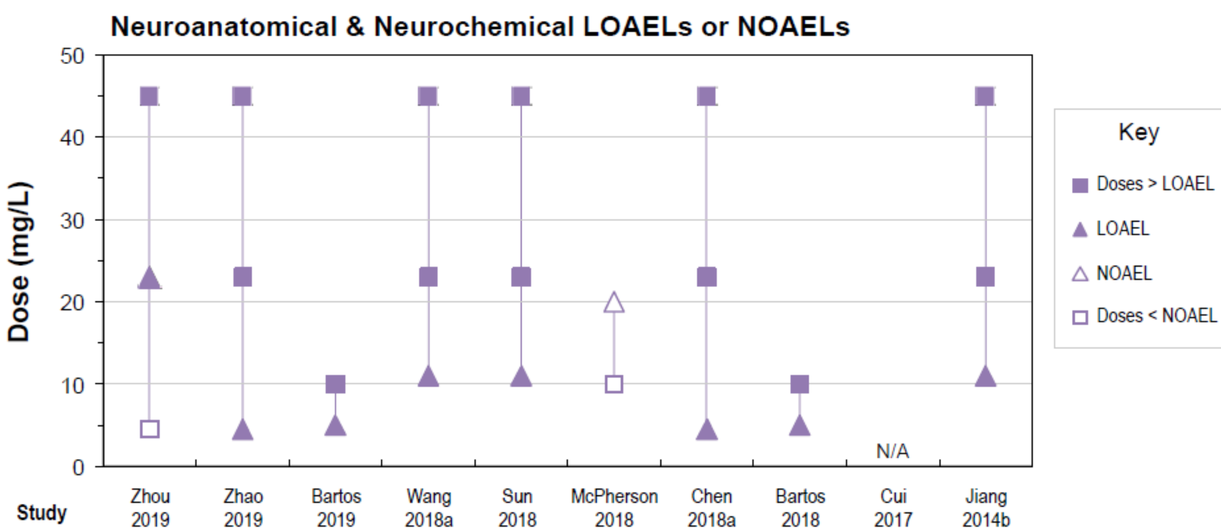
[157] See for example EPA (2008a), pp. 44, A-4; EPA (2008b), pp. 59, A-3; EPA (2008c), pp. 32, A-3.

Table 2. Examples of rodent studies of prenatal exposure to fluoride that could provide LOAELs or NOAELs for neurotoxicity.

Study	Animal	Exposure period	[F^-] in drinking water[a] (mg/L)	Number of animals per group (n)	Learning and memory		Neuroanatomical or neurochemical effects	
					LOAEL (mg/L)	NOAEL (mg/L)	LOAEL (mg/L)	NOAEL (mg/L)
Zhou et al. (2019)	Rats, Sprague-Dawley	Prenatal[b] + 6 months	4.5, 23, 45	6	23	4.5	23	4.5
Zhao et al. (2019)	Rats, Sprague-Dawley	Prenatal[b] + 60 days	4.5, 23, 45	5	23	4.5	4.5	None
Bartos et al. (2019)	Rats, Wistar (female)	Prenatal + 21 days	5, 10	9-10	5	None	5	None
Wang et al. (2018a)	Mice, ICR (female)	Prenatal (from day 7) + 21 days	11, 23, 45	15	23	11	11	None
Sun et al. (2018)	Mice, Kunming	Prenatal + 21 days	11, 23, 45	6	23	11	11	None
McPherson et al. (2018)	Rats, Long Evans Hooded (male)	Prenatal (from day 6) + 90 days	10, 20[c]	11-23	None	20	None	20
Chen et al. (2018a)	Rats, Sprague-Dawley	Prenatal[b] + 6 months	4.5, 23, 45	6	23	4.5	4.5	None
Bartos et al. (2018)	Rats, Wistar (female)	Prenatal + 21 days	5, 10	9-10	5	None	5	None
Cui et al. (2017)	Rats, Sprague-Dawley	Prenatal[b] + 60 days	4.5, 23, 45	12	4.5	None	N/A	N/A
Jiang et al. (2014b)	Rats, Sprague-Dawley	Prenatal[b] + 2 months	11, 23, 45	12	11	None	11	None

[a] Treatment groups in addition to the control group.
[b] Exposure of the mother began before pregnancy.
[c] Animals were given 0, 10, or 20 mg/L fluoride in drinking water, plus 3.24 ppm fluoride in feed. An additional control group had 0 mg/L fluoride in drinking water plus 20.5 ppm fluoride in feed (McPherson et al. 2018).





123. Based on the dose-response data from these studies, the following values could be used as the Point of Departure.

124. **LOAEL of 5 mg/L**: The lowest observed adverse effect levels in the studies were fluoride concentrations of 4.5 and 5 mg/L. Of the six studies that used this concentration, three

found adverse effects on learning,[158] and two of the other three studies, which did not find effects on learning, *did find alterations in the brain.*[159] Two of the three studies reporting effects on learning at 5 mg/L controlled for litter effects, which suggests that the failure to control for litter effects is unlikely to explain the reported effects at this concentration.[160] A 5 mg/L LOAEL was selected, therefore, as one of the Points of Departure (PODs) for learning impairment from prenatal fluoride exposure.

125. **LOAEL of 23 mg/L**: Seven of the 10 studies used 23 mg/L as one of the treatment doses, and all 7 of these studies found impaired performance on the cognitive tests, with 6 of the 7 studies finding changes in the brain as well. 23 mg/L appears, therefore, to be a reliable "Observed Adverse Effect Level," particularly in light of the six studies (discussed above) which found adverse effects at < 5 mg/L. Although not the *lowest* observed effect level, it is assumed to be one for purposes of this Point of Departure.

126. **LOAEL of 45 mg/L**: As can be seen in the above figures, 45 mg/L is clearly an "observed adverse effect level," just as it has been in many other animal studies on fluoride neurotoxicity. It would be difficult to justify selecting 45 mg/L as the LOAEL because it is the *highest* observed adverse effect level in this group of studies, not the *lowest*. Nevertheless, for purposes of capturing the broadest possible range of RfDs that can be derived from the animal literature, a 45 mg/L LOAEL was selected as one of the Points of Departure.

[158] Cui et al. (2017); Bartos et al. (2018; 2019). In the Chen study (which had only six rats per group), there is some indication of an effect on learning in the 4.5 mg/L group, albeit not statistically significant (Chen et al. 2018a, Figure 1).

[159] Chen et al. (2018a); Zhao et al. (2019). Other studies have also found alterations in the brain at ≤ 5 mg/L, including Liu et al. (2010; 2011); Zhang et al. (2015a); Niu et al. (2018a); Varner et al. (1998); and Yu et al. (2019).

[160] Bartos et al. (2018; 2019).

127. **<u>N</u>OAEL of 11 mg/L**: Four of the prenatal studies used 10 or 11 mg/L for the low-dose group.[161] The two studies of mice failed to find a significant effect on learning at this level,[162] and, as such, 11 mg/L could be selected as a NOAEL. The fact that the two studies that did not find effects at 11 mg/L found them at higher concentrations (23 and 45 mg/L) would be a factor weighing in favor of this choice, as the animal models were sensitive enough to find an effect. It bears considering, however, that the two studies finding no effects on learning at 11 mg/L did find alterations in the brain at this level,[163] which is consistent with 11 mg/L being a LOAEL, rather than a NOAEL. However, for purposes of reflecting the spectrum of RfDs that can be derived from the animal literature, 11 mg/L was treated as a NOAEL for one of the PODs.

128. **<u>N</u>OAEL of 20 mg/L**: The highest possible NOAEL that can be selected from these prenatal studies is the 20 mg/L no-effect finding from McPherson et al.[164] As discussed earlier, there are limitations with the McPherson study that may have made it less sensitive to detecting an effect, including strain of rat used and lack of first trimester exposure. Further, the study did find an adverse neurotoxic effect in the 20 mg/L group (i.e., increased pain sensitivity), and, as such, 20 mg/L is not a true NOAEL in the study. Nevertheless, for the purpose of illustrating the upper-bound range of RfDs that can be derived from the animal literature, a 20 mg/L NOAEL will be treated as a POD for the analysis.

C. Conversion of POD Concentrations (mg/L) to Doses (mg/kg/day)

129. Reference Doses (RfDs) are expressed in terms of dose (i.e., milligrams per kilogram of bodyweight, or mg/kg/day), not in terms of water concentration. To calculate RfDs from the Points of Departure, therefore, the unit of measurement (i.e., water fluoride concentration)

[161] Wang et al. (2018a); Sun et al. (2018); McPherson et al. (2018); Jiang et al. (2014b).
[162] Sun et al. (2018); Wang et al. (2018a).
[163] Sun et al. (2018); Wang et al. (2018a).
[164] McPherson et al. (2018).

needs to be converted into a dosage metric.

130. The NTP's 2016 review provides data that facilitate this analysis.[165] In its review, the NTP estimated the doses for dozens of rodent studies by using EPA's default water consumption rates and body weight data for the species, strain, and sex of the animals studied.[166] A review of NTP's data shows that the average ratio of fluoride concentration (mg/L) to intake rate or dose (mg/kg/day) is 6.8, and that this ratio is generally higher for rats (typically 6 to 10) than for mice (typically 3.8 to 5). For purposes of this analysis, the low end of this range was chosen for each species (6 for rats, 3.8 for mice). The practical effect of selecting the low-end of this range, is that the estimated doses will likely *overestimate* the actual dose, and thereby *inflate* the RfDs derived from these Points of Departure.[167] The net result of this non-conservative approach will be RfDs that are *less* protective of human health.

D. Selecting the Uncertainty Factors

131. Consistent with EPA's standard risk assessment procedures,[168] the *Guidelines* provide that "uncertainty factors" (UFs) should be applied to the point of departure (POD) to ensure that the resulting RfD is protective of health.[169]

132. Uncertainty factors are applied to account for expected variations in susceptibility among humans (*intra*species variability, or UF_H), expected differences in susceptibility between animals and humans (*inter*species variability, or UF_A), and, where applicable, differences in the

165 NTP (2016), Appendix 19.
166 NTP (2016), p. 118.
167 For example, using this method gives an intake rate of 0.83 mg/kg/day for rats for the 5 mg/L fluoride concentration (Table 5). However, Bartos et al. (2018; 2019) give an estimate of 0.6 mg/kg/day for their rats at this fluoride concentration. Using the same approach described below with Table 5 for a LOAEL of 5 mg/L, equivalent to an intake rate of 0.6 mg/kg/day, gives an RfD of 0.0005, compared with 0.0007 in Table 5.
168 EPA (2018a), pp. xvii-xxiv.
169 EPA (1998a), pp. 58-59.

length of exposure between the study and human conditions (subchronic to chronic, or UF_S), research gaps in the overall database (database deficiency, UF_D), and converting from a LOAEL to a NOAEL.[170] These uncertainty factors are "typically multiples of 10," although they can be reduced to factors of 3 or 1 if warranted by available information.[171,172]

1. <u>*Intra*species Variability (UF_H)</u>

133. EPA recognizes that susceptibility to toxic substances is not uniform across the human population, and that due to differences in *toxicokinetics* and/or *toxicodynamics* some subsets of the population will be more vulnerable to harm than others.[173]

134. *Toxicokinetics* refers to the "processes which determine the extent and duration of exposure of the target organ or site of toxicity to the active chemical species," while *toxicodynamics* refers to the "processes involved in the translation of such exposure of the target organ or site of action into the generation of a toxic effect."[174] Put more simply, toxicokinetics governs how much of the chemical gets to the target site (i.e., access), while toxicodynamics governs how much of the chemical is necessary at the target site to cause the adverse effect (i.e., sensitivity).

135. If there are no chemical-specific data on toxicokinetics and toxicodynamics, EPA uses a default UF_H of 10.[175] This default factor of 10 is "considered to be appropriate in the absence of convincing data to the contrary."[176] Consistent with this, EPA has used a UF_H of 10 in each of

[170] EPA (1998a), p. 59; EPA (2018a), p. xxii; EPA (2016), pp. xix-xx.
[171] EPA (1998a), p. 59; EPA (2018a), p. xxii; EPA (2016), p. xix.
[172] As discussed by Martin et al. (2013), default uncertainty factors, while sometimes viewed as overly protective, do not represent worst-case situations and cannot be safely assumed to be adequately protective of the most exposed individuals or the most susceptible individuals, nor can they be safely assumed to be protective for effects of mixtures of chemicals.
[173] EPA (2011b), p. 14; EPA (2016), p. 2-15; EPA (2018a), p. 2-12.
[174] Renwick (1993), p. 276.
[175] EPA (2018a), pp. 2-12, 2-13.
[176] EPA (2013a), p. 5-17.

the nine risk assessments where it has established an RfD or RfC pursuant to the *Guidelines* (Table 3).

136. In the case of fluoride, there is evidence that affirmatively demonstrates substantial variability in how humans respond to fluoride, including differences in retention (toxicokinetics) and differences in response (toxicodynamics). These data are discussed below in the Risk Characterization. While the magnitude of this variability is difficult to quantify, the data *support* the need for an uncertainty factor as opposed to providing "convincing data" *against* one. Accordingly, pursuant to standard EPA procedure, a value of 10 for UF_H was assigned.

Table 3. Summary of RfDs or RfCs developed in compliance with EPA's Guidelines for Neurotoxicity Risk Assessment.

Chemical	LOAEL	NOAEL	Uncertainty Factors: UF_H	UF_A	UF_S	UF_D	Composite	RfD or RfC[a]	Reference
BDE-47	10.5 mg/kg	0.7 mg/kg	10	10	3	10	3000	0.1 µg/kg/day	EPA (2008a)
BDE-99	0.8 mg/kg	0.4 mg/kg	10	10	3	10	3000	0.1 µg/kg/day	EPA (2008b)
BDE-153	0.9 mg/kg	0.45 mg/kg	10	10	3	10	3000	0.2 µg/kg/day	EPA (2008c)
BDE-209	20.1 mg/kg	2.22 mg/kg	10	10	3	1	300	7 µg/kg/day	EPA (2008d)
Chlorine Dioxide and Chlorite	6 mg/kg/day	3 mg/kg/day	10	10	1	1	100	0.03 mg/kg/day	EPA (2000b)
2-Hexanone	143 mg/kg/day	Not observed	10	10	1	10	1000	0.005 mg/kg/day	EPA (2009b)
Methanol	1000 ppm ($1310 mg/m^3$)	500 ppm ($655 mg/m^3$)	10	3	1	3	100	$20 mg/m^3$	EPA (2013a)
RDX	8 mg/kg/day	4 mg/kg/day	10	3	1	10	300	0.004 mg/kg/day	EPA (2018a; 2018h)
Trimethylbenzenes	$492 mg/m^3$	$123 mg/m^3$	10	3	3	3	300	0.01 mg/kg/day	EPA (2016)

[a] Where EPA established both an RfD (mg/kg/day) and an RfC (mg/m^3) for a chemical, the RfD is presented.

Table 4. Comparison of $BW^{1/1}$ and $BW^{3/4}$ in estimating oral exposure in humans from a 10 mg/kg exposure to rats, mice, and a dog.[a]

Absolute animal intake or administered dose	Species	BW(h)/BW(a)	Scaling = $BW^{1/1}$: BW scaling factor	Scaling = $BW^{1/1}$: BW scaled human intake or oral dose (mg/kg)	Scaling = $BW^{3/4}$: BW scaling factor	Scaling = $BW^{3/4}$: BW scaled human intake or oral dose (mg/kg)
0.25 mg / 0.025 kg	Mouse	70 / 0.025 = 2800	$2800^{1/1} = 2800$	(2800 × 0.25 mg = 700 mg) 700 mg / 70 kg = 10 mg/kg	$2800^{3/4} = 385$	(385 × 0.25 mg = 96 mg) 96 mg / 70 kg = 1.4 mg/kg
2.5 mg / 0.25 kg	Rat	70 / 0.25 = 280	$280^{1/1} = 280$	(280 × 2.5 mg = 700 mg) 700 mg / 70 kg = 10 mg/kg	$280^{3/4} = 68$	(68 × 2.5 mg = 170 mg) 170 mg / 70 kg = 2.4 mg/kg
120 mg / 12 kg	Dog	70 / 12 = 5.8	$5.8^{1/1} = 5.8$	(5.8 × 120 mg = 700 mg) 700 mg / 70 kg = 10 mg/kg	$5.8^{3/4} = 3.7$	(3.7 × 120 mg = 444 mg) 444 mg / 70 kg = 6.4 mg/kg

[a] Taken from Table A-1 in EPA (2011b), p. 29.

2. *Inter*species Variability (UF_A)

137. EPA recognizes that susceptibility to toxic substances can differ across species. As with human-to-human variability, animal-to-human variability is also rooted in principles of toxicokinetics and toxicodynamics.

138. To adjust for differences in toxicokinetics between animals and humans, EPA has developed a hierarchical framework of approaches for ascertaining the "human equivalent dose" (HED) of doses given to animals.[177] EPA's "optimal" approach for determining the HED is to use a physiologically based toxicokinetic model (PBTK).[178] Where a PBTK model is not available, the "intermediate" approach is to use chemical-specific information that, while falling short of a full PBTK model, provides some reliable guidance.[179] Where there is no reliable chemical-specific information on kinetics, EPA uses a default allometric scaling method.[180]

139. Allometric scaling is "scaling of physiological rates or quantities to relative growth and size (mass or volume) of one animal species relative to another species."[181] Under EPA's recommended method for allometric scaling (BW¾ Method), the HED equates to 24% of the dose given to rats, and 14% of the dose given to mice (see Table 4 above).[182]

140. The BW¾ Method "predominantly addresses factors involved in estimating toxicokinetics, as well as some toxicodynamic factors."[183] EPA thus maintains a residual default UF of 3 to allow for residual uncertainty from toxicodynamics, unless there is chemical-specific information available.[184]

[177] EPA (2011b), pp. 18-21; EPA (2018a), p. 2-10.
[178] EPA (2011b), p. 19.
[179] EPA (2011b), p. 19.
[180] EPA (2011b), p. 19.
[181] EPA (2011b), p. 1.
[182] EPA (2011b), p. 29, Table A-1; EPA (2018a), p. 2-12.
[183] EPA (2011b), p. 17.
[184] EPA (2011b), p. 21; EPA (2016), p. 2-15; EPA (2018a), pp. 2-12, 2-13.

141. The following factors were considered to account for interspecies differences in both the toxicokinetics and toxicodynamics of fluoride.

142. *Toxicokinetic Considerations:* A full PBTK model has not yet been developed for fluoride that would allow for the calculation of HEDs from doses given to animals. As such, EPA's preferred approach for controlling for interspecies toxicokinetics is not available. By contrast, there is chemical-specific information for fluoride that could support application of EPA's intermediate approach. As discussed by the NRC, rats require higher levels of fluoride in their water to achieve the same level of fluoride in their blood.[185] Dunipace estimated that rats require about 5 times more fluoride in water than humans to reach the same plasma concentration of fluoride,[186] while Den Besten's team has reported a larger margin for mice, with a difference of about a factor of 10.[187] The data from Dunipace and Den Besten support a toxicokinetics adjustment of 5 for rats and 10 for mice, which are slightly *higher* than, but roughly consistent with, the adjustments under the default BW ¾ Method (4 for rats, 7 for mice). The chemical-specific information for fluoride thus supports the general validity of the BW¾ Method, but would be more protective. The BW¾ Method, which is roughly consistent with the chemical-specific information, but slightly *less* protective, was selected as the method for the toxicokinetics adjustment.

185 NRC (2006), pp. 98-99; pp. 442-446, Appendix D; NRC (2009), pp. 88-89.
186 NRC (2006), pp. 98, 442.
187 Zhang et al. (2014).

143. *Toxicodynamic Considerations:* It has long been recognized that rodents are less susceptible (i.e., more resistant) to certain toxic effects from fluoride ingestion than are humans.[188] Rats, for example, have been reported to require 10 to 25 times more fluoride than humans to develop dental fluorosis.[189] Differences in toxicokinetics contribute to rodents being less sensitive to fluorosis, but the differences appear larger than would be expected if they were due solely to kinetics. The fluorosis data support the existence of differential toxicodynamics between rodents and humans, but it is unclear if this difference would also apply to neurotoxicity, as this has not yet been the subject of study. Conversely, there are no data to suggest that humans are *more resistant* to fluoride neurotoxicity than animals. In the absence of data, EPA's default uncertainty factor of 3 was selected to account for interspecies differences in toxicodynamic differences.

3. LOAEL to NOAEL

144. When EPA uses a LOAEL from animal data as the Point of Departure, it applies an additional uncertainty factor of 10 to convert the LOAEL into an estimated NOAEL.[190] Consistent with EPA practice, the three LOAEL-based PODs were adjusted by a factor of 10.

4. Composite Uncertainty Factor

145. The "composite" uncertainty factor is the product of all uncertainty factors used in an analysis. The composite uncertainty factor applied here to the NOAEL-based PODs is **30**, which is the same value that EPA has been using in its draft risk evaluations under TSCA.[191] The

[188] Roholm (1937), pp. 265, 318; Lehman and Fitzhugh (1954), p. 33; Angmar-Mansson and Whitford (1982), p. 339.

[189] Roholm (1937), pp. 265, 318; Angmar-Mansson and Whitford (1982), p. 339.

[190] EPA (1998a), p. 59.

[191] In my expert report, I also calculated alternate RfDs where I applied additional uncertainty factors that EPA uses to account for subchronic animal exposures and deficiencies in the fluoride database (e.g. no data on formula-feeding during the neonatal period). For purposes of simplicity, I have not included those calculations in this declaration.

composite uncertainty factor applied here to the LOAEL-based PODs is **300**, which is on the low-end of the range of the composite uncertainty factors that EPA has used in its neurotoxicity risk assessments (see Table 3 above).

E. RfD Calculations from Animal Data

146. Table 5 summarizes the RfD calculations for each of the five Points of Departure (POD) listed above. The RfDs range from 0.0007 to 0.006 mg/kg/day for the LOAEL-based PODs, and 0.01 to 0.03 mg/kg/day for the NOAEL-based PODs. The least protective RfD that can be derived from the literature in a manner consistent with EPA practice is thus **0.03 mg/kg/day**.

Table 5. Calculation of the RfD from the selected Points of Departure (POD), based on the studies summarized in Table 2.

A Observation	B Intake rate	C POD_{HED}	D NOAEL	E $UF_H = 10$	F $UF_A = 3$	G RfD
LOAEL or NOAEL from Table 6 mg/L	Column A / 6 (rats) or 3.8 (mice) mg/kg/day	Column B × 0.24 (rats) or 0.14 (mice) mg/kg/day	Column C / 10 (LOAEL) or 1 (NOAEL) mg/kg/day	Column D / 10 mg/kg/day	Column E / 3 mg/kg/day	Column F mg/kg/day
5 mg/L, LOAEL (rats)	0.83	0.20	0.020	0.0020	0.00067	0.0007
23 mg/L, LOAEL (rats)	3.8	0.91	0.091	0.0091	0.0030	0.003
45 mg/L, LOAEL (rats)	7.5	1.8	0.18	0.018	0.0060	0.006
11 mg/L, NOAEL (mice)	2.9	0.41	0.41	0.041	0.014	0.01
20 mg/L, NOAEL (rats)	3.3	0.79	0.79	0.079	0.026	0.03

Column A: The observed LOAEL or NOAEL from Table 2.

Column B: The observed LOAEL or NOAEL converted from mg/L to an intake rate (dose) in mg/kg/day. For rats, the LOAEL or NOAEL is divided by 6; for mice, the NOAEL is divided by 3.8 (see explanation in text).

Column C: The intake rate for rats or mice converted to a human equivalent dose (HED) using the $BW^{3/4}$ method (see explanation in text). The HED = 24% of the intake rate for rats or 14% of the intake rate for mice.

Column D: NOAEL as already obtained (NOAEL / 1) or as estimated from a LOAEL (LOAEL / 10).

Column E: The estimated NOAEL after application of an intraspecies uncertainty factor (UF_H), where $UF_H = 10$. The NOAEL from Column D is divided by UF_H (i.e., NOAEL / 10).

Column F: The estimated NOAEL after application of an additional uncertainty factor for interspecies variability (UF_A), where $UF_A = 10$. The adjusted NOAEL from Column E is divided by UF_A (i.e., NOAEL / 3).

Column G: The value of the Reference Dose (RfD) obtained with only UF_H and UF_A. RfD = the NOAEL value in Column F, rounded to 1 significant digit.

VII. EXPOSURE ASSESSMENT

147. The only condition of use at issue in this case, as I understand it, is the addition of fluoridation chemicals to drinking water. Because of this, I limited the scope of my exposure assessment to exposures directly attributable to fluoridated water (0.7 mg/L). The purpose of my assessment was to enable a comparison of the doses people ingest from fluoridated water with the toxicity values (LOAELS and NOAELs) derived from the animal studies. I did not consider fluoride intake from dental products, pesticides, industrial pollution, occupational exposures, black tea, or other sources.

148. In my assessment, I considered fluoride exposures among both the general public as well as subsets of the population known to consume elevated amounts of water. For the source data, I relied primarily on the NRC's 2006 report which presented estimates of fluoride intake from water containing 0.7 mg/L fluoride. The NRC's estimates were based on an EPA analysis of community water intake data that were collected in a national survey by the US Department of Agriculture (USDA) in the 1990s.[192] The USDA survey was "designed to obtain a statistically representative sample of the United States population," and EPA stated that data from this survey "may be used in risk assessment analyses where exposures that occur through ingestion of water are of concern."[193]

149. Based on NRC's data, human exposure to fluoride from fluoridated water is estimated to range from an average of 0.011 mg/kg/day for adults to a "high" of 0.14 mg/kg/day for 95th percentile-exposed infants.

150. Following my initial report, a criticism was raised that I should have conducted a

[192] The USDA survey is called the "Continuing Survey of Food Intakes by Individuals," or CSFII for short.

[193] EPA (2000a), p. 5-5.

systematic review of all water intake data published subsequent to the NRC's report. In response to this criticism, I reviewed an updated and comprehensive review of water intake data that EPA published in 2019. The review was published as an update to EPA's *Exposure Factors Handbook* ("*Handbook*"), which is a document "intended for use by exposure and risk assessors both within and outside the U.S. EPA as a reference tool and primary source of exposure factor information."[194]

151. In its 2019 report, EPA presented the results of its "comprehensive review of the scientific literature [on water intake] through 2017" and provided EPA's determination as to "the most up-to-date and scientifically sound" data[195] to use for tap water consumption in the US.[196] The report thus provides the community water intake values[197] that EPA now recommends using for risk assessment for each age group in the population.[198]

152. The water intake data that EPA identifies in its 2019 report are consistent with EPA's older water intake data that I relied upon in my initial assessment. For example, whereas I selected 0.011 mg/kg/day as an average adult exposure, the EPA's updated data produce mean intakes by adults of 0.011-0.013 mg/kg/day (i.e., the same as or slightly higher than my estimate).[199] Further, whereas I selected 0.14 mg/kg/day as the 95th percentile exposure among

[194] EPA (2011a), p. 1-3.

[195] EPA (2019b), p. 1-5.

[196] EPA selected its own analysis of water intake data from NHANES's 2005-2010 surveys as the "key study" to use for all age groups in the general population and for pregnant and lactating women. For formula-fed babies, EPA selected an analysis by Kahn of the USDA's CSFII survey, which is the same survey that the NRC relied upon for its estimates in 2006.

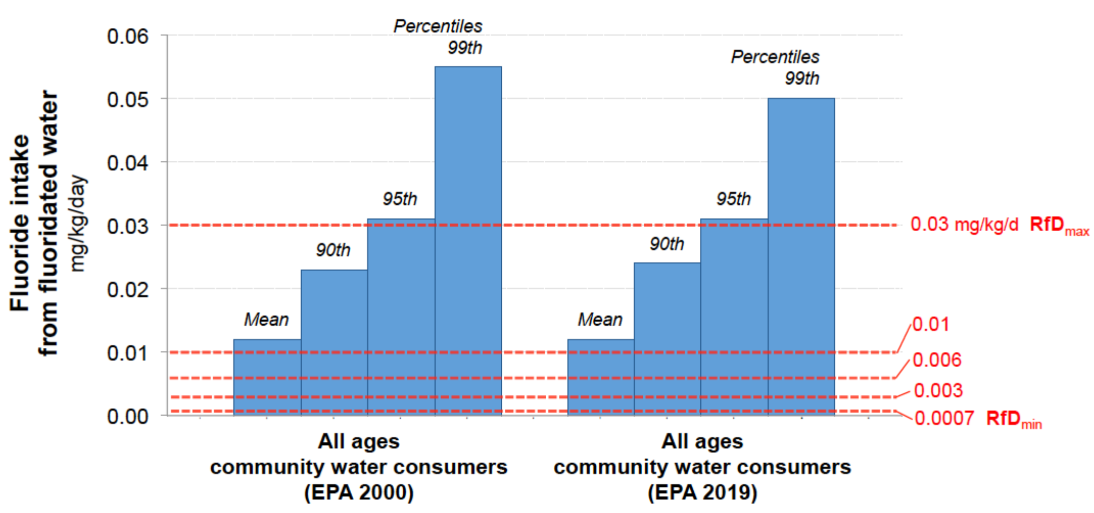
[197] EPA's report presents water intake in terms of milliliters of water consumed per kilogram of bodyweight per day (mL/kg/day). This permits a direct estimation of fluoride exposure from fluoridated water because the concentration of fluoride is known (0.7 micrograms per milliliter). By way of example, if a person drinks 100 milliliters of fluoridated water per kilogram of bodyweight, they will receive a dose of 70 *micro*grams of fluoride per kilogram, which is more commonly expressed as 0.07 *milli*grams per kilogram (i.e., 0.07 mg/kg/day).

[198] EPA (2019b), pp. 3-1, 3-4, 3-7, & 3-9.

[199] EPA (2019b), pp. 3-4.

bottle-fed infants, EPA's updated data produce 95th percentile values ranging from 0.13 mg/kg/day to 0.2 mg/kg/day (i.e., higher than my estimate).[200]

153. The similarity between the two EPA datasets can be seen in the following figure. The figure shows the fluoride exposure from water for *all* community water consumers for *all* age groups combined. The left side of the figure shows EPA's 2000 data (that NRC and I relied upon), while the right side of the figure shows EPA's 2019 data. To help put these exposures in context, the figure also shows the five reference doses from the animal neurotoxicity data (Table 5).



Intakes based on EPA 2000 Water Ingestion in USA, Appendix E, p. IV-3; and EPA 2019 Exposures Handbook, Chap. 3, Table 3-21.

154. As can be seen in the figure, the two datasets show that a *substantial* percentage of the population that consume fluoridated tap water exceed each of the 5 RfDs for neurotoxicity, including the least protective RfD.

155. One limitation with EPA's water intake data (from both 2000 and 2019) is that they do not include consumption of community water that is added to commercial beverages, such as soda and juice.[201] This underestimates actual exposure to fluoridated water, since commercial

[200] EPA (2019b), pp. 3-9.
[201] EPA (2000a), p. viii.

beverages have become a significant source of exposure to fluoridated water for many people.[202]

156. Another limitation with EPA's water intake data is that they are based on short-term surveys (i.e., surveys taken on two non-consecutive days), which creates a source of uncertainty when extrapolating to long-term exposures. This uncertainty is minimized, however, by the large numbers of people surveyed in the studies, and the use of non-consecutive days for the survey. While not perfect, EPA has recognized these data as the most scientifically sound data to use for risk assessment.

VIII. RISK CHARACTERIZATION

157. The risk characterization step of a risk assessment integrates the evidence of hazard, exposure, and dose-response in a clear and transparent manner, and provides a description of the risk. The *Guidelines* recognize multiple ways of describing risk, including (i) characterization of highly-exposed and/or susceptible individuals; (ii) estimation of the number of individuals exposed; (iii) comparing human exposures against the RfD; and (iv) "Margin of Exposure" analysis.[203]

A. Characterization of Highly Exposed and/or Highly Susceptible Populations

158. Susceptibility to a chemical may be "intrinsic" (biological, e.g., life stage) or "extrinsic" (acquired, e.g., lifestyle),[204] although many individuals may have *both* intrinsic *and* extrinsic susceptibility.

159. EPA has recognized that life stage is an important source of intrinsic susceptibility to neurotoxicants, and has identified the prenatal, infant, and elderly stages of life as "critical periods for exposure."[205] According to the EPA, "It is a well-established principle that there are

[202] E.g., Heilman et al. (1999); Kiritsy et al. (1996); Turner et al. (1998).
[203] EPA (1998a), pp. 63-66.
[204] EPA (2017).
[205] EPA (1998a), p. 65.

critical developmental periods for the disruption of functional competence, which include both the prenatal and postnatal periods to the time of sexual maturation, and the effect of a toxicant is likely to vary depending on the time and degree of exposure."[206] In light of this, a "population subgroup is susceptible if exposure occurs during a period of sensitivity."[207]

160. As described below, there are large, identifiable subsets of the population that are likely more susceptible to the neurotoxic effects of fluoride than the general population, including pregnant women and their fetuses, bottle-fed infants, the elderly, and individuals with renal impairment.

1. Pregnant Women and Their Fetuses

161. Multiple converging lines of evidence support the fetal period as a critical period of susceptibility to fluoride's neurotoxic effects. First, it is well established that fluoride crosses the placenta and reaches the fetus.[208] Second, due to the absence of an effective blood brain barrier,[209] the fluoride that reaches the fetus also reaches the brain—a fact that has been confirmed by both animal and human studies.[210] Third, fluoride has the capacity to lower thyroid function, particularly among individuals with low iodine intakes, and EPA has recognized that alterations to thyroid function (e.g., reductions in thyroid hormone concentrations) during pregnancy can cause cognitive disorders and other neurological harm to the child.[211] Fourth, most studies of prenatal fluoride exposures in animals have documented neuroanatomical, neurochemical, and/or cognitive problems. Fifth, all prospective cohort studies that included individual measurements of

[206] EPA (1998a), p. 46.
[207] EPA (2008a), p. 42.
[208] NRC (2006), p. 193.
[209] EPA (2009b), p. 58.
[210] E.g., McPherson et al. (2018); Mullenix et al. (1995); Du et al. (1992).
[211] EPA (1998a), p. 50; EPA (2008a), p. 40; EPA (2008b), p. 54; EPA (2013b), cover letter, p. 2. See also Rodier (1995); Zoeller and Rovet (2004); Patel et al. (2011); Suárez-Rodríguez et al. (2012); Modesto et al. (2015); Bellinger (2018).

prenatal fluoride exposure have found significant adverse associations with neurocognitive harm, including IQ loss and inattention.[212]

162. The number of pregnant women exposed to fluoridated water each year is large. The CDC estimates that there are approximately 4 million children born in the U.S. each year, and therefore about 4 million pregnancies.[213] With approximately two-thirds of the U.S. population living in communities where fluoridation chemicals are added to water, about 2.5 million pregnancies can be expected to occur each year in fluoridated areas.

163. Of paramount concern are pregnant women who have an iodine deficiency. The CDC considers the average iodine status (median urinary iodine concentration) of women of childbearing age (12-19 years and 20-39 years) in the U.S. to be in the "adequate intake" range, but the 10th percentiles by ethnicity and for the total population are in the "insufficient intake" range, indicating that more than 10% of women of childbearing age in the U.S. are deficient in iodine.[214] Caldwell et al. report that 35% of pregnant women and 38% of nonpregnant women in the U.S. have urinary iodine concentrations below the level considered adequate.[215] In addition, the CDC notes that even higher intakes of iodine are required for pregnant and lactating women; thus an even greater percentage of American women are likely to be deficient in iodine with respect to the demands of pregnancy and lactation.[216] Pearce suggests that iodine deficiency in the U.S. may be becoming more prevalent, especially among pregnant women.[217]

164. While the effects of fluoride exposure among pregnant women with iodine

[212] Bashash et al. (2017; 2018); Green et al. (2019); Valdez-Jiménez et al. (2017).
[213] Centers for Disease Control. Births and Natality. Available at: http://www.cdc.gov/nchs/fastats/births.htm
[214] CDC (2008), Chapter 4a, pp. 90-100.
[215] Caldwell et al. (2011).
[216] CDC (2008), Chapter 4a, pp. 90-100.
[217] Pearce (2015).

deficiency have not yet been specifically studied, there is a clear basis for concern. The NRC reported that high fluoride intake appears to exacerbate the effects of low iodine intake on thyroid function in both animals and humans.[218] Consistent with this, Malin et al. found that an increase in urinary fluoride was associated with an increase in thyroid stimulating hormone (TSH)—an indicator of decreased thyroid function—among iodine-deficient adults in Canada.[219] A decrease in thyroid function during pregnancy, even in the absence of clinical symptoms in the mother, is associated with reduced IQ and other neurological effects in the offspring.[220]

2. Bottle-Fed Infants

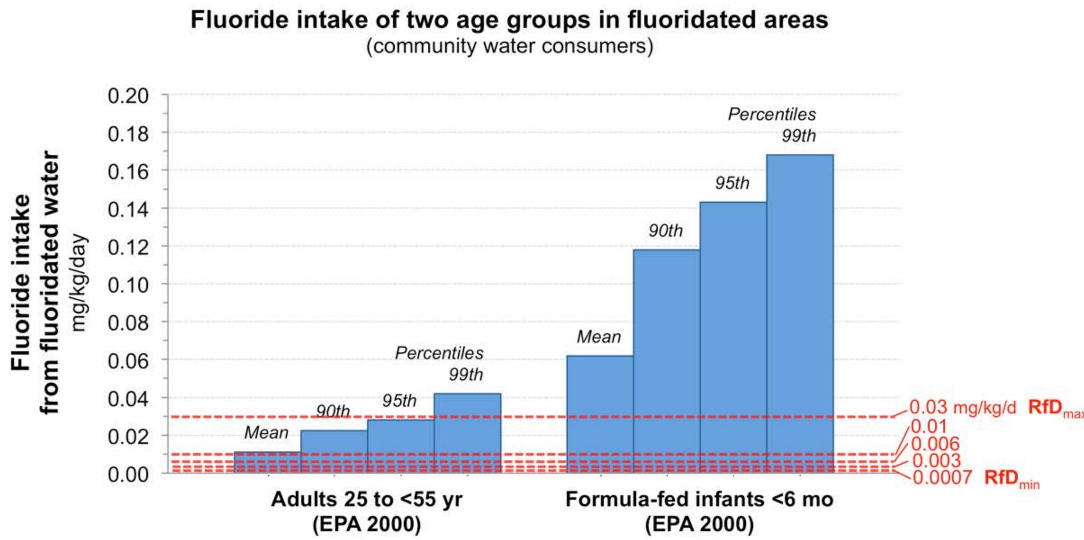
165. A bottle-fed infant has a combination of *both* intrinsic *and* extrinsic susceptibility to fluoridated water.

166. *Intrinsic Susceptibility*: The blood brain barrier does not finish developing until 6 months of age,[221] and, as such, the fluoride ingested during early infancy will likely reach the brain more readily than during the later childhood and adult years. The brain is also undergoing "rapid development" during infancy, with the growth rate of the brain peaking at 4 months of age.[222] The EPA has thus described the neonatal stage of life as "a critical window of development."[223]

167. *Extrinsic Susceptibility*: Infants have the highest intake of fluid per unit body weight of any age group among humans, given their mostly liquid diet at that age. This can be seen in the following figure, which uses EPA's 2000 water intake data to compare the community water

[218] NRC (2006), pp. 227, 234, 262.
[219] Malin et al. (2018).
[220] For example, see Haddow et al. (1999); Pop et al. (1999; 2003); Morreale de Escobar et al. (2000; 2004); Klein et al. (2001); Vermiglio et al. (2004); LaFranci et al. (2005); Kooistra et al. (2006); Roman (2007); Zoeller and Rovet (2004); Patel et al. (2011); Suárez-Rodríguez et al. (2012); Modesto et al. (2015); Moleti et al. (2016).
[221] EPA (2009b), p. 58.
[222] EPA (2013a), p. 5-4; EPA (2008a), p. 42.
[223] EPA (2008a), p. 42.

intake of adults (on the left) with the community water intake of bottle-fed infants (on the right).



Intakes based on EPA 2000 Water Intake Estimates in US, p. IV-3.

168. According to the CDC, 75% of infants born in 2015 were formula-fed at least partially during their first six months, including 17% of infants who were *exclusively* formula-fed.[224] Data vary by ethnicity, with Hispanics, whites and Asians having breastfeeding rates similar to or greater than the national averages and African Americans having substantially lower rates. Breastfeeding rates tend to be highest for higher family income and maternal education levels.

169. Breastfeeding rates in the U.S. have increased substantially in recent years from a low point in the early 1970s.[225] While increased breastfeeding rates are to be encouraged for a number of reasons, it is important to remember that for many infants in the U.S., breastfeeding is not an option; these include cases of infant adoption or fostering, as well as cases of death or illness of the mother.

[224] CDC (2018; n.d.).
[225] DHEW (1979), pp. 2-6, especially Tables A and B.

170. Most commercial infant formula, historically and currently, has been in powder form, for which the cost is approximately half that of ready-to-feed formula, per unit volume of formula as fed.[226] Based on national data collected during 2005-2007, the CDC reported that approximately 83-93% of babies are fed formula prepared from powder from cans.[227] For approximately 70-78% of infants in the same national survey, formula is reconstituted with tap water at least some of the time.[228]

171. Based on the available information, it can reasonably be assumed that the majority of formula-fed infants in the U.S. are fed powdered formula reconstituted with water, often or usually tap water. Especially for low-income homes (where breastfeeding is less likely), it is reasonable to assume that many or most infants are fed formula prepared from powder using tap water, which in much of the country is fluoridated. In addition, for approximately 20% of infants, tap water is boiled before it is used to prepare formula;[229] if this tap water is fluoridated, the resulting fluoride concentration in the formula will be higher than if the water had not been boiled.[230]

172. Fomon et al. estimated that infants consuming powdered formula prepared with fluoridated water (1 mg/L) will ingest between 0.116 and 0.164 mg/kg/day.[231] If Fomon's estimate is adjusted to account for the lower concentration of fluoride now added to water (0.7 mg/L), the result is a daily intake of 0.08 to 0.115 mg/kg/day, which is 80 to 115 times higher than the amount that Fomon et al. estimated for breast-fed infants (0.001 mg/kg/day).[232] By Fomon's estimates,

[226] O'Connor (2009).
[227] CDC (2017), Table 3.16.
[228] CDC (2017), Table 3.97.
[229] CDC (2017), Table 3.98.
[230] For example, see Juárez-López et al. (2011).
[231] Fomon et al. (2000), Table 2.
[232] Fomon et al. (2000), Table 2.

essentially all formula-fed infants will exceed the RfDs for neurotoxicity if their formula is prepared with fluoridated tap water.

173. Fomon's estimates agree well with recent data from Harriehausen et al., who surveyed 114 parents in Houston to determine brand and type of formula, total volume of formula consumed over 24 hours, and infant weight.[233] Most of the parents in the study (corresponding to 92.1% of the infants) reported using powdered formula, which is consistent with the literature described above.[234] Harriehausen et al. estimated that over 50% of infants fed formula made with fluoridated water will exceed 0.1 mg/kg/day during the first 4 months of life (Table 6).

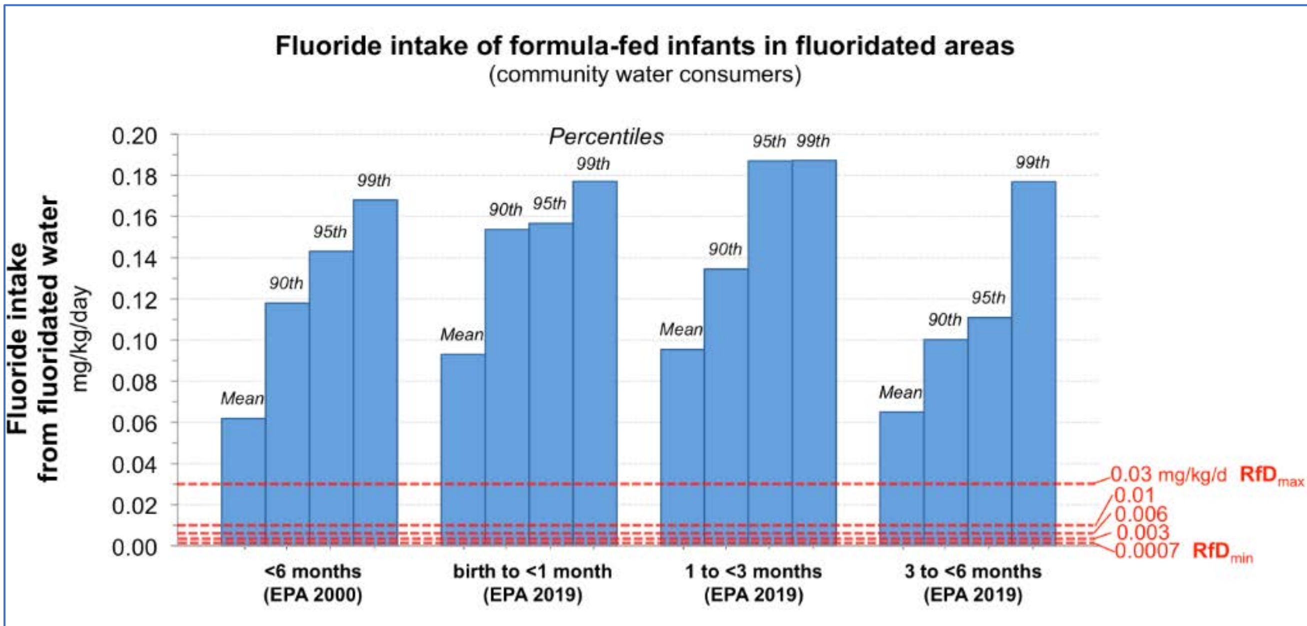
Table 6. Estimated fluoride ingestion from infant formula, assuming fluoridated water at 0.7 mg/L.[a]

	Age				
Category	2 months	4 months	6 months	9 months	12 months
Number of infants	32	23	27	21	11
Predicted fluoride intake					
Mean (mg/kg/day)	0.110	0.112	0.090	0.066	0.053
Variance	0.0033	0.0016	0.0018	0.0012	0.0009
Standard deviation[b]	0.057	0.040	0.042	0.035	0.03
Distribution of fluoride intake					
> 0.1 mg/kg/day (%)	59.4	56.5	33.3	14.3	9.1
< 0.1 mg/kg/day (%)	40.6	43.5	66.7	85.7	90.9

[a] Data from Harriehausen et al. (2019), Table 3.
[b] Calculated from the variance reported by Harriehausen et al. (2019), Table 3.

[233] Harriehausen et al. (2019).
[234] Harriehausen et al. (2019), Table 2.

174. The estimates from both Harriehausen and Fomon are consistent with EPA's most up-to-date and scientifically sound estimates for formula-fed infants.[235] According to EPA's updated estimates, mean fluoride exposures during the first 6 months of life for infants receiving formula reconstituted with fluoridated water are **0.07 to 0.10 mg/kg/day** (age-dependent), with 95th percentile exposures of **0.13 to 0.2 mg/kg/day** for the first 3 months and **0.13 mg/kg/day** for the next three months.[236] These intakes are very high, and far exceed even the least protective RfD, as shown in the following figure.



Intakes based on EPA 2000 Water Ingestion in USA, Appendix E, p. IV-3; and EPA 2019 Exposures Handbook, Chap. 3, Table 3-21.

175. In its *Guidelines*, EPA considers the potential for postnatal toxicant exposures to interact with breastmilk composition.[237] EPA was referring to animal studies, but the principle would apply to humans as well: Replacement of the mother's milk with a substitute that contains a toxic agent would be an extremely important source of postnatal exposure for infants and children

[235] EPA (2019b), p. 3-9.
[236] EPA (2019b), p. 3-9.
[237] EPA (1998a), p. 46.

to that toxic agent. Few, if any, animal studies reproduce the effect of formula-feeding of human infants, in terms of a water-based formula containing fluoride being substituted for the mother's milk; thus this very important developmental period is routinely missed in most developmental studies on fluoride.

176. Consistent with the high fluoride intakes produced by formula feeding, studies have found that bottle-fed babies have higher rates of dental fluorosis (a disorder of enamel caused by excess fluoride intake) in their permanent teeth.[238] Studies have also documented an increased prevalence and severity of dental fluorosis in the African American community, which is consistent with the high rate of formula feeding in this population.[239]

177. While fluoride exposure during infancy is known to produce abnormal physiological changes in the body (e.g., dental fluorosis), there has been a paucity of research on the neurodevelopmental effects of this exposure. In the developmental studies on fluoride neurotoxicity to date, the pups have been *breastfed*. Consequently, the existing animal data do not reflect the neurotoxic effects that may occur during the neonatal period.

178. Studies in humans have found lower IQ scores among formula-fed babies versus breastfed babies,[240] but up until this year,[241] no study had investigated the role that fluoridated water may have in this association. Specific differences in brain activation and regional volumes of gray matter have been reported among formula-fed children, indicating developmental changes in children in comparison with breastfed children.[242] Such effects (and other adverse effects of

[238] Hong et al. (2006a; 2006b); Forsman (1977); Walton and Messer (1981); Fomon and Ekstrand (1999); Fomon et al. (2000).

[239] EPA (2010), pp. 33-34; Exhibit 34 to Casey Hannan Deposition; CDC (2005), Table 23.

[240] For example, see Fomon (2001); Wolf 2003; Belfort et al. (2013); Horta et al. (2015; 2018); Victora et al. (2015; 2016); Kanazawa (2015); Boutwell et al. (2018). Many studies have controlled for possible confounders such as maternal IQ, maternal education, and family income.

[241] Till et al. (2020).

[242] Ou et al. (2016).

formula-feeding compared with breastfeeding, especially compared with exclusive breastfeeding for at least the first several months) could, in principle, be due to loss of the enhanced mother-child bonding associated with breast-feeding,[243] to deficiency of an essential nutrient (e.g., long-chain saturated fatty acids) in the formula,[244] to the presence of a toxic contaminant in the water used to prepare the infant formula,[245] or to some combination of these factors.

179. Given the *a priori* basis for concern that fluoridated water may adversely affect the neurological system of bottle-fed infants, the recent findings from Till et al. must be taken very seriously.[246] Using a prospective cohort study design, Till et al. found that fluoridated water consumption during infancy is associated with a large and significant reduction in non-verbal IQ at age 4 (i.e., a loss of 9.3 non-verbal IQ points for each 0.5 mg/L increase in exposure). Although the study did not find a statistically significant association with Full-Scale IQ after excluding several outliers, this could be a result of imprecision in the exposure estimates, or might reflect differential impacts of pre- and post-natal exposure.

180. As noted earlier, CDC data indicate that 17% of babies are *exclusively* fed formula for their first six months of life (i.e., never breast-fed).[247] Assuming 2.5 million live births per year in fluoridated areas, approximately 1.9 million infants living in fluoridated areas will be formula-fed for at least part of the time during their first six months, including 425,000 infants who are *exclusively* formula-fed.[248] Approximately 70-78% of these infants will have their formula made

[243] Horta et al. (2018).
[244] Horta et al. (2018).
[245] Goyer (1995).
[246] Till et al. (2020).
[247] CDC (2018; n.d.).
[248] CDC (2018; n.d.).

with fluoridated tap water (some of which will be boiled and have higher concentrations), at least part of the time.[249]

3. Elderly

181. The elderly have also been identified by the EPA as an at-risk group for neurotoxicity.[250] According to the EPA, the elderly are "at particular risk because of the limited ability of the nervous system to regenerate or compensate to neurotoxic insult."[251]

182. The NRC has described the possible relationship of fluoride exposure, especially exposure to aluminum fluoride complexes, to the development of Alzheimer's disease.[252] The NRC based its concern, in part, on studies reporting pathological lesions in the brain of fluoride-treated rodents that parallel the changes in humans with dementia.[253] A recent study by Cao et al. found that exposure to fluoride for 3 months among mice genetically prone to degenerative brain changes, produced more severe, and earlier development of, neuropathological lesions than in controls, including lesions associated with Alzheimer's.[254] Goschorska et al. have recently postulated that fluoride plays a likely role in the initiation and progression of Alzheimer's disease, based largely on the neuroanatomical and neurochemical changes seen in the brains of fluoride-treated animals.[255]

183. While epidemiological data on fluoride and cognition in the elderly remain relatively sparse, Li et al. reported a very high rate of cognitive impairment (81.1%) in an endemic fluorosis area.[256] Li did not find a linear relationship between urinary fluoride and the severity of

[249] CDC (2017), Table 3.97; Juárez-López et al. (2011).
[250] EPA (1998a), p. 65; see also NRC (2006), p. 351.
[251] EPA (1998a), p. 65.
[252] NRC (2006), pp. 210-212.
[253] NRC (2006), pp. 222.
[254] Cao et al. (2019).
[255] Goschorska et al. (2018).
[256] Li et al. (2016), p. 59.

cognitive impairment within the endemic fluorosis area; however urinary fluoride levels among those with any form of cognitive impairment were significantly higher than those with normal cognition.[257] Russ et al. described a longitudinal study involving nearly all people born in Scotland in 1921, who were passively followed for diagnoses of dementia after 2004.[258] Residential locations after age 60 (or at death or at time of diagnosis of dementia) were used to estimate exposure to aluminum and fluoride (separately) in drinking water. The authors found that even relatively low levels of aluminum and fluoride were associated with an increased prevalence of dementia and suggested further research.[259]

184. While more research is needed to clarify fluoride's effects in the elderly population, there are a multitude of factors which support an increased vulnerability to fluoride's neurological effects among the elderly. Studies have found that water fluoridation significantly increases the level of fluoride in bone, and these levels increase with age.[260] In the post-menopausal and elderly years, the fluoride that is taken into bone can be released back into the blood stream as bones begin to break down, leading to increased levels of fluoride in the blood.[261] Compounding this, renal function declines with age, and because of this the elderly kidney can be expected to be less efficient in clearing fluoride from the bloodstream. The net result is that more fluoride will be circulating in the bloodstream, and due to age-related increases in the permeability of the blood-brain barrier, will reach the brain more readily.[262]

185. Although the impact of fluoride on the elderly brain has not received as much

[257] Li et al. (2016), Figure 2, Table 3.
[258] Russ et al. (2019).
[259] Russ et al. (2019).
[260] Alhava et al. (1980); Arnala et al. (1985); Eble et al. (1992); Chachra et al. (2010).
[261] Itai et al. (2010).
[262] Increased permeability of the blood-brain barrier is associated with ordinary aging, as well as with diseases such as Alzheimer's and Parkinson's, both of which are common among elderly people. For example, see Mooradian (1994); Zeevi et al. (2010); Rosenberg (2014); and Pan and Nicolazzo (2018).

scholarly attention as the impact on the developing brain, this population is likely at higher risk of toxicity than healthy adults, particularly among those with elevated accumulation of fluoride in the bone following long-term residence in a fluoridated area.

Renal Impairment

186. It is well recognized that people with renal impairment (kidney disease) are less able to excrete fluoride, resulting in higher concentrations of fluoride in the body and greater susceptibility to adverse health effects from fluoride exposure.[263] The World Health Organization states that it "is known that persons suffering from certain forms of renal impairment have a lower margin of safety for the effects of fluoride than the average person."[264] In addition, a number of papers report an association between renal impairment and reduced IQ or other cognitive impairment,[265] which is consistent with higher fluoride retention (and often higher water intake and consequent higher fluoride intake). The role of fluoride in these IQ deficits has not yet been the subject of epidemiological study.

4. Other Predisposing Factors

187. There are a number of other factors that are known, or reasonably anticipated, to increase susceptibility to the chronic toxic effects of fluoride exposure, including neurotoxicity. These factors include:

188. *Diseases that Increase Water Intake*: The NRC identified population subgroups whose water intake "is likely to be substantially above the national average for the corresponding sex and age group" as susceptible subpopulations with respect to fluoride exposure.[266] Health

[263] For example, see Marumo and Iwanami (2001); NRC (2006), pp. 30, 292, 351; Ibarra-Santana (2007); Schiffl (2008).
[264] WHO (2004), p. 6.
[265] For example, see Madero et al. (2008); Mendley et al. (2015); Chen et al. (2018b).
[266] NRC (2006), p. 30.

conditions that affect water intake include "diabetes mellitus, especially if untreated or poorly controlled; disorders of water and sodium metabolism, such as diabetes insipidus; [and] renal problems resulting in reduced clearance of fluoride."[267] According to the NRC, adults with diabetes mellitus can ingest 0.05 mg/kg/day from fluoridated water alone, while children with diabetes mellitus can have fluoride intakes as high as 0.07 mg/kg/day.[268] For children and adults with nephrogenic diabetes insipidus, NRC estimated waterborne fluoride intakes of 0.11 mg/kg/day.[269,270] Each of these intakes exceeds the least protective RfD.

189. *Nutrient Deficiencies*: Nutritional deficiencies can contribute to increased susceptibility to fluoride toxicity.[271] Calcium deficiency and iodine deficiency are expected to be particularly important in terms of vulnerability to neurotoxic effects of fluoride, but deficiencies of magnesium, vitamin C, protein, and other nutrients have also been associated with increased susceptibility to the effects of fluoride exposure.

190. *Genetic Susceptibilities*: A number of studies have shown associations between specific genetic arrangements and a greater susceptibility to the chronic effects of fluoride exposure,[272] including dental fluorosis and alterations to reproductive hormones.[273] While a complete picture of the relationship between genes, gene regulation, and adverse effects of fluoride exposure remains to be developed, it is already quite clear that some people or groups of people

[267] NRC (2006), p. 30.
[268] NRC (2006), p. 35, Table 2-4.
[269] NRC (2006), p. 35, Table 2-4.
[270] Consistent with this, case reports have documented moderate to severe dental fluorosis among children with diabetes insipidus who drank water with 0.5 to 1 mg/L NRC (2006), p. 33.
[271] See for example, NRC (2006). p. 265; Pandit et al. (1940); Marier (1977).
[272] Reviewed by Pramanik and Saha (2017). See also Lavryashina et al. (2003); Tu et al. (2011); Liu et al. (2006); Huang et al. (2008); Ba et al. (2011); Zhao et al. (2015); Zhou et al. (2016); Zhang et al. (2013b); Pei et al. (2017); Jiang et al. (2015); Zhang et al. (2015b); Cui et al. (2018); Küchler et al. (2018); Bhagavatula Naga (2009).
[273] Zhao et al. (2015); Zhou et al. (2016); Ma et al. (2017); An et al. (2019).

are inherently more vulnerable than others to adverse effects of fluoride exposure and require a greater level of protection from fluoride exposures.[274] The implications to neurotoxicity have not yet been extensively studied, but two recent studies from China, including one with extensive control for covariates, suggest that certain genotypes may significantly magnify fluoride's impact on IQ in some individuals.[275] A third, smaller study reported a contrary result.[276] More research is needed to clarify this issue, but in light of the broader literature on genetic susceptibility to chronic fluoride toxicity, it is reasonable to suspect that genetics plays a role in rendering some individuals more vulnerable to fluoride's neurological effects.

B. Margin of Exposure (MOE)

1. Introduction to the MOE Approach and Its Similarity to the RfD Approach

191. Under the *Guidelines*, neurotoxic risk can be described either through a comparison of the human exposures to the RfD, or by calculating the "Margin of Exposure" (MOE).[277] Although the two approaches use slightly different frameworks, they produce the same results. If comparison of human exposure with the RfD shows a risk, a risk will be shown by MOE as well, and vice versa.

192. RfD and MOE analyses produce the same results because they use the same Point of Departure (i.e., NOAEL, LOAEL, or BMDL) for the toxicity value, the same data for human exposure, and the same composite uncertainty factor to assess whether human exposure poses a risk. Where the two methods differ is in how they put these three pieces together and the terminology they use, as will now be discussed.

193. In an RfD analysis, human exposure is compared against the Reference Dose. As

[274] Wu et al. (2015); Yang et al. (2016); Pei et al. (2017); Li et al. (2017); Yang et al. (2018b).
[275] Cui et al. (2018); Zhang et al. (2015b).
[276] Pang et al. (2018).
[277] EPA (1998a), pp. 65-66; Federal Register (1998), pp. 26949-26950.

discussed earlier, the Reference Dose is the Point of Departure (i.e., NOAEL, LOAEL, or BMDL) divided by the composite uncertainty factor. In an MOE analysis, by contrast, human exposure is *compared directly against the Point of Departure.* If the ratio (i.e., Actual MOE) between the Point of Departure and human exposure is less than the composite uncertainty factor (i.e., Acceptable MOE), an unacceptable risk is presumed to exist.[278,279] In short, the composite uncertainty factor is the standard for judging whether human exposure is unacceptably close to the toxicity value under both frameworks.

2. MOE Analysis

194. As part of the risk assessment, I conducted an MOE analysis to characterize risk because this is EPA's preferred method to characterize non-cancer risk under TSCA, as evident by its risk evaluations under both Section 5 (new chemicals)[280] and Section 6 (existing chemicals).[281]

195. *Points of Departure*: The same five Points of Departure (converted into Human Equivalent Doses) that were used for the derivation of the Reference Doses, as discussed above (see Table 5), were used for the MOE analysis.

196. *Acceptable MOEs (Benchmark MOEs)*: The same composite uncertainty factors that were used for the RfD derivation were selected as the Acceptable MOEs: 30 for the NOAEL-based PODs, and 300 for the LOAEL-based PODs. In EPA's draft risk evaluations under Section 6 of TSCA, EPA has used composite uncertainty factors of 30 for NOAEL-based PODs, and has

[278] EPA (2016), p. 61; EPA (2012), p. 13-8.
[279] EPA sometimes refers to the risk as a "risk of concern." EPA (2007), p. 13; EPA (2000c), p. C-12.
[280] See for example EPA (2018b; 2018c; 2018d; 2018e; 2018f; 2018g; 2019a).
[281] In its draft risk evaluations under Section 6 thus far, EPA has used MOE to characterize non-cancer risk. (EPA 2019c; 2019d; 2020). In its Final Rule for Risk Evaluation under Section 6, however, EPA described the MOE method as "just one of several approaches to risk characterization" that may be used under TSCA (Federal Register 2017, p. 33735).

characterized this as a relatively small uncertainty factor that "indicates greater certainty in the data (because fewer of the default UFs relevant to a given POD . . . were applied)."[282] EPA has contrasted this with a composite uncertainty factor of 1,000, which "would indicate more uncertainty in risk estimation and extrapolation."[283]

197. *Human Exposure*: At the time I conducted this analysis, EPA had not yet released any of its Section 6 draft risk evaluations. I relied, therefore, on EPA's risk evaluations under Section 5 for guidance on the human exposure assessment. In the Section 5 risk evaluations, EPA considers the highest-exposed group in the population. When dealing with chemicals that may be present in drinking water, therefore, EPA's MOE analyses separately consider the exposures of *infants*.[284]

198. Based on the guidance from the Section 5 risk evaluations, I relied on the NRC's 2006 data to calculate a range of exposures representing the general adult population along with highly exposed population subgroups, including bottle-fed infants and individuals with high water intakes (for example, due to medical conditions or to physical exertion).[285]

199. In EPA's draft risk evaluations under Section 6, EPA has used the 95th percentile exposure to represent highly exposed individuals.[286] This is the same percentile exposure I used

[282] EPA (2019d), p. 301.

[283] EPA (2019d), p. 301.

[284] See for example EPA (2018b; 2018c; 2018d; 2018e; 2018f; 2018g; 2019a).

[285] For the general adult population, I combined NRC's estimates for adult consumers of municipal water, ages 20-24 and 25-54 years (0.011 mg/kg/day). As an example of elderly adults (ages 65+), I included the 90th percentile of adult consumers of municipal water (0.022 mg/kg/day). To account for individuals with high water intakes, I used the NRC's waterborne fluoride intake estimates (at 0.7 mg/L) for adult athletes and physical laborers (0.05 mg/kg/day), children with diabetes mellitus (0.07 mg/kg/day), and individuals with nephrogenic diabetes insipidus (0.1 mg/kg/day). For bottle-fed infants**,** I estimated a typical (0.1 mg/kg/day) and high (0.14 mg/kg/day) exposure based on the data from Fomon et al. (2000), Harriehausen et al. (2019), and NRC (2006). None of these exposure estimates, even those labeled "high," is an upper bound or maximum exposure.

[286] E.g., EPA (2019d), pp. 266, 300; EPA (2020), p. 108.

for high exposures among bottle-fed infants, and a higher percentile exposure than I used for the elderly (90th percentile).

200. Table 7 and the two figures below show the results of the MOE analyses. The first figure shows the results using the three LOAEL-Based PODs, while the second figure shows the results using the NOAEL-based PODs. As can be seen, the Actual MOEs are below the Acceptable MOEs for each group using every POD (including the least protective), with the exception of *average* adults and 90th percentile elderly when using the NOAEL-based PODs. If EPA's recommended 95th percentile exposure data (0.031 mg/kg/day) is used as the exposure for adults, risks are present even when using the least protective PODs.

201. The margins between the neurotoxicity levels in animals and the exposure levels in humans are far smaller than what EPA considers "acceptable." In fact, the Actual MOEs are so small that unacceptable risks would still be indicated for infants for each POD if the doses from animal studies had no adjustment to convert to the Human Equivalent Doses (HEDs) (i.e., no allometric scaling). Under EPA's framework for characterizing risk, therefore, it is apparent that fluoridation chemicals in drinking water present an unacceptable risk of neurotoxicity.[287]

[287] EPA (2016), p. 61.

Table 7. Calculated Margins of Exposure (MOEs)[a] for selected subgroups of the human population for the NOAELs and LOAELs for fluoride in Table 5.

Observation[b]	Intake rate[c]	Human Equivalent Dose (HED)[d]	Estimated human exposures[e]					
LOAEL or NOAEL	LOAEL or NOAEL	LOAEL or NOAEL	Adults (average)	Elderly adults (90th percentile)	Athletes and laborers (high)	DM patients (high)	Bottle-fed infants (typical) NDI patients (high)	Bottle-fed infants (high)
mg/L	mg/kg/day	mg/kg/day	0.011 mg/kg/day	0.022 mg/kg/day	0.05 mg/kg/day	0.07 mg/kg/day	0.1 mg/kg/day	0.14 mg/kg/day
5 mg/L, LOAEL (rats)	0.83	0.20	18	9.1	4.0	2.9	2.0	1.4
23 mg/L, LOAEL (rats)	3.8	0.91	83	41	18	13	9.1	6.5
45 mg/L, LOAEL (rats)	7.5	1.8	163	82	36	26	18	13
11 mg/L, NOAEL (mice)	2.9	0.41	37	19	8.2	5.9	4.1	2.9
20 mg/L, NOAEL (rats)	3.3	0.79	72	36	16	11	7.9	5.6

[a] A Margin of Exposure (MOE) is equal to the LOAEL or NOAEL (mg/kg/day) divided by an estimated human exposure (mg/kg/day). Usually, the benchmark MOE = 1000 for assessments based on a LOAEL and 100 for assessments based on an NOAEL. Since allometric scaling between animals and humans has been used to obtain the Human Equivalent Dose, the benchmark MOE is 300 for LOAELs and 30 for NOAELs. An MOE less than the benchmark MOE indicates an "unacceptable risk."

[b] These LOAEL and NOAEL values (mg/L) for fluoride are summarized in Table 2.

[c] The intake rates (mg/kg/day) in this column correspond to the LOAELs and NOAELs in the first column (mg/L), converted to intake rates (mg/kg/day) as summarized in Table 5. For rats, the intake rate equals the LOAEL or NOAEL divided by 6. For mice, the intake rate equals the NOAEL divided by 3.8.

[d] The Human Equivalent Dose (HED) is calculated from the intake rate for rats or mice as summarized in Table 5. The HED = the intake rate for rats × 0.24 or the intake rate for mice × 0.14.

[e] The estimated human exposures are for fluoride exposures from drinking water alone, assuming a fluoride concentration of 0.7 mg/L in the drinking water. Sources are as follows:
Adults (average), NRC (2006), p. 430, Table B-11, average consumers ages 20-54;
Elderly adults (90th percentile), NRC (2006), p. 431, Table B-12, 90th percentile consumers ages 65+;
Athletes and laborers (high), NRC (2006), p. 35, Table 2-4, high consumers (but not upper bound);
DM patients (high), NRC (2006), p. 35, Table 2-4, patients with diabetes mellitus, high consumers (but not upper bound);
Bottle-fed infants (typical), based on Fomon et al. (2000) and Harriehausen et al. (2019);
NDI patients (high), NRC (2006), p. 35, Table 2-4, patients with nephrogenic diabetes insipidus, high consumers (but not upper bound); and
Bottle-fed infants (high), NRC (2006), p. 432, Table B-13, infants < 0.5 years old, 95th percentile consumers (but not upper bound).

Table 8. Calculated Margins of Exposure (MOEs)[a] for selected human population subgroups for the NOAELs and LOAELs for fluoride in Section 4.

Observation[b]	Intake rate[c]	Human Equivalent Dose (HED)[d]	Actual MOEs Estimated human exposures[e]					
LOAELs from animal studies								
LOAEL or NOAEL	LOAEL or NOAEL	LOAEL or NOAEL	Adults (average)	Elderly adults (90th percentile)	Athletes and laborers (high)	DM patients (high)	Bottle-fed infants (typical) NDI patients (high)	Bottle-fed infants (high)
mg/L	mg/kg/day	mg/kg/day	0.011 mg/kg/day	0.022 mg/kg/day	0.05 mg/kg/day	0.07 mg/kg/day	0.1 mg/kg/day	0.14 mg/kg/day
5 mg/L, LOAEL (rats) ◇	0.83	0.20	18	9.1	4.0	2.9	2.0	1.4
23 mg/L, LOAEL (rats) □	3.8	0.91	83	41	18	13	9.1	6.5
45 mg/L, LOAEL (rats) △	7.5	1.8	163	82	36	26	18	13
11 mg/L, NOAEL (mice)	2.9	0.41	37	19	8.2	5.9	4.1	2.9
20 mg/L, NOAEL (rats)	3.3	0.79	72	36	16	11	7.9	5.6

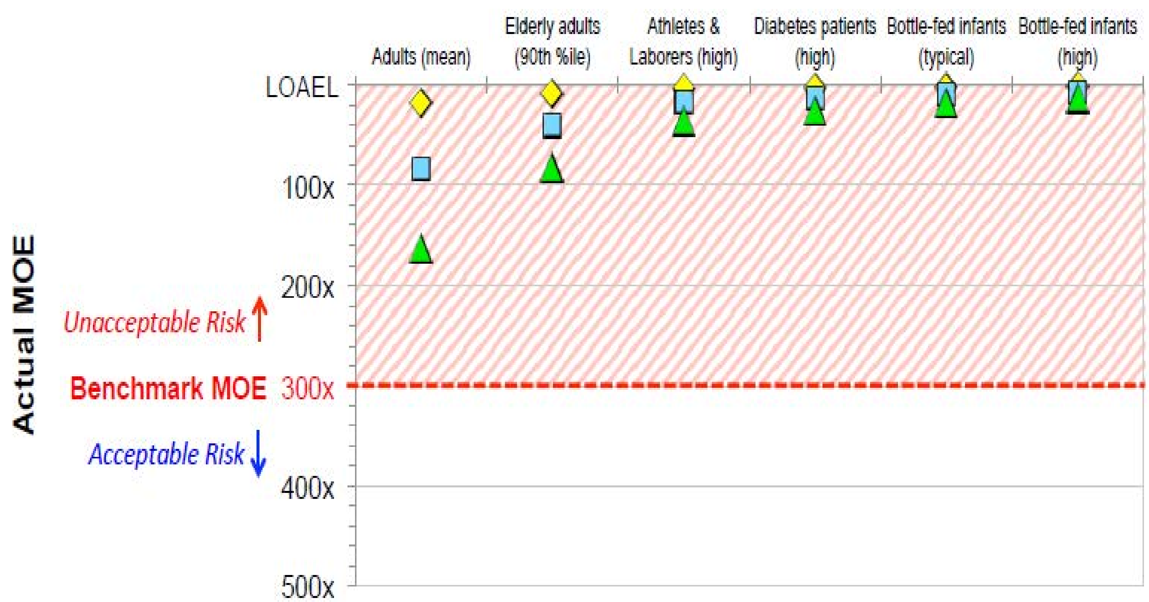


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Table 8. Calculated Margins of Exposure (MOEs)[a] for selected human population subgroups for the NOAELs and LOAELs for fluoride in Section 4.

Observation[b]	Intake rate[c]	Human Equivalent Dose (HED)[d]	Actual MOEs Estimated human exposures[e]					
NOAELs from animal studies LOAEL or NOAEL	LOAEL or NOAEL	LOAEL or NOAEL	Adults (average)	Elderly adults (90th percentile)	Athletes and laborers (high)	DM patients (high)	Bottle-fed infants (typical) NDI patients (high)	Bottle-fed infants (high)
mg/L	mg/kg/day	mg/kg/day	0.011 mg/kg/day	0.022 mg/kg/day	0.05 mg/kg/day	0.07 mg/kg/day	0.1 mg/kg/day	0.14 mg/kg/day
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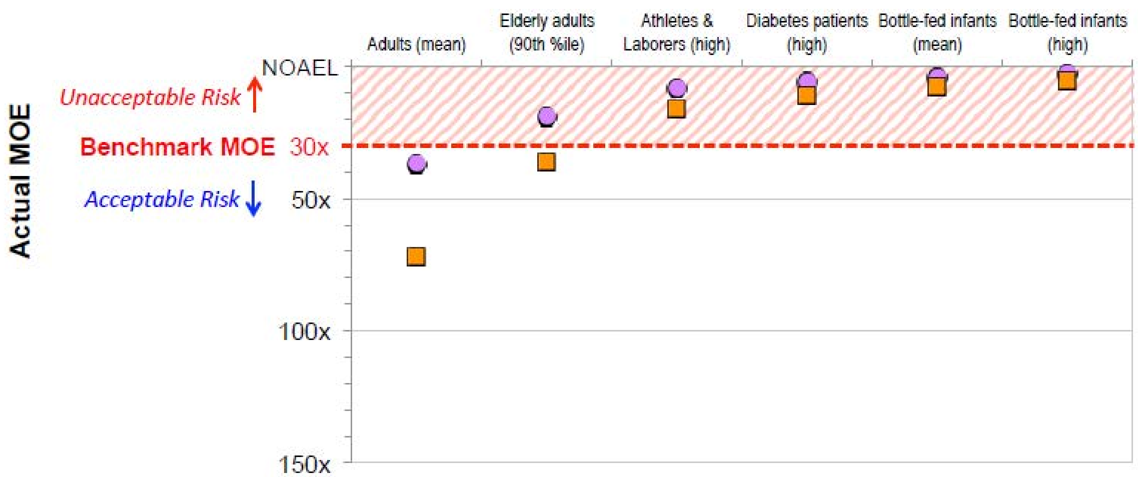
20 mg/L, NOAEL (rats)	3.3	0.79	72	36	16	11	7.9	5.6



C. Assumptions and Key Sources of Uncertainty

202. Uncertainties are inherent to the field of risk assessment; they are to be expected. As discussed throughout my expert report, there are uncertainties involved in a risk assessment of fluoride neurotoxicity,[288] including:

203. *Uncertainties in the Animal Data*: The Points of Departure for both the RfDs and MOEs are derived from developmental animal studies that, while published in the peer-reviewed biomedical literature, have methodological limitations, including lack of control for litter effects, lack of blinding, lack of exposure during the full window of vulnerability (in utero *and* infancy), lack of long-term chronic exposures, and failure to rule out a contributing role of motor and sensory effects in the observed learning/memory deficits. As discussed earlier, the net effect of these limitations is uncertain. On one hand, lack of blinding can inflate the effect size, while on the other hand, lack of exposure during the full window of vulnerability and lack of chronic exposures can deflate it. Similarly, while lack of control for litter effects can create false positives, it can also create false negatives as well.[289] Further, to the extent that fluoride is causing the learning/memory deficits indirectly through a motor/sensory mechanism,[290] this would still be a neurotoxic effect and is thus not a basis to forego risk assessment, particularly since body weight changes do not appear to be a mediating mechanism in the studies from which the Points of Departure have been derived.

[288] For purposes of brevity, my initial expert report did not reiterate each of these uncertainties in the risk characterization section, although I considered them as part of my assessment.
[289] Zorrilla (1997), p. 144; Lazic and Essioux (2013), p. 3.
[290] NTP (2016), p. vii.

204. While there are some uncertainties in the animal data (which is not unusual[291]), there is reasonable confidence that the observed effects are both real and relevant. First, the animal studies have been overwhelmingly consistent—across numerous laboratories and study designs—in finding adverse effects on the brain, both structural and functional, which supports the conclusion that the effects are not an artifact of a methodological limitation. Second, the effect of fluoride on cognition has been detected in studies that have specifically controlled for litter effects and body weight changes, thus suggesting that fluoride's effect on the brain is independent of these concerns.[292] Third, there are extensive human epidemiological data reporting associations between fluoride and reduced IQ, and the existence of these data adds plausibility to the animal data, and vice versa. Fourth, the finding of unacceptable risk through an MOE analysis of the animal toxicity values is consistent with Dr. Grandjean's BMD analysis of the human data, which shows that the level of exposure associated with reduced IQ in humans is well below the levels of exposure produced by fluoridation. The confluence of the animal and human data thus adds strong overall confidence to the assessment.

205. *Uncertainties in the Extrapolation to Humans*: As discussed above, the extrapolation of animal data to humans involves some inherent uncertainty. There does not yet exist a physiologically based toxicokinetic model (PBTK) for fluoride, which would be the optimal method for adjusting for toxicokinetics.[293] This uncertainty has been accounted for by EPA's use of allometric scaling method which accounts for the expected difference in

[291] As discussed earlier, the principal animal studies that EPA has relied upon for its neurotoxicity risk assessments have also had methodological limitations, including failure to control for litter effects. There was also significant uncertainty in the animal data that EPA used for its unreasonable risk determinations for its draft NMP evaluation (e.g., there were only 6 studies available for the endpoint of concern, and three found no effect).

[292] Bartos (2018; 2019).

[293] EPA (2011b), p. 19.

toxicokinetics), and use of an uncertainty factor of 3 (to address the expected difference in toxicodynamics). The use of default allometric scaling for fluoride is consistent with chemical-specific research on fluoride showing that rats and mice require approximately 5 to 10 times more fluoride, respectively, to obtain the same concentration of fluoride in the blood.[294] The allometric scaling thus has a chemical-specific justification for fluoride, which provides confidence to the assessment. But, importantly, even if *no* allometric scaling is done to assess the risk of infant exposures, the MOEs still indicate unacceptable risks for *all* PODs.

206. The use of non-protective (i.e., non-conservative) assumptions provides additional confidence to the assessment. These non-protective assumptions include: (1) the use of 45 mg/L as a LOAEL, despite the fact that studies have found adverse effects well below this concentration; (2) the use of 20 mg/L as a NOAEL in McPherson (2018), despite the fact that the study found a neurotoxic effect at this concentration (i.e., increased pain sensitivity); and (3) conversion of water fluoride concentrations (mg/L) into doses (mg/kg/day) using the lowest end of the reported ratio, which results in Points of Departure that are likely higher than the actual dosages the animals received.

207. *Uncertainties in the Exposure Assessment*: As discussed above, I obtained most of my initial exposure estimates from the NRC's 2006 report, which in turn were based on EPA's own water intake data from 2000,[295] and have also reviewed EPA's 2019 report in which the Agency identified the "most up-to-date and scientifically sound" water intake data to use for risk assessment. Both of EPA's water intake reports (from 2000 and 2019) are based on short-term (2-day) surveys, which introduces some uncertainty when extrapolating to long-term exposures.

[294] NRC (2006), pp. 98, 442; Zhang et al. (2014).
[295] NRC (2006), Appendix B; EPA (2000a).

Long-term surveys, however, do not exist, and the uncertainty of using 2-day surveys is minimized by the large, nationally-representative scale of the survey data. EPA has stated that it has medium-to-high confidence in the reliability of these data, and that the data are well suited for risk assessment of water-based exposures.[296]

208. *Uncertainties in the Human Data*: One of the major strengths of the database on fluoride neurotoxicity is that there is a large body of human data, including five prospective cohort studies that have individual measurements of exposure during the fetal and neonatal period. The large extent of human data for fluoride far surpasses what EPA has used for its draft risk assessments of other chemicals under Section 6,[297] where the Agency has often had to rely *solely* on animal data.

209. The emergence of prospective cohort data on early life exposures to "optimal" levels of fluoride (from salt and water fluoridation programs)[298] addresses the two primary criticisms that have been made with respect to the cross-sectional studies of populations with elevated levels of fluoride in water: i.e., (1) that cross-sectional studies are limited in establishing causation because the exposures are measured after the effect (i.e., IQ loss) has occurred; and (2) the cross-sectional studies involve exposures that are generally higher than what people receive through artificially fluoridated water. The fact that the prospective cohort studies have found cognitive deficits at "optimal" levels of exposure that are consistent with the effects observed in the cross-sectional studies adds *substantial* confidence to the risk characterization.

210. While the human data are very robust, data gaps do remain, particularly with

[296] EPA (2000a), p. 5-5; EPA (2019b), pp. 3-6 & 3-10.

[297] As discussed earlier, the data available for fluoride are also substantially more robust than the data EPA has considered in making hazard determinations for other neurotoxicants.

[298] I understand that Dr. Hu and Dr. Lanphear will be addressing the criticisms with respect to imprecise exposure estimates, and thus I do not address that issue here.

respect to how the causative doses may vary across the population based on life-stage (e.g., the elderly), and other intrinsic sources of susceptibility, such as renal impairment, nutritional deficiencies, and genetic predisposition. These data gaps make it difficult to quantify the extent to which susceptibility varies across the population; the available data on chronic fluoride toxicity, however, provide a high level of confidence that human susceptibility to fluoride varies by a considerable margin, particularly in a population as large and diverse as the United States. Of particular concern are individuals with co-existing susceptibilities, such as pregnant women with iodine deficiencies, neonates that are bottle-fed with fluoridated water, and elderly individuals with diabetes.

211. To account for the *known* (but not yet quantified) variability in human susceptibility, I utilized EPA's default uncertainty factor of 10. This is consistent with EPA's standard practice, including EPA's Section 6 risk evaluations under TSCA. While I derived the Points of Departure from studies on susceptible (i.e., prenatally exposed) animals, the studies did not account for the full range of expected susceptibility in the human population. The studies did not, for example, attempt to replicate the formula-feeding practices of human infants, as all rodents were breast-fed during the critical neonatal period. Nor did the studies attempt to examine the effect of a co-existing iodine deficiency in the mother, or any other factor (e.g., renal impairment, calcium deficiency, etc) that would be expected to exacerbate the effects of prenatal fluoride exposure. Since hundreds of millions of Americans are now exposed to fluoridation chemicals on a regular basis, the spectrum of susceptibility will likely exceed the susceptibility examined in the available animal studies. An uncertainty factor of 10 is thus appropriate and necessary.

IX. RISK DETERMINATION

212. Under TSCA, a risk evaluation has a fifth and final step that is not included within the *Guidelines*: the Risk Determination. In the Risk Determination, EPA assesses whether the risks identified by the Margin of Exposure (MOE) analysis are "unreasonable." In making this determination, EPA considers "relevant risk-related factors," including (i) the effects of the chemical substance under the conditions of use; (ii) number of people exposed; (iii) whether susceptible subpopulations are exposed; (iv) the severity of the hazard; and (v) uncertainties in the data.

213. In practice, EPA's Risk Determination analyses do not address each of the "relevant risk factors" identified above. Severity of the hazard, for example, is rarely discussed. Assessments of uncertainties in EPA's Risk Determinations has also been rather cursory. In the NMP risk evaluation, for instance, the discussion of uncertainties in the analysis was largely limited to the assumptions involved in estimating worker exposure to chemicals in the absence of actual monitoring data.[299] Although EPA's risk estimates were based on an endpoint for which there were only 6 animal studies (with only 3 showing an effect), EPA did not re-address the underlying uncertainties in these data. The Risk Determination should thus not be mistaken as an exhaustive re-examination of all issues previously addressed; instead they tend to be brief and written in summary form.

214. At the time I conducted my initial assessment in this case, EPA had not yet released any risk evaluations under Section 6. For guidance, therefore, I relied on the risk characterization

[299] EPA (2019d), pp. 301-335.

considerations identified in the *Guidelines*,[300] as well as risk evaluations that EPA had recently completed on "new chemicals" under Section 5.

215. The factors that EPA considers under Section 5 substantially overlap with the factors that EPA considers under Section 6. Specifically, EPA considers the following three factors: (i) the hazardous nature of the chemical (as determined by toxicity values in animal studies);[301] (ii) the extent of human exposure to the chemical, and (iii) the Margin of Exposure (MOE). As I described in my report, fluoride meets each of these three criteria for unreasonable risk.

216. Importantly, whether one considers the factors under Section 5 or Section 6, the risk of neurotoxicity posed by fluoridation chemicals constitutes a clear and unreasonable risk, as will now be discussed.

A. Effects of Fluoridation Chemicals Under the Condition of Use

217. In most of the risk evaluations that EPA has conducted thus far under Section 6, the Agency did not have actual human data on health effects associated with the condition of use. EPA had to rely, therefore, on animal data alone. This is not the case with fluoridation. Critically, there are four prospective cohort studies that have examined the impact of optimal fluoride exposures, including two that examined the specific condition of use (water fluoridation) at issue.[302] Under the *Guidelines*, prospective cohort data permit "direct estimates of risks attributed to a particular

[300] EPA (1998a), pp. 63-66.

[301] Under Section 5, a chemical is "considered to have high human health hazard if there is evidence of adverse effects in humans or conclusive evidence of severe effects in animal studies with a **N**OAEL of less than or equal to 10 mg/kg/day." EPA (2018b; 2018c; 2018d; 2018e; 2018f; 2018g; 2019a). This criterion is readily satisfied with fluoride, as the **L**OAELs for cognitive deficits and brain abnormalities are below 10 mg/kg/day. Fluoride is thus a "high human health hazard" under Section 5.

[302] Bashash et al (2017, 2018); Green et al. (2019); Till et al. (2020).

exposure."[303] The effects of fluoridation chemicals under the condition of use are thus well characterized, particularly in comparison to chemicals (e.g., NMP, 1-BP) for which EPA has made unreasonable risk findings under TSCA.

B. Number of Peopled Exposed to Fluoridation Chemicals

218. EPA has recognized that "the significance of the risk is dependent upon both the hazard (or toxicity) of the chemical substance and *the extent of exposure* to the substance."[304] Although EPA made this statement in the context of Section 5, EPA considers the extent of exposure to be a relevant factor under Section 6 as well. In the Section 6 risk determinations, the number of people (usually workers) who are exposed to the chemical are identified under each condition of use.[305]

219. This factor weighs in favor of an unreasonable risk finding for fluoridation chemicals. The extent of human exposure to fluoridation chemicals is nothing short of massive, much like lead exposure was during the era of leaded gasoline. Today, approximately 200 million Americans, or nearly 2/3 of the population, have municipal water to which fluoridation chemicals are added. Moreover, most of the remaining population living in "non-fluoridated" areas will routinely consume fluoridation chemicals in processed beverages and foods, as many beverages and foods are produced in fluoridated areas.[306] To put these numbers in perspective, EPA has found unreasonable risks for conditions of use involving as few as 1,046[307] and 1,900 occupationally-

[303] EPA (1998a), p. 17.
[304] EPA (2018b; 2018c; 2018d; 2018e; 2018f; 2018g; 2019a).
[305] EPA (2019d), pp. 299, 303-335; EPA (2019c), pp. 255-289.
[306] See, for example, Kiritsy et al. (1996); Turner et al. (1998); Heilman et al. (1999).
[307] EPA (2019c), p. 264.

exposed workers.[308] With such widespread exposure to fluoridation chemicals among the general population, even small risks can amount to widespread harm.

C. Exposure of Susceptible Subpopulations to Fluoridation Chemicals

220. One of the consequences from widely dispersing a toxicant through the environment (versus the use of industrial chemicals *within* manufacturing facilities) is that susceptible members of the general public may be exposed. This is the case with fluoridation chemicals. Each year, there are approximately **2.5 million pregnancies** in fluoridated areas; *in utero* exposures are thus widespread. Many of those exposed *in utero* will also be exposed during the sensitive neonatal period, with upwards of **1.9 million infants** living in fluoridated areas being fed formula at least part of the time, including **400,000 infants** who are *exclusively* formula-fed for their first six months. While these numbers do not account for those who use bottled water, the numbers will be substantial regardless.

D. The Severity of the Hazard (Cognitive Deficits/IQ Loss)

221. The principal hazard at issue from exposure to fluoridation chemicals is IQ loss. The prospective studies have found an approximate 5 to 6 point drop in IQ as maternal urinary fluoride levels increase from 0 to 1 mg/L.[309] To put this in perspective, EPA has recognized that a loss of a single IQ point is associated with a loss in lifetime earnings,[310] and EPA's Clean Air Science Advisory Council has stated that "a population loss of 1-2 IQ points is highly significant from a public health perspective" and should be prevented in 99.5% of the population.[311]

[308] EPA (2019d), pp. 307, 311.
[309] Bashash et al. (2017); Green et al. (2019).
[310] EPA (2008e), p. 5-28.
[311] Federal Register (2008), p. 67000.

Consistent with this, EPA has established reference doses for chemicals based on observed cognitive deficits in animal studies (see Table 1 above). Cognitive deficits, including in the range observed in fluoridated areas, are a sufficiently severe effect on human health to warrant prevention, as EPA has recognized in other contexts.

E. Uncertainties

222. Uncertainties are a pervasive aspect of risk assessment; their existence does not negate a finding of risk. As would be expected, there are uncertainties in the fluoride dataset, arising in part from methodological limitations in the available animal studies (e.g., lack of control for litter effects, lack of blinding, lack of studies on neonatal exposures, lack of chronic experiments, etc.). The impact of these limitations on the observed learning and memory deficits is not yet defined. The clear suggestion from the observed findings, however, is that fluoride causes alterations to the brain and behavior. Further, the uncertainties that remain in the animal data are largely offset by the existence of high-quality prospective studies that have *consistently* detected significant associations between "optimal" fluoride exposures and cognitive deficits. While I understand that EPA's experts in this case question whether the "causal" relationship between fluoridation and IQ loss has been proven, the *Guidelines* do not require proof of causation; they require sufficient evidence of association.[312]

223. Another factor weighing in favor of an unreasonable risk finding is that the exposure estimates are more straightforward—and permit greater confidence—than the exposure estimates that EPA has had to extrapolate for other chemicals under TSCA. In its NMP risk evaluation, for example, EPA had to make "assumptions about glove use, glove effectiveness,

[312] EPA (1998a), p. 53.

duration of contact with NMP, concentration of NMP, and amount of skin surface contact with NMP" in order to come up with estimates of human exposure under the conditions of use.[315] Estimating exposure to fluoridation chemicals involves much less uncertainty, as the concentration of fluoride in the water is defined (0.7 mg/L), and the EPA has extensive empirical data on water consumption in the U.S. that the Agency has described as "scientifically sound."

224. Based on the available scientific evidence that now exists on the hazards, exposures, and risks of fluoride ingestion, the widespread addition of fluoridation chemicals to drinking water and processed foods in the United States presents an unreasonable risk to human health.

I declare under penalty of perjury, under the laws of the United States, that the foregoing is true and correct to the best of my knowledge and belief.

Executed on May 20, 2020, in Oak Ridge, Tennessee.

Kathleen M. Thiessen

KATHLEEN THIESSEN, PH.D.

[315] EPA (2019d), Table 5-1.

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Appendix A

Recent Animal Studies of Fluoride Neurotoxicity (Tables A-1 and A-2)

Table A-1. Summary of animal studies that have investigated neuroanatomical and neurochemical endpoints of fluoride toxicity.

Paper	Animal	Sex	Maturity at Start of Exposure	Length of Exposure	Animals per group
Adebayo et al. 2013	Albino Rats	Male	Post-Weaning[f]	1 week	6
Adedara et al. 2017a & b	Wistar Rats	Male	8 weeks old	45 days	12
Agustina et al. 2018	Wistar Rats	Male	Adults	30 days	8
Akinrinade et al. 2015a & b	Wistar Rats	Male	Adults	30 days	5
Ameeramja et al. 2018	Wistar Albino Rats	Female	2 to 3 months old	30 days	6
Atmaca et al. 2014	Wistar Rats	Male	Post-Weaning[f]	21 days	7
Balaji et al. 2015	Swiss Albino Mice	Female	Adults	30 days	6 (of 7)
Banala and Karnati 2015	Wistar Rats	Both	Prenatal	Prenatal + 14, 21 & 30 days	5
Banji et al. 2013	Wistar Rats	Both	Gestational day 6	Prenatal + 15 days	6
Bartos et al. 2018	Wistar Rats	Female	Prenatal	Prenatal + 21 days	5
Bartos et al 2019	Wistar Rats	Both	Pre-Pregnancy	Prenatal + 21 days	5
Basha and Madhusudhan 2010	Wistar Albino Rats	Both	Pre-Pregnancy	Prenatal + 21 days	6
Basha et al. 2011a & b	Wistar Albino Rats	Both	Multigenerational	Prenatal + 12 weeks (3rd generation)	6
Basha and Sujitha 2012a & b	Wistar Rats	Male	3 months old	1 month	6
Basha and Saumya 2013	Albino Mice	Both	Adults	45 days	6
Bharti and Srivastava 2009	Wistar Rats	Female	Adults	28 days	6
Bharti et al. 2012	Wistar Rats	Female	Adults	7 days	6
Chauhan et al. 2013	Sprague-Dawley Rats	Female	6 months old	3 to 6 weeks	4 (of 8)
Chen et al. 2018a	Sprague-Dawley Rats	Female	Pre-Pregnancy	Prenatal + 6 months	6
Chouhan and Flora 2008	Albino Rats	Male	Adults	10 weeks	6
Chouhan et al. 2010	Wistar Albino Rats	Male	Adults	12 weeks	5-6 (of 6)
Dec et al. 2019	Wistar Rats	Males	Pre-Pregnancy	Prenatal + 90 days	6 (of 12)
Dong et al. 2015	Sprague-Dawley Rats	Both	1 month old	>10 months	30
Dong et al. 2015	Sprague-Dawley Rats	Both	10 months pre-birth	Prenatal + 1, 7, 14, 21, & 28 days	10
Flora et al. 2009	Swiss Mice	Male	Adults	10 weeks	5
Flora et al. 2012	Swiss Mice	Male	Adults	28 weeks	5 (of 12)
Ge et al. 2011	Wistar Albino Rats	Both	Pre-Pregnancy	Prenatal + 20 days	8
Ge et al. 2018	ICR Mice	Both	Pre-Pregnancy	Prenatal + 90 days	6
Güner et al. 2016	Wistar Albino Rats	Both	Adult	Prenatal + 1, 3, & 5 months	5

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Table A-1. Summary of animal studies that have investigated neuroanatomical and neurochemical endpoints of fluoride toxicity - *Continued*

Paper	Treatment groups	LOAEL[b]	Specific Effect	Hippocampus?
Adebayo et al. 2013	100 mg/L	100 mg/L	Oxidative stress, reduced brain weight	No
Adedara et al. 2017a & b	6.8 mg/L	6.8 mg/L	Oxidative stress, reduced AChE activity, inflammation, Caspase-3 activity	No
Agustina et al. 2018	2.3, 4.5 & 9 mg/kg/day	4.5 mg/kg/day	Reduced number of Purkinje cells	No
Akinrinade et al. 2015a & b	1 & 5 mg/L	1 mg/L	Oxidative stress, inflammation, neuronal damage	No
Ameeramja et al. 2018	136 mg/L	136 mg/L	Oxidative stress	No
Atmaca et al. 2014	100 mg/L	100 mg/L	Oxidative stress & neuronal degeneration	Yes
Balaji et al. 2015	45 & 90 mg/L	45 mg/L	Inhibition of cholinesterase & increased oxidative stress	No
Banala and Karnati 2015	9 mg/L	9 mg/L	Oxidative stress	No
Banji et al. 2013	9 mg/kg/day	9 mg/kg/day	Oxidative stress	No
Bartos et al. 2018	5 & 10 mg/L (=0.6 & 1.2 mg/kg/d)	5 mg/L	Decreased nicotinic receptors & oxidative stress	Yes
Bartos et al. 2019	5 & 10 mg/L (=0.6 & 1.2 mg/kg/d)	5 mg/L	Increased oxidative stress as reflected by decreased CAT, GPT, and GOT	Yes
Basha and Madhusudhan 2010	50 & 150 mg/L	50 mg/L	Oxidative stress & reduced brain protein content	No
Basha et al. 2011a & b	100 & 200 mg/L	100 mg/L	Oxidative stress, reduced brain weight, and histological changes	Yes
Basha and Sujitha 2012a & b	270 mg/L	270 mg/L	Oxidative stress & decreased acetylcholinesterase activity	No
Basha and Saumya 2013	270 mg/L	270 mg/L	Mitochondrial disturbances & Oxidative stress	No
Bharti and Srivastava 2009	150 mg/L	150 mg/L	Oxidative stress	No
Bharti et al. 2012	150 mg/L	150 mg/L	Decreased acetylcholinesterase activity	No
Chauhan et al. 2013	11.3 mg/kg/day	11.3 mg/kg/day	Oxidative stress	No
Chen et al. 2018a	4.5, 23, 45 mg/L	4.5 mg/L	Impaired synaptogenesis	Yes
Chouhan and Flora 2008	10, 50, & 100 mg/L	100 mg/L[c]	Oxidative stress	No
Chouhan et al. 2010	1, 10, 50 & 100 mg/L	1 mg/L	Oxidative stress, alterations in neurotransmitters, neuronal lesions, & increased AChE activity	No
Dec et al. 2019	23 mg/L	23 mg/L	Evidence of inflammatory processes (reduced activity of cyclooxygenases (COX1 & COX2) and increase in prostaglandins)	Yes
Dong et al. 2015	50 mg/L (adults)	50 mg/L	Decrease in muscarinic nicotinic receptors	No
Dong et al. 2015	50 mg/L (offspring)	50 mg/L	Decrease in muscarinic nicotinic receptors	No
Flora et al. 2009	50 mg/L	50 mg/L	Oxidative stress, alteration in neurotransmitters, DNA damage, increased AChE activity	No
Flora et al. 2012	50 mg/L	50 mg/L	Oxidative stress, neuronal degeneration, DNA damage, Protein interaction	Yes
Ge et al. 2011	100 mg/L (+25 mg/kg in food)	100 mg/L	Alteration in protein expression	No
Ge et al. 2018	50 & 100 mg/L	50 mg/L	Alterations of synapse-related proteins	No
Güner et al. 2016	13.6 & 45 mg/L	13.6 mg/L	Neurodegenerative changes & catalase immunoreactivity	Yes

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Table A-1. Summary of animal studies that have investigated neuroanatomical and neurochemical endpoints of fluoride toxicity - *Continued*

Paper	Animal	Sex	Maturity at Start of Exposure	Length of Exposure	Animals per group
Hamza et al. 2015	Wistar Albino Rats	Male	Adults	30 days	10
Han et al. 2014	Kumming Mice	Male	Sexually matured mice	180 days	4 (of 15)
Hassan and Abdel-Aziz 2010	Wistar Albino Rats	Male	Adults	5 weeks	6
Inkielwicz-Stepniak and Czarnowski 2010	Wistar Han Rats	Male	6 weeks old	4 weeks	6
Jia et al. 2019	CD1 Mice	Both	Prenatal	Prenatal (day 9) + 19 days	5 (of 20)
Jiang et al. 2014a	Sprague-Dawley Rats	Male	Weaned	3 months	8
Jiang et al. 2014b	Sprague-Dawley Rats	Both	Pre-pregnancy	Prenatal + 2 months	3-12 (of 12)
Jiang et al. 2019	Sprague-Dawley Rats	Male	Post-weaning[f]	10 weeks	7
Kaur et al. 2009	Sprague-Dawley Rats	Female	Adults	8 weeks	6-7 (of 8)
Khan et al. 2018	Wistar Rats	Both	Post-weaning[f]	28 days	6
Kinawy 2019	Rats	Male	Prenatal (6th day)	Prenatal + Weaning or 70 days	8
Li et al. 2019	Kumming Mice	Both	Adults	90, 120 & 150 days	8 (of 30)
Liu et al. 2010	Sprague-Dawley Rats	Both	Post-weaning[f]	6 months	10 (of 24)
Liu et al. 2011	Sprague-Dawley Rats	Both	Post-weaning[f]	3 & 6 months	12 (of 24)
Lou et al. 2013	Sprague-Dawley Rats	Both	Post-weaning[f]	6 months	20
Ma et al. 2015	C57/BL Mice	Male	4 weeks old	4 weeks	8
Mansour and Tawfik 2012	Albino Rats	Male	Adults	5 weeks	6
McPherson et al. 2018	Long Evans Hooded Rats	Male	Prenatal	Prenatal (day 6) + 90 days	6 (of ~23)
Nabavi et al. 2012a	Wistar Rats	Male	8 to 12 weeks old	1 week	10
Nabavi et al. 2012b	Wistar Rats	Male	Post-weaning[f]	1 week	10
Nabavi et al. 2013	Wistar Rats	Male	7 days old	7 days	10
Niu et al. 2009	Wistar Albino Rats	Both	Day of birth	6, 8, 10, & 12 weeks	8
Niu et al. 2014	Kumming Mice	Male	Prenatal	Prenatal + 56 days	15
Niu et al. 2015a	Kumming Mice	?	Adults	60 days	5 (of 15)
Niu et al. 2015b	Kumming Mice	Both	Prenatal	Prenatal + 56 days	6
Niu et al. 2018a	Sprague-Dawley Rats	Female	Post-weaning[f]	60 days	3 (of 10)
Niu et al. 2018b	Kumming Mice	Both	Adults	60 days	5 (of 12)

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Table A-1. Summary of animal studies that have investigated neuroanatomical and neurochemical endpoints of fluoride toxicity – *Continued*

Paper	Treatment groups	LOAEL	Specific Effect	Hippocampus?
Hamza et al. 2015	4.7 mg/kg/day	4.7 mg/kg/day	Increased oxidative stress	No
Han et al. 2014	11, 23, and 45	23 mg/L[c]	Altered mRNA expression	Yes
Hassan and Abdel-Aziz 2010	4.7 mg/kg/day	4.7 mg/kg/day	Oxidative stress	No
Inkielwicz-Stepniak and Czarnowski 2010	25 mg/L	25 mg/L	Oxidative stress	No
Jia et al. 2019	6 & 113 mg/L	None	No reduction in neuronal density	No
Jiang et al. 2014a	54 mg/L	54 mg/L	Decreased glutamate levels	Yes
Jiang et al. 2014b	11, 23, and 45 mg/L	11 mg/L	Neuronal degeneration, decreased glucose utilization	Yes
Jiang et al. 2019	23 & 45 mg/L	23 mg/L	Impaired neurogenesis & synaptic plasticity	Yes
Kaur et al. 2009	125 mg/L	125 mg/L	Oxidative stress, alteration in neurotransmitters, & neuronal degeneration	No
Khan et al. 2018	20 mg/L	20 mg/L	Inhibition of AChE and increase in oxidative stress	No
Kinawy 2019	678 mg/L	678 mg/L	Oxidative stress	Yes
Li et al. 2019	68 mg/L	68 mg/L	Altered mRNA expression of anxiety & depression-related genes	Yes
Liu et al. 2010	5 & 50 mg/L	5 mg/L	Reductions in nicotinic receptors & activation of photoho-ERK1/2	No
Liu et al. 2011	5 & 50 mg/L	5 mg/L	Increased apoptosis & phosphorylation	No
Lou et al. 2013	10 & 50 mg/L	10 mg/L	Mitochondrial disturbances in neurons, altered protein expression	No
Ma et al. 2015	23 & 45.6 mg/L	23 mg/L	Increased BDNF expression	Yes
Mansour and Tawfik 2012	4.7 mg/kg/day	4.7 mg/kg/day	Oxidative stress	No
McPherson et al. 2018	10 & 20 mg/L (+ food exposure group)	None	No neuronal damage or glia reactivity	Yes
Nabavi et al. 2012a	270 mg/L	270 mg/L	Oxidative stress	No
Nabavi et al. 2012b	270 mg/L	270 mg/L	Oxidative stress	No
Nabavi et al. 2013	270 mg/L	270 mg/L	Oxidative stress	No
Niu et al. 2009	68 mg/L	68 mg/L	Decreased glutamate levels & altered enzyme activity	Yes
Niu et al. 2014	68 mg/L	68 mg/L	Altered protein expression	Yes
Niu et al. 2015a	11, 23, and 45 mg/L	23 mg/L	Microtubule lesions in neurons	Yes
Niu et al. 2015b	68 mg/L	68 mg/L	Alterations in protein expression	No
Niu et al. 2018a	4.5, 23, and 45 mg/L	4.5 mg/L	Endoplasmic reticulum stress	Yes
Niu et al. 2018b	11, 23, and 45 mg/L	11 mg/L	Myelin damage, and alteration to synaptic structure	Yes

Table continued next page

Table A-1. Summary of animal studies that have investigated neuroanatomical and neurochemical endpoints of fluoride toxicity – *Continued*

Paper	Animal	Sex	Maturity at Start of Exposure	Length of Exposure	Animals per group
Pal and Sarkar 2014	Wistar Rats	Male	Post-weaning[f]	30 days	6 to 8
Pan et al. 2015	Sprague-Dawley Rats	Male	3 weeks after weaning	30 days	15
Pereira et al. 2011	Wistar Rats	Male	30 days old	30 days	4-10 (of 15)
Pulungan et al. 2018	Wistar Rats	Male	12-16 weeks old	30 days	8
Qian et al. 2013	Sprague-Dawley Rats	Male	Newly weaned	6 months	2-20 (of 20)
Reddy et al. 2009	Swiss Albino Mice	Female	Adults	14 days	6
Reddy et al. 2014	Wistar Rats	Male	4 months old	90 days	6
Rogalska et al. 2017	Wistar Rats	Both	8 weeks old	4 weeks	6-8
Samanta et al. 2016	Sprague-Dawley Rats	Female	Post-weaning[f]	16 weeks	5
Sarkar et al. 2014	Wistar Rats	Male	Post-weaning[f]	30 days	6
Shalini and Sharma 2015	Wistar Albino Rats	Female	Adults	60 days	10
Sharma et al. 2014	Swiss Albino Mice	Male	1.5 months old	30 days	7
Sharma et al. 2018	Swiss Albino Mice	Both	1 month old	30 days	7
Shen et al. 2019	Wistar Rats	Both	1 month old	12 & 24 weeks	30
Sun et al. 2017	Sprague-Dawley Rats	Both	Pre-Pregnancy	Prenatal + 14 or 28 days	10
Sun et al. 2018	Kumming Mice	Female	Prenatal	Prenatal + 21 days	6 (of 12)
Teng et al. 2018	Sprague-Dawley Rats	Male	Recently weaned	18 months	6-7 (of 13)
Trivedi et al. 2007	Swiss Albino Rats	Male	Young adults	30 days	10
Wang et al. 2018a	ICR Mice	Female	Prenatal	Prenatal (7th day) + 21 days	6 (of 15)
Wang et al. 2018b	Wistar Albino Rats	Male	12-weeks old	8 weeks	10 (of 24)
Wei et al. 2018	Sprague-Dawley Rats	Both	1 month old	>6 months	15
Wei et al. 2018	Sprague-Dawley Rats	Both	Pre-Pregnancy	Prenatal + 28 days	6-10 (of 10)
Yan et al. 2016	Wistar Rats	Both	5 weeks old	10 weeks	20
Yang et al. 2018a	Wistar Rats	Male	6 weeks old	4 & 12 weeks	4-6 (of 10)
Yu et al. 2019	ICR Mice	Male	Newly weaned	3 & 6 months	20
Yuan et al. 2019	Kumming Mice	Male	7 weeks old	90 days	12 (of 24)
Zhang et al. 2013a	Wistar Rats	Male	6 weeks old	3 months	3 (of 10)
Zhang et al. 2015a	Sprague-Dawley Rats	Both	2 months old	3 months & 6 months	10 (of 20)

Table continued next page

Table A-1. Summary of animal studies that have investigated neuroanatomical and neurochemical endpoints of fluoride toxicity – *Continued*

Paper	Treatment groups	LOAEL	Specific Effect	Hippocampus?
Pal and Sarkar 2014	9 mg/kg/day	9 mg/kg/day	Oxidative stress, inhibited enzymes, altered neurotransmitters, reduced protein content	No
Pan et al. 2015	9 mg/kg/day	N/A[d]	Altered protein expression	Yes
Pereira et al. 2011	45 mg/L	45 mg/L	Alterations in neurotransmitters	Yes
Pulungan et al. 2018	2.3, 4.5 & 9 mg/kg/day	none	No reduction in number of pyramidal cells in medial prefrontal cortex	No
Qian et al. 2013	23 mg/L	23 mg/L	Impaired synaptic plasticity, oxidative stress, altered protein expression	Yes
Reddy et al. 2009	9 mg/kg/day	9 mg/kg/day	Oxidative stress & altered enzyme activity	No
Reddy et al. 2014	9, 27, & 45 mg/L	9 mg/L	Oxidative stress, alterations in neurotransmitters, and immunosuppression	No
Rogalska et al. 2017	4.5 & 23 mg/L	23 mg/L[c]	Increased glucose uptake	Yes
Samanta et al. 2016	5.9 mg/kg/day	5.9 mg/kg/day	Oxidative stress, cellular degeneration, apoptosis	No
Sarkar et al. 2014	9 mg/kg/day	9 mg/kg/day	Oxidative stress, inhibited enzymes, & reduced protein content	No
Shalini and Sharma 2015	10 mg/L	10 mg/L	Oxidative stress, reduced protein content & AChE activity	No
Sharma et al. 2014	120 mg/L	120 mg/L	Oxidative stress & cellular degeneration	Yes
Sharma et al. 2018	54 mg/L	54 mg/L	Oxidative stress and neuronal damage	Yes
Shen et al. 2019	200 mg/L	200 mg/L	Apoptosis and degeneration of nerve cells in spinal cord	No
Sun et al. 2017	45 mg/L	45 mg/L	Altered gene expression & apoptosis	Yes
Sun et al. 2018	11, 23, and 45 mg/L	11 mg/L (Fig 3b)	Altered mRNA expression	Yes
Teng et al. 2018	8.25, 16.5, & 33 mg/L	16.5 mg/L[c]	Elevated calcium in hippocampus	Yes
Trivedi et al. 2007	2.7 & 5.4 mg/kg/day	2.7 mg/kg/day	Reduced protein content	No
Wang et al. 2018a	11, 23, and 45 mg/L	11 mg/L (Fig 4b)	Altered expression of mi-RNAs	No
Wang et al. 2018b	45 mg/L	45 mg/L	Cellular degeneration, DNA damage	Yes
Wei et al. 2018	50 mg/L (adults)	50 mg/L	Neuronal injury (as evident by damage to Nissl bodies)	No
Wei et al. 2018	50 mg/L (offspring)	50 mg/L	Neuronal injury (as evident by damage to Nissl bodies)	No
Yan et al. 2016	60 & 120 mg/L	60 mg/L	Increased apoptosis & inflammation	Yes
Yang et al. 2018a	60 & 120 mg/L	60 mg/L	Apoptosis, altered protein expression, increased inflammation	Yes
Yu et al. 2019	2.3 & 13.6 mg/L	2.3 mg/L	Alterations of L-type calcium channels	Yes
Yuan et al. 2019	23, 45, 68 mg/L	23 mg/L	Reduced brain protein content, impaired insulin signaling pathway, reduced brain organ coefficient	Yes
Zhang et al. 2013a	45 mg/L	45 mg/L	Oxidative stress, neuronal loss, altered protein expression	Yes
Zhang et al. 2015a	5 & 50 mg/L	5 mg/L	Increased oxidative stress & activation of AGE/RAGE Pathway	Yes

Table continued next page

Table A-1. Summary of animal studies that have investigated neuroanatomical and neurochemical endpoints of fluoride toxicity – *Continued*

Paper	Animal	Sex	Maturity at Start of Exposure	Length of Exposure	Animals per group
Zhang et al. 2017a	Sprague-Dawley Rats	Both	Pre-Pregnancy	Prenatal + 14 or 28 days	15-20 (of 20)
Zhang et al. 2019	Wistar Rats	Both	4 weeks old	3 months	2-3 (of 20)
Zhao et al. 2019	Sprague-Dawley Rats	Both	Pre-Pregnancy	Prenatal + 60 days	2-5 (of 15)
Zheng et al. 2016	Sprague-Dawley Rats	Male	Newly weaned	3 months	20
Zhou et al. 2019	Sprague-Dawley Rats	Female	Pre-Pregnancy	Prenatal + 6 months	6
Zhu et al. 2011 & Zhang et al. 2011	Sprague-Dawley Rats	Male	Just weaned	9 months	6 (of 12)
Zhu et al. 2017	Sprague-Dawley Rats	Both	Prenatal	Prenatal + 21 or 42 days	6 (of 8)

Table continued next page

Table A-1. Summary of animal studies that have investigated neuroanatomical and neurochemical endpoints of fluoride toxicity – *Continued*

Paper	Treatment groups	LOAEL	Specific Effect	Hippocampus?
Zhang et al. 2017a	45 mg/L	45 mg/L	Impaired synaptic plasticity	No
Zhang et al. 2019	25, 50, 100 mg/L	50 mg/L[e]	Autophagy in hippocampus	Yes
Zhao et al. 2019	4.5, 23, 45 mg/L	4.5 mg/L [Fig 6e]	Mitochondrial disturbances	Yes
Zheng et al. 2016	45 mg/L	45 mg/L	Increased apoptosis	Yes
Zhou et al. 2019	4.5, 23, 50 mg/L	23 mg/L[c]	Decreased neurons, suppressed autophagy, and enhanced apoptosis in hippocampus	Yes
Zhu et al. 2011 & Zhang et al. 2011	7, 13.6, & 27 mg/L	13.6 mg/L[c]	Decrease in synaptic membrane fluidity & increased calcium	Yes
Zhu et al. 2017	34 mg/L	34 mg/L	Altered protein expression in ERK/CREB signaling pathway	Yes

[a] Where the study does not identify the sex of the animals, it is assumed that both sexes were studied.

[b] A LOAEL refers to the lowest observed adverse effect level where a statistically significant result was observed.

[c] At least one effect was seen at lower treatment doses (as reflected by a visually apparent dose-related trend), but the effect(s) at the lower treatment levels did not reach statistical significance.

[d] The authors did not perform a statistical analysis to determine if the observed changes were statistically significant.

[e] Ultrastructural observations of the rat hippocampal CA1 cells identified changes in the 25 mg/L group (i.e., increased lipofuscin content), but a statistical analysis of these changes was not performed.

[f] Where the study does not identify the age of the animal at the start of the experiment, it is assumed that the animals had already completed weaning.

Table A-2. Summary of animal studies that have investigated toxic effects of fluoride on learning and memory

Paper	Strain	Sex[a]	Maturity at Start of Exposure	Length of Exposure	Animals per group
Banala and Karnati 2015	Wistar Rats	Both	Prenatal	Prenatal + 30 days	5
Bartos et al. 2018	Wistar Rats	Female	Prenatal (day 0)	Prenatal + 21 days	9-10
Bartos et al. 2019	Wistar Rats	Both	Prenatal (day 0)	Prenatal + 21 days	9-10
Basha et al. 2011b	Wistar Albino Rats	Both	Prenatal/Multigenerational	Prenatal + 30 days	6
Basha & Sujitha 2012b	Wistar Rats	Male	3 months old	1 month	6
Bera et al. 2007	Wistar Rats	Both	Prenatal (day 1)	Prenatal (day 1) + 9 days	6-12
Chen et al. 2018a	Sprague-Dawley Rats	Female	2 months pre-gestation	Prenatal + 6 months	6
Chioca et al. 2008	Wistar Rats	Male	Adult	30 days	15 (of 18)
Cui et al. 2017	Sprague-Dawley Rats	Both	Pre-Pregnancy	Prenatal + 60 days	12
Dong et al. 2015	Sprague-Dawley Rats	Both	One month old	10 months	30
Dong et al. 2015	Sprague-Dawley Rats	Both	10 months pre-birth	Prenatal + 1 to 28 days	10
Ge et al. 2018	ICR Mice	Both	Pre-Pregnancy	Prenatal + 60 days	6
Han et al. 2014	Kunming Mice	Male	Sexually matured mice	180 days	15
Jetti et al. 2016	Wistar Rats	Male	Adult	30 days	6
Jiang et al. 2014a	Sprague-Dawley Rats	Male	Weaned	3 months	8
Jiang et al. 2014b	Sprague-Dawley Rats	Both	Pre-pregnancy	Prenatal + 2 months	12
Liu et al. 2010	Sprague-Dawley Rats	Both	Adult	6 months	10 (of 24)
Liu et al. 2014	BaB/C Mice	Male	4 weeks old	4 weeks	11-12 (of 12)

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Table A-2. Summary of animal studies that have investigated toxic effects of fluoride on learning and memory - *Continued*

Paper	Strain	Sex[a]	Maturity at Start of Exposure	Length of Exposure	Animals per group
McPherson et al. 2018	Long-Evans Hooded Rats	Male	Prenatal (day 6)	Prenatal (day 6) + 90 days	11-23 (of ~23)
Niu et al. 2009	Wistar Albino Rats	Both	Postnatal (day 0)	6 to 12 weeks	8
Niu et al. 2014	Kunming Mice	Male	Prenatal	Prenatal + 56 days	15
Niu et al. 2018a	Sprague Dawley Rats	Female	Post-weaning[g]	2 months	6 (of 10)
Pereira et al. 2011	Wistar Rats	Male	30 days old	30 days	14-15
Pulungan et al. 2018	Wistar Rats	Male	12 to 16 weeks old	30 days	8
Raghu et al. 2013	Wistar Rats	Male	1 month old	30 days	6
Shalini and Sharma 2015	Wistar Albino Rats	Female	Adults	60 days	10
Sharma et al. 2018	Swiss Albino Mice	Male	1 month old	30 days	7
Sun et al. 2018	Kunming Mice	Both	Prenatal	Prenatal + 21 days	6 (of 12)
Wang et al. 2018a	ICR Mice	Female	Prenatal	Prenatal (day 7) + 21 days	15
Whitford et al. 2009	Sprague-Dawley Rats	Female	8 days after weaning	8 months	8
Yang et al. 2018a	Wistar Rats	Male	6 weeks old	4 to 12 weeks	10
Yuan et al. 2019	Kunming Mice	Male	7 weeks old	12 weeks	12 (of 24)
Zhang et al. 2013a	Wistar Rats	Male	6 weeks old	3 months	3 (of 10)
Zhang et al. 2019	Wistar Rats	Both	4 weeks old	3 months	15 (of 20)
Zhao et al. 2019	Sprague-Dawley Rats	Both	Pre-Pregnancy	Prenatal + 60 days	5 (of 15)
Zheng et al. 2016	Sprague-Dawley Rats	Male	Newly weaned	3 months	20
Zhou et al. 2019	Sprague-Dawley Rats	Female	Pre-Pregnancy	Prenatal + 6 months	6
Zhu et al. 2017	Sprague-Dawley Rats	Both	Prenatal	Prenatal + 42 days	8

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Table A-2. Summary of animal studies that have investigated toxic effects of fluoride on learning and memory - *Continued*

Paper	Treatment groups	Selected Tests	LOAEL[b]
Banala and Karnati 2015	20 mg/L	Maze Learning	20 mg/L
Bartos et al. 2018	5 & 10 mg/L	Step Down Inhibitory Avoidance	5 mg/L
Bartos et al. 2019	5 & 10 mg/L	Step Down Inhibitory Avoidance	5 mg/L
Basha et al. 2011b	100 & 200 mg/L	T Maze	100 mg/L
Basha & Sujitha 2012b	270 mg/L	T Maze	270 mg/L
Bera et al. 2007	1.13 & 2.3 mg/kg/day	Active Avoidance / Novel Object Recognition	2.3 mg/kg
Chen et al. 2018a	4.5, 23, & 45 mg/L	Morris Water Maze	23 mg/L[c]
Chioca et al. 2008	23 & 45 mg/L (5.15 & 10.77 mg/kg/day)	Open Field / Two-Way Active Avoidance	23 mg/L
Cui et al. 2017	4.5, 23, & 45 mg/L	Morris Water Maze	4.5 mg/L
Dong et al. 2015	50 mg/L	Morris Water Maze	50 mg/L (adults)
Dong et al. 2015	50 mg/L	Morris Water Maze	50 mg/L (pups)
Ge et al. 2018	50 & 100 mg/L	Morris Water Maze	50 mg/L
Han et al. 2014	11, 23, and 45 mg/L	Novel Object Recognition / Open Field	45 mg/L[d]
Jetti et al. 2016	100 mg/L	T Maze / Passive Avoidance	100 mg/L
Jiang et al. 2014a	55 mg/L	Morris Water Maze	55 mg/L
Jiang et al. 2014b	11, 23, & 45 mg/L	Morris Water Maze	11 mg/L
Liu et al. 2010	2.3 & 23 mg/L	Morris Water Maze	2.3 mg/L
Liu et al. 2014	0.9, 2.3, and 4.5 mg/L	Morris Water Maze / Novel Object Recognition / Elevated-Plus Maze	2.3 mg/L[c]

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Table A-2. Summary of animal studies that have investigated toxic effects of fluoride on learning and memory - *Continued*

Paper	Treatment groups	Selected Tests	LOAEL[b]
McPherson et al. 2018	10 & 20 mg/L (+food exposure group)	Open Field / Elevated Plus Maze / Passive Avoidance / Morris Water Maze / Y Maze	None
Niu et al. 2009	68 mg/L	Y Maze	68 mg/L
Niu et al. 2014	68 mg/L	Novel Object Recognition	68 mg/L[e]
Niu et al. 2018a	4.5, 23, & 45 mg/L	Morris Water Maze	23 mg/L[c]
Pereira et al. 2011	45 mg/L	Open Field	45 mg/L
Pulungan et al. 2018	2.3, 4.5 & 9 mg/kg/day	Y Maze	None[f]
Raghu et al. 2013	100 mg/L	T Maze / Passive Avoidance	100 mg/L
Shalini and Sharma 2015	10 mg/L	Maze Test	10 mg/L
Sharma et al. 2018	68 mg/L	Morris Water Maze / Classic Maze	68 mg/L
Sun et al. 2018	11, 23, & 45 mg/L	Radial Arm Maze / Open Field	23 mg/L[c]
Wang et al. 2018a	11, 23, & 45 mg/L	Open Field / Eight-Arm Maze	23 mg/L[c]
Whitford et al. 2009	2.9, 5.7, & 11.5 mg/kg/day	Appetitive Based Learning	None
Yang et al. 2018a	60 & 120 mg/L	Morris Water Maze / Open Field	60 mg/L
Yuan et al. 2019	23, 45, & 68 mg/L	Y Maze	23 mg/L
Zhang et al. 2013a	45 mg/L	Y Maze	45 mg/L
Zhang et al. 2019	25, 50, & 100 mg/L	Morris Water Maze	100 mg/L[c]
Zhao et al. 2019	4.5, 23, & 45 mg/L	Morris Water Maze	23 mg/L
Zheng et al. 2016	45 mg/L	Morris Water Maze / Open Field	45 mg/L
Zhou et al. 2019	4.5, 23, & 45 mg/L	Morris Water Maze	23 mg/L[c]
Zhu et al. 2017	45 mg/L (8 to 11 mg/kg/day)	Morris Water Maze	45 mg/L

[a] Where the study does not identify the sex of the animals, it is assumed that both sexes were studied.

[b] A LOAEL refers to the lowest observed adverse effect level where a statistically significant result was observed.

[c] At least one effect was seen at lower treatment doses (as reflected by a visually apparent dose-related trend), but the effect(s) at the lower treatment levels did not reach statistical significance.

[d] At least one statistically significant effect was seen at lower treatment doses but for a neurological endpoint that is not specific to learning or memory impairments.

[e] The effect in the fluoride + lead treatment group was statistically significant, but the effect in the fluoride-only treatment group did not reach statistical significance.

[f] A statistically significant effect was observed in the low treatment dose group (5 mg/kg/day) when compared to the control, but there were no significant differences between the control and mid/high dose treatment groups (10 mg/kg/day & 20 mg/kg/day).

[g] Where the study does not identify the age of the animal at the start of the experiment, it is assumed that the animals had already completed weaning

Appendix B

KATHLEEN M. THIESSEN, Ph.D.
Senior Scientist
Oak Ridge Center for Risk Analysis, Inc.

Education

Ph.D.	1986	Genetics, University of Tennessee-Oak Ridge, Graduate School of Biomedical Sciences, Oak Ridge, TN
B.A.	1981	Biology and Chemistry (*Summa cum laude*), Covenant College, GA

Capabilities

Health Effects Assessment
Dose and Risk Assessment
Analysis of Environmental Transport and Exposure Pathways
Uncertainty and Sensitivity Analysis
Technical Writing/Editing, Technical and Public Presentations

Experience Summary

Dr. Thiessen is experienced in the evaluation of exposures, doses, and risks to human health from trace levels of contaminants in the environment and in the use of uncertainty analysis for environmental and health risk assessment. She has served on two National Research Council subcommittees, one charged with the review of fluoride exposure and toxicology, and one dealing with guidance levels for air contaminants (including hydrogen fluoride) in submarines. Dr. Thiessen has also written two reports for the U.S. Environmental Protection Agency, one on the health effects of hydrogen fluoride and related compounds, and one on the health effects of mercuric chloride. Dr. Thiessen has led several working groups on urban contamination and dose reconstruction for the International Atomic Energy Agency's programs on environmental transport modeling and has served on the coordinating committees of the programs; she currently leads a working group on assessment of exposures and countermeasures in urban environments. She also serves on a committee for the preparation of a new International Atomic Energy Agency report on modeling the impacts of planned discharges or radioactivity, and she is involved in the preparation of an IAEA guidance document on implementation of remediation strategies following accidental releases of radioactivity. Dr. Thiessen participated in two symposia on reconstruction of internal doses from Fukushima releases organized by Japan's National Institute of Radiological Sciences, and she has served as a consultant on environmental modeling issues to the Korea Atomic Energy Research Institute and on uncertainty analysis to the National Council on Radiation Protection and Measurements. Dr. Thiessen contributed to the development of a risk-based screening approach to prioritize further investigation of contaminants and exposure situations in various assessment contexts, and she led in the application of risk-based screening techniques for the reconstruction of doses and health risks associated with releases of chemicals and radionuclides from the U.S. Department of Energy's Oak Ridge (Tennessee) facilities. Dr. Thiessen also led an analysis of human exposures, doses, and health risks to off-site individuals associated with historic releases of radionuclides to the Clinch River from the Oak Ridge facilities.

Experience

1992-present Senior Scientist and Director, Oak Ridge Center for Risk Analysis, Inc. (Formerly *SENES* Oak Ridge, Inc., Center for Risk Analysis), Oak Ridge, TN.

- Review of data on contaminant exposure and toxicology.
- Analysis of environmental transport and exposure pathways.
- Screening techniques for environmental assessment.
- Dose reconstruction.
- Uncertainty analysis for environmental assessment.
- International model validation using Chernobyl data sets.
- Working Group Leader for International Atomic Energy Agency research programs.
- Project coordination.
- Technical review.

1991-1992 Consultant and Technical Writer. Environmental Sciences Division, Oak Ridge National Laboratory, Oak Ridge, TN.

1987-1992 Lecturer in Genetics. University of Tennessee, Oak Ridge Graduate School of Biomedical Sciences.

1986-1989 Oak Ridge National Laboratory, Health and Safety Research Division, Chemical Hazard Evaluation Program.

- Assessment of health effects from chemicals.
- Risk assessment.
- Technical review.

Publications and Technical Reports

Periáñez, R., Thiessen, K.M., Chouhan, S.L., Mancini, F., Navarro, E., Sdouz, G., and Trifunović, D. 2016. Mid-range atmospheric dispersion modelling. Intercomparison of simple models in EMRAS-2 project. *Journal of Environmental Radioactivity* 162-163:225-234.

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Associations of low level of fluoride exposure with dental fluorosis among U.S. children and adolescents, NHANES 2015–2016

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ABSTRACT

Drinking water fluoridation was a mid-twentieth century innovation based on the medical hypothesis that consuming low doses of fluoride at the teeth forming years provided protection against dental decays. Numerous studies showed that high level exposure to fluoride could cause dental and skeleton fluorosis. However, there was limited study focusing on the fluorosis effect of low levels of exposure to fluoride. Therefore, our study aimed to examine whether the low level of fluoride exposure (measured in blood plasma and household tap water) was associated with the risk of dental fluorosis based on data of the National Health and Nutrition Examination Survey (NHANES) 2015–2016. We analyzed data in 2098 children and adolescents who had Dean's Index scores, and water and plasma fluoride measures. The Dean's Index score was measured by calibrated dental examiners using the modified Dean's fluorosis classification system. Fluoride was measured in plasma and household tap water. In this study, we found that the rate of fluoride concentration in water above the recommended level of 0.7 mg/L was 25%, but the prevalence of dental fluorosis was 70%. Binary logistic regression adjusted for covariates showed that higher water fluoride concentrations (0.31–0.50, 0.51–0.70, > 0.70 compared 0.00–0.30) were associated with higher odds of dental fluorosis (OR = 1.48, 95% CI: 1.13–1.96, $p = 0.005$; OR = 1.92, 95% CI: 1.44–2.58, $p < 0.001$, and OR = 2.30, 95% CI: 1.75–3.07, $p < 0.001$, respectively). The pattern of regression between plasma fluoride and dental fluorosis was similar. Inclusion, our study showed that even low level of water or plasma fluoride exposure was associated with increased the risk of dental fluorosis. The safety of public health approach of drinking water fluoridation for global dental caries reduction are urgently needed further research.

1. Introduction

Fluoride is the ionic form of the naturally occurring fluorine element. People can consume adequate amounts of fluoride from fluoridated water, foods and beverages, and toothpaste and other dental products containing fluoride (Buzalaf, 2018; Levy et al., 2001). The anion increases the structural stability of teeth and bones through interactions with calcium phosphates (Bronckers et al., 2009). Oral exposure to fluoride primarily via consumption of fluoridated water has been shown to be associated with decreased prevalence of dental caries in children Featherstone, 1999). In response to these findings, community water fluoridation programs were developed to add fluoride to drinking water for preventing tooth decay. In 1962, the U.S. Public Health Service recommended fluoride concentrations in water of 0.7–1.2 mg/L to prevent dental decay (U.S. Department of Health and Human Services Federal Panel on Community Water Fluoridation, 2015).

Exposure to excessive fluoride levels can result in dental fluorosis, characterized by increased porosity of the subsurface enamel and well mineralized surface layer of the enamel. The water fluoride level of 2.0 mg/L is reported to be the threshold that can cause severe dental fluorosis in U.S. children (Selwitz et al., 1998), whereas Rango et al. (2014) found that the children barely had severe dental fluorosis with water fluoride concentrations < 4.0 mg/L in Ethiopian. Although with different thresholds of fluoride level for dental fluorosis, all these studies have confirmed high fluoride exposure can cause dental fluorosis (Ayoob and Gupta, 2006). However, the evidence on the potentially harmful effects of chronic exposure to low level of fluoride on children's dental development is relatively insufficient.

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In many countries, small amounts of fluoride were added to drinking water, salt, or milk to reduce incidence of tooth decay. In the U.S., fluoridation of public water supplies was started in 1945 (U.S. Department of Health and Human Services Federal Panel on Community Water Fluoridation, 2015). Recent years, studies showed that the prevalence of dental fluorosis was increasing after water fluoridation programs (Neurath et al., 2019; Wiener et al., 2018). A more recent analysis of NHANES data in 200–2002 and 2011–2012 found that prevalence of dental fluorosis increased from 29.7% to 61.3% (Wiener et al., 2018). So, water fluoridation has become a controversial public health intervention these years (Peckham and Awofeso, 2014; Spencer and Limeback, 2018). In order to minimize the unwanted effect caused by water fluoridation, more research might be needed to reevaluate the current policy on water fluoridation programs.

In the U.S., fluoridation is not required by the U.S. Environmental Protection Agency (EPA), which is prohibited by the Safe Drinking Water Act from requiring the addition of any substance to drinking water for preventive health care purposes. The Centers for Disease Control and Prevention (CDC), which is one of the major operating components of HHS, provides recommendations about the optimal levels of fluoride in drinking water. A large number of studies have reported that adequate fluoride intakes can reduce the risk of dental decays (Iheozor-Ejiofor et al., 2015; Slade et al., 2018), but more and more studies showed that low level of fluoride exposure was also related with some adverse effects, such as neurotoxic to children (Agalakova and Nadei, 2020; Bai et al., 2020; Bashash et al., 2017; Malin et al., 2019a,b). They showed that low level of fluoride exposure was related with decreased IQ scores in children (Agalakova and Nadei, 2020). Another study also reported fluoride from chronic systemic exposure accumulates highly in the pineal gland, which might contribute to changes in sleep cycle regulation and sleep behaviors (Malin et al., 2019a). Dental fluorosis was the most common adverse effect caused by excessive fluoride exposure, but the dose-effect relationship between low level of fluoride and dental fluorosis were still unclear. Therefore, our study aimed to examine whether the recommend fluoride exposure (measured in blood plasma and household tap water) was still associated with dental fluorosis. This study is helpful to understanding the adverse effects of fluoride exposure and balancing the benefits with any potential risks.

2. Materials and methods

2.1. Participants

This study utilized data from the National Health and Nutrition Examination Survey (NHANES) collected from 2015 to 2016, which included both dental fluorosis clinical assessment and fluoride biomonitoring data. The NHANES is conducted biennially to collect the nationally representative sample by the Centers for Disease Control and Prevention and designed to assess health and nutrition status of people of all ages living in the U.S. Details of the NHANES research procedures are available on the NHANES website (Centers for Disease Control and Prevention, 2020). In the use of the data, we have completely followed the "data use restrictions" (Centers for Disease Control and Prevention, 2021) and ensured the data only used for statistical analysis or reporting purposes.

Dental fluorosis clinical assessment was assessed among 3478 participants aged 6–29 years. Plasma fluoride concentrations were measured among 2547 participants aged 6–19 years and tap water fluoride concentrations were measured among 4070 participants aged 0–19 years. Our analysis included children and adolescents aged 6–19 years because these participants had both fluoride measurements and dental fluorosis assessments. Our sample included participants who had fluoride measurements, dental fluorosis assessment and complete data for all covariates and outcomes. There were 2098 participants who met inclusion criteria for analyses. Of those, 1808 participants had plasma fluoride levels and 2071 participants had water fluoride levels. Participant selection was depicted in Fig. S1. Supplemental Table S1 compared demographic characteristics of the current overall study sample (n = 2098) and all participants ages 6–19 over the same years (NHANES 2015–2016).

2.2. Dental fluorosis assessment

The dental fluorosis clinical assessment was conducted at the NHANES mobile examination center (MEC) by dental examiners, who were dentists (D.D.S. or D.M.D.) licensed in at least one U.S. state. Each tooth was scored according to the Dean's Fluorosis Index (DFI) and assigned one of the DFI disease severity categories, based on the area of the tooth surface with visible fluorosis and presence of pitting (NHANES Dental Examiners Procedures Manual, 2016). Six categories were used for tooth assessment: normal (translucent, smooth, glossy, pale creamy white, DFI = 0), questionable (slight aberrations, a few white spots, DFI = 0.5), very mild fluorosis (less than 25% of tooth has small, white areas, DFI = 1), mild fluorosis (between 25% and 50% of the tooth has white areas, DFI = 2), moderate fluorosis (50% or more of the tooth with all surfaces involved, with or without brown stains, DFI = 3), or severe fluorosis (all enamel is involved and has discrete or confluent pitting, DFI = 4) (NHANES Dental Examiners Procedures Manual, 2016). Missing teeth, deciduous (primary) teeth, permanent teeth not fully erupted, and teeth in which more than one-half of the visible surface area was obscured by a restoration, caries, or orthodontic appliance were not assessed. A tooth having a non-fluoride opacity was assessed as non-fluoride opacity. The basis for classifying a person's fluorosis status was the categorization of the two most affected teeth. The lesser affected tooth was to be used to identify the person's status if the two most affected teeth were not equally affected (NHANES Dental Examiners Procedures Manual, 2016).

2.3. Plasma fluoride measures

Plasma fluoride levels were influenced by many factors, including total fluoride intake, type of intake, renal function, rate of metabolism, etc. Fluoride concentrations were measured in blood plasma samples (Centers for Disease Control and Prevention, 2017b). Plasma samples were processed, stored, and shipped to the College of Dental Medicine, Georgia Regents University, Augusta, GA for analysis. The ion-specific electrode and hexamethyldisiloxane (HMDS) method was used to measure the plasma fluoride concentrations. Plasma fluoride was measured in duplicate using the same sample and the average of two results was employed. The lower limit of detection (LLOD) for plasma fluoride was 0.25 nmol. Approximately 68.76% (1475/2145) of detected participants in NHNES 2015–2016, had values at or above the LLOD for plasma fluoride. For analytes with analytic results below LLOD, an imputed fill value (0.18), which was the LLOD divided by the square root of 2, was assigned in the analyte results field.

2.4. Water fluoride measures

Fluoride concentrations in water samples were measured electrometrically using the ion-specific electrode (Centers for Disease Control and Prevention, 2017a). Water samples are processed, stored, and shipped to the College of Dental Medicine, Georgia Regents University, Augusta, GA for analysis. Water fluoride was measured in duplicate using the same sample and the average of two results was employed. The lower limit of detection (LLOD) for water fluoride was 0.1 mg/L. Approximately 87.66% (3495/3987) of detected participants in NHNES 2015–2016, had values at or above the LLOD for water fluoride. For analytes with analytic results below LLOD, an imputed fill value (0.07), which was the LLOD divided by the square root of 2, was assigned in the analyte results field.

2.5. Covariates

Covariates were determined according to the prior empirical evidence associated with fluoride exposure and dental fluorosis. They included: age, gender, body mass index, race/ethnicity, the ratio of family income to poverty, and season of sample collection. Questionnaires were used to collect demographic of age (yrs.), sex (male, female), race/ethnicity (Mexican American, other Hispanic, non-Hispanic White, non-Hispanic Black, non-Hispanic Asian, other race), six-month time period when surveyed (November 1 through April 30, May 1 through October 31) and the ratio of family income to poverty. BMI and BMI categories (underweight, normal weight, overweight, and obese) were collected from body measure data.

2.6. Statistical analyses

Means and proportions were calculated for descriptive analyses of demographic variables as well as fluoride exposure and dental fluorosis measures. A Pearson correlation examined the relationship between logarithm (base 10)-transformed plasma and water fluoride concentrations. Dental fluorosis was identified according to DFI score, which was defined no fluorosis (DFI $\leq$ 0.5) and fluorosis (DFI $\geq$ 1). To examine the relationship between water fluoride exposure and dental fluorosis, water fluoride (mg/L) levels was transformed into a 4-category variable, which was: 0.00–0.30 (0 = reference level), 0.31–0.50 (1 = level 1), 0.51–0.70 (2 = level 2), and > 0.70 (3 = level 3). To examine the relationship between plasma fluoride exposure and dental fluorosis, plasma fluoride (μmol/L) levels was transformed into a 4-category variable, which was: 0.00–0.30 (0 = reference level), 0.31–0.40 (1 = level 1), 0.41–0.50 (2 = level 2), and > 0.50 (3 = level 3). Binary logistic regression analyses were used to determine the association between fluoride exposure and the occurrence of dental fluorosis, controlling for age, sex, race/ethnicity, BMI categories, the ratio of family income to poverty and six-month time period when surveyed. Data analysis was conducted with r software (R version 4.0.2). The two-sided *p* values < 0.05 were statistically significant.

3. Results

3.1. Demographic characteristics

Demographic characteristics were presented in Table 1. Table S1 compared demographics between current study participants and all participants aged 6–19 years in NHANES 2015–2016. The number of overall group was 2098 with an average age of 12.19 years, including 1054 boys and 1044 girls. Among the 2098 participants, 1808 subjects had plasma fluoride concentrations and 2071 had water fluoride concentrations. The proportions of subjects in variables including age categories, sex, BMI categories, race, six-month time period when surveyed, were similar across overall group, plasma fluoride sample group, and water fluoride sample group.

3.2. Fluoride levels

Descriptive statistics for water fluoride levels and plasma fluoride levels were presented in Table 2. Geometric mean of household tap water fluoride concentration was 0.33 mg/L, which was below the U.S. Public Health Service recommended concentration of 0.7 mg/L (U.S. Department of Health and Human Services Federal Panel on Community Water Fluoridation, 2015). However, values between the 75th and 95th percentiles were above this level ranging from 0.71 to 1.02 mg/L. The water fluoride concentrations in males were comparable with those in females, but fluoride levels in plasma in males were higher than those in females (Table 2, Fig. S2). Both the water and plasma fluoride levels in children were higher than those in adolescents (Table 2, Fig. S3). Fluoride concentrations in plasma and tap water were light positively correlated (r = 0.41, p < 0.001), which presented in Fig. 1. The correlation patterns in subgroups males and females were similar (Fig. S4).

Table 1
Demographic characteristics of selected samples in NHANES 2015–2016.

Demographic characteristic	Overall sample n = 2098	Plasma fluoride sample n = 1808	Water fluoride sample n = 2071
Age (yrs.); mean (SD)	12.19 (3.77)	12.37 (3.78)	12.18 (3.77)
Age categories; N (%)			
Children (6–11 yrs.)	995 (47.43%)	819 (45.30%)	985 (47.56%)
Adolescents (12–19 yrs.)	1103 (52.57%)	989 (54.70%)	1086 (52.44%)
Sex; N (%)			
Male	1054 (50.24%)	917 (50.72%)	1038 (50.12%)
Female	1044 (49.76%)	891 (49.28%)	1033 (49.88%)
BMI; mean (SD)	21.88 (6.02)	22.12 (6.13)	21.88 (6.02)
BMI Categories; N (%):			
Underweight	57 (2.72%)	44 (2.43%)	56 (2.70%)
Normal Weight	1203 (57.34%)	1029 (56.91%)	1186 (57.27%)
Overweight	374 (17.83%)	328 (18.14%)	369 (17.82%)
Obese	464 (22.12%)	407 (22.51%)	460 (22.21%)
Race/ethnicity			
Mexican American; N (%)	456 (21.73%)	417 (23.1%)	451 (21.78%)
Other Hispanic	254 (12.11%)	232 (12.8%)	250 (12.07%)
Non-Hispanic White	612 (29.17%)	519 (28.7%)	601 (29.02%)
Non-Hispanic Black	461 (21.97%)	376 (20.8%)	457 (22.07%)
Non-Hispanic Asian	181 (8.63%)	158 (8.7%)	179 (8.64%)
Other Race-Including Multi-Racial	134 (6.39%)	106 (5.9%)	133 (6.42%)
Ratio of family income to poverty; mean (SD)	2.06 (1.49)	2.03 (1.48)	2.05 (1.49)
Six month time period when surveyed			
November 1 through April 30	984 (46.90%)	851 (47.1%)	972 (46.93%)
May 1 through October 31	1114 (53.10%)	957 (52.9%)	1099 (53.07%)

3.3. Dental fluorosis

The proportion of dental fluorosis severity by different fluoride levels in drinking water and plasma was presented in Tables 3 and 4. Generally, the proportion of participants had normal teeth was relatively low, which was just 13%. Compared with the lowest fluoride level group, severity of fluorosis increased with higher exposure to fluoride, although there were a few exceptions. For example, those exposed to > 0.70 mg/L of water fluoride had less severe fluorosis than those exposed to 0.00–0.30 mg/L (Table 4), which might just because the number of participants with severe fluorosis was too less.

3.4. Regression analysis between fluoride levels and dental fluorosis

Regression results for fluoride levels and fluorosis were presented in Table 5 and adjusted variables in the regression were presented in Tables S4 and S5. Binary logistic regression adjusted for covariates showed that higher water fluoride concentrations (0.31–0.50, 0.51–0.70, > 0.70 compared 0.00–0.30) were associated with higher odds of dental fluorosis (OR = 1.48, 95% CI: 1.13–1.96, p = 0.005; OR = 1.92, 95% CI: 1.44–2.58, p < 0.001, and OR = 2.30, 95% CI: 1.75–3.07, p < 0.001, respectively). The pattern of regression between plasma fluoride and dental fluorosis was similar, which showed the higher plasma fluoride concentrations (0.31–0.40, 0.41–0.50, > 0.50 compared 0.00–0.30) were associated with higher odds of dental fluorosis (OR = 1.49, 95% CI: 1.14–1.96, p = 0.004; OR = 1.61, 95% CI: 1.15–2.29, p = 0.007, and OR = 1.64, 95% CI: 1.18–2.28, p = 0.003, respectively). We also further explored regression analysis for fluoride levels and

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Table 2
Descriptive statistics of fluoride exposure levels.

Measure	Number	Arithmetic mean (standard deviation)	Geometric mean	Median	5th percentile	25th percentile	75th percentile	95th percentile
Water fluoride (mg/L)								
All	2071	0.46 (0.40)	0.33	0.44	0.07	0.16	0.70	1.02
Male	1038	0.48 (0.41)	0.33	0.44	0.07	0.16	0.70	1.04
Female	1033	0.47 (0.38)	0.33	0.44	0.07	0.17	0.69	1.00
Children	985	0.52(0.44)	0.36	0.47	0.07	0.18	0.72	1.12
Adolescents	1086	0.43(0.35)	0.31	0.37	0.07	0.15	0.68	0.86
Plasma fluoride (μmol/L)								
All	1808	0.35 (0.22)	0.31	0.30	0.18	0.18	0.41	0.71
Male	917	0.36 (0.19)	0.32	0.32	0.18	0.18	0.43	0.70
Female	891	0.34 (0.25)	0.29	0.29	0.18	0.18	0.39	0.71
Children	819	0.38 (0.24)	0.33	0.33	0.18	0.25	0.45	0.73
Adolescents	989	0.32 (0.20)	0.29	0.28	0.18	0.28	0.38	0.66

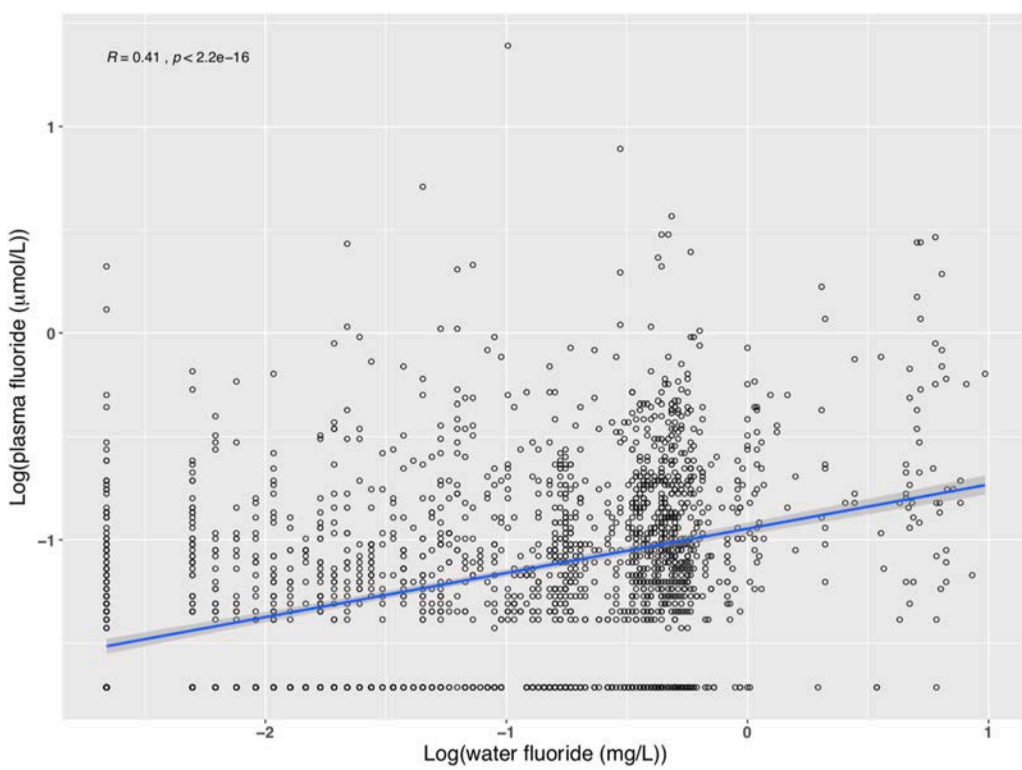

Fig. 1. Pearson's correlations between log 10-transformed water fluoride and plasma fluoride (n = 2107).

Table 3
Number and frequency (percent) of Dean's Index score for children aged 6–19 years in the 3 sample groups.

	Fluorosis severity level					
	Normal (DFI = 0)	Questionable (DFI = 0.5)	Very mild (DFI = 1)	Mild (DFI = 2)	Moderate (DFI = 3)	Severe (DFI = 4)
Overall sample n = 2098	288 (13.73)	348 (16.59)	1223 (58.29)	202 (9.63)	34 (1.62)	3 (0.14)
Water fluoride sample n = 2071	285 (13.76)	346 (16.71)	1206 (58.23)	197 (9.51)	34 (1.64)	3 (0.14)
Plasma fluoride sample n = 1808	243 (13.44)	297 (16.43)	1054 (58.30)	181 (10.01)	30 (1.66)	3 (0.17)

fluorosis by age (Table S2) and gender (Table S3). The patterns of regression results in children (aged 6–11 years) and adolescents (aged 12–19 years) were similar, but the patterns in different gender were changed. Higher plasma fluoride concentrations were associated with higher odds of dental fluorosis in females, but the associations in male groups were almost disappeared (Table S3).

4. Discussion

No fluoride deficiency disease had ever been documented for humans. However, municipal fluoridation was a mid-twentieth century innovation based on the medical hypothesis that consuming low doses of fluoride at the teeth forming years provided protection against dental decays. In this study, we found that the rate of fluoride concentration in water above the recommended level of 0.7 mg/L was 25%, but the prevalence of dental fluorosis was 70% in the NHANES 2015–2016 survey, which was higher than that in the previous 2010–2012 survey of 65% (Neurath et al., 2019). The rate of combined moderate and severe degrees was relatively low with 1.8%. To accurately assess the impact of low levels of fluoride exposure on children and adolescents, we selected

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Table 4
Number and distribution (percent) of fluorosis severity level by different fluoride levels in drinking water and plasma.

	Fluorosis severity level						
	Normal (DFI = 0)	Questionable (DFI = 0.5)	Very Mild (DFI = 1)	Mild (DFI = 2)	Moderate (DFI = 3)	Severe (DFI = 4)	Total
Water fluoride (mg/L)							
0.00–0.30	134 (15.46)	198 (22.84)	483 (55.71)	39 (4.50)	11 (1.27)	2 (0.23)	867 (41.86)
0.31–0.50	68 (19.21)	42 (11.86)	186 (52.54)	51 (14.41)	7 (1.98)	0 (0.00)	354 (17.09)
0.51–0.70	29 (7.83)	61 (16.49)	241 (65.14)	31(8.38)	7 (1.89)	1 (0.27)	370 (17.87)
> 0.70	54 (11.25)	45 (9.38)	296 (61.67)	76 (15.83)	9 (1.88)	0 (0.00)	480 (23.18)
Total	285 (13.76)	346 (16.71)	1206 (58.23)	197 (9.51)	34 (1.64)	3 (0.14)	
Plasma fluoride (μmol/L)							
0.00–0.30	136 (14.66)	179 (19.29)	533 (57.44)	65 (7.00)	14 (1.51)	1 (0.11)	928 (51.33)
0.31–0.40	50 (12.38)	57 (14.11)	247 (61.14)	45 (11.14)	4 (1.00)	1 (0.25)	404 (22.35)
0.41–0.50	28 (12.73)	28 (12.73)	131 (59.55)	27 (12.27)	5 (2.27)	1 (0.45)	220 (12.17)
> 0.50	29 (11.33)	33 (12.89)	143 (55.86)	44 (17.19)	7 (2.73)	0 (0.00)	256 (14.16)
Total	243 (13.44)	297 (16.43)	1054 (58.30)	181 (10.01)	30 (1.66)	3 (0.17)	

Table 5
Associations between water fluoride, plasma fluoride and occurrence of dental fluorosis.[ab]

Fluoride levels	n	Fluorosis[a]	
		Odds ratio (95%CI)	*p*-value
Water fluoride (mg/L)			
0.00–0.30	867	Reference	
0.31–0.50	354	1.48 (1.13–1.96)	0.005**
0.51–0.70	370	1.92 (1.44–2.58)	< 0.001**
> 0.70	480	2.30 (1.75–3.07)	< 0.001**
Plasma fluoride (μmol/L)			
0.00–0.30	928	Reference	
0.31–0.40	404	1.49 (1.14–1.96)	0.004**
0.41–0.50	220	1.61 (1.15–2.29)	0.007**
> 0.50	256	1.64 (1.18–2.28)	0.003**

** $p < 0.01$.
[a] Fluorosis: 0 = No fluorosis (DFI ≤ 0.5); 1 = Fluorosis (DFI ≥ 1).
[b] Regression analyses were adjusted for age, sex, race/ethnicity, body mass index categories, ratio of family income to poverty, and six month time period when surveyed. The regression analysis was carried out separately for water fluoride and plasma fluoride.

both water fluoride and plasma fluoride as external and internal exposure indicators, respectively, and observed that the levels of both were positively associated with the increased risk of dental fluorosis.

People of different ages have different excretion rates of fluoride. For adults, about 50% of absorbed fluoride is retained, and stored in bones and teeth. The other 50% is excreted in urine (VidaZohoori and MarslandDuckworth, 2017). However, in young children, up to 80% of absorbed fluoride is retained because of the more need for the development of the body (Whitford, 1999). In our study, we found that the concentration of plasma fluoride in children was higher than that in adolescents, which could be contributed by the less excretion fluoride in children. But there was a strange result that the water fluoride concentration in children was also higher than that in adolescents, which also contributed the higher level of plasma fluoride in children. As more and more researches had indicated that even low-to-moderate exposure to fluoride was related to a number of adverse health effects in children, such as neurotoxicity (Agalakova and Nadei, 2020; Green et al., 2019; Spencer and Limeback, 2018), changes in sleep cycle (Malin et al., 2019a), alteration of kidney and liver function (Malin et al., 2019b), et al. All these studies implicated that younger children were the suspected population to fluoride. However, all people with different ages were exposed to the same level of fluoride (0.7 mg/L) in drinking water with the water fluoridation system. So, in order to against the adverse effect by fluoride exposure in youngers, children should be provided with alternative sources of drinking water.

In our study, the level of plasma fluoride in males was higher than that in females, when the level of water fluoride was similar with each other. The reasons for this were complex. One possible reason for this might be that males might intake more fluoride from drinking water than females, because males had more weight than females (p = 0.009, showed in Fig. S5) and needed more water. Once absorbed, a portion of fluoride was deposited in the skeleton and most of the remainder was excreted in urine, and to a smaller degree in feces and sweat. Another reason might be a differential excretion rate of fluoride between genders, which might cause different effects. In Green et al. study, they reported that maternal exposure to higher levels of fluoride was associated with lower IQ scores in boys but not significant in girls (Green et al., 2019). Zhou et al. (2019) also reported that gender potentially modified the associations of dental prevalence with relative mitochondrial DNA levels, which showed a stronger inverse relationship between dental fluorosis prevalence and relative mitochondrial DNA levels in boys than in girls.

The main type of drinking water sources in U.S. was being mainly from tap water. Previous analysis of NHANES 2005–2014 showed that 85% of the U.S. children and adolescents on average drunk tap water (Sanders and Slade, 2018). In order to reduce the risk and severity of dental caries of children, the U.S. Public Health Service had recommended the addition of fluoride to drinking tap water since 1945, and 63.4% of the U.S. population had accessed to a fluoridated community water system in 2018 (Centers for Disease Control and Prevention, 2018). So, there was easy to understand that fluoride concentrations in plasma was correlated with that in tap water. But the correlation coefficient was not high. One reason for this might be that only about 60% of fluoride intake was from fluoridated drinking water (U.S. Department of Health and Human Services Federal Panel on Community Water Fluoridation, 2015).

In order to minimize the unwanted effect caused by water fluoridation, we might need to reevaluate the current policy on national water fluoridation program, which is overseed by the Department of Health and Human Services (HHS). Water fluoridation had become a controversial public health intervention these years (Peckham and Awofeso, 2014; Spencer and Limeback, 2018). Fluoridation was not required by EPA, which was prohibited by the Safe Drinking Water Act from requiring the addition of any substance to drinking water for preventive health care purposes. As some areas of the country had high levels of naturally occurring fluoride which could dissolve easily into ground water as it moved through bedrock, EPA had a non-enforceable standard for fluoride of 2.0 mg/L in drinking water to protect children against dental fluorosis (https://www.epa.gov/sdwa/drinking-water-regulations-and-contaminants). As there were numerous studies supported that low level of fluoride consumption had been shown to be associated with decreased prevalence of dental caries (Featherstone, 1999; Iheozor-Ejiofor et al., 2015). In many countries, including the U. S., small amounts of fluoride were added to drinking water, salt, or milk to reduce the incidence of tooth decay. In the U.S., fluoridation of public water supplies was started in 1945. The Centers for Disease Control and Prevention (CDC), which is one of the major operating components of

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HHS, provides recommendations about the optimal levels of fluoride in drinking water. However, a large increase in prevalence of dental fluorosis occurred among recent 30 years, which might relate with the widespread use of fluoride toothpastes and dental treatments (Neurath et al., 2019).

In our study, we observed that even low level of water or plasma fluoride exposure was associated with increased the risk of dental fluorosis. This result was consistent with a European review, which concluded that water fluoridation was a crude and rather ineffective policy to prevent dental caries without a detectable threshold for dental damage (European Commission, 2011). Previous studies reported there was a linear dose-response relationship between the serious of dental fluorosis and fluoride intake, and indicated that dental fluorosis could occur even at very low fluoride intake from water (Butler et al., 1985; Fejerskov et al., 1996). In Peckham's review, the authors concluded that available evidences suggested that fluoride had a potential to cause major adverse human health problems, while having only a modest dental caries prevention effect (Peckham and Awofeso, 2014). Therefore, the intervention of drinking water fluoridation is really needed further research.

Our study also had some limitations. Due to the cross-sectional design, this study had less power in terms of the causal inference of the associations between fluoride exposure and dental fluorosis. Secondly, the assessment of drinking water fluoride and plasma fluoride might not be satisfactory in reflecting exposure level in the years when the permanent teeth of the participants forming (birth to 8 years). As fluoridated water policy have implemented since 1960s, to a certain extent, we hypothesized that a single measurement of blood fluoride and water fluoride reflected the level of long-term exposure. However, since participants were enrolled during or after 2015, the year that the HHS recommended lowering water fluoride concentrations from 0.7 to 1.2 mg/L to 0.7 mg/L to minimize the risk of dental fluorosis (Fluoridation 2015), the water fluoride concentrations during the years when the permanent teeth of the participants forming might be higher than those observed in this study. We also collected the water fluoride data in the year of 2013–2014 from NHANES (Centers for Disease Control and Prevention, 2020), and found that water fluoride concentrations were reduced significantly after lowering the recommended water fluoride concentration (Fig. S6). Thirdly, NHANES did not provide data on participants' length of time at their current residence, thus we could not get their duration of exposure to the water fluoride concentrations measured in this study.

5. Conclusions

Low level of water or plasma fluoride exposure was associated with increased risk of dental fluorosis. The safety of public health approach of drinking water fluoridation for global dental caries reduction are urgently needed further research.

CRediT authorship contribution statement

Haitao Dong: Conceptualization, Writing - original draft. **Xin Yang**: Investigation, Software. **Shixuan Zhang**: Data curation, Resources. **Xueting Wang**: Investigation. **Chunlan Guo**: Software. **Xinyuan Zhang**: Methodology. **Junxiang Ma**: Data curation. **Piye Niu**: Project administration. **Tian Chen**: Software, Writing - review & editing.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supplementary material

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.ecoenv.2021.112439.

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February 7, 2022
White House Environmental Justice Advisory Council

Subject: Public Comment from Good Energy Collective to the White House Environmental Justice Advisory Council in response to the public meeting held January 26-27, 2022

Dear Esteemed Members of the White House Environmental Justice Advisory Council:

Good Energy Collective (GEC) is pleased to submit comments to the White House Environmental Justice Advisory Council (WHEJAC). President Joseph R. Biden established the WHEJAC with the important task of advising the White House Council on Environmental Quality (CEQ) and the newly established Interagency Council on how to address environmental injustice in the United States.[1]

GEC is working to identify policy solutions that address lasting environmental injustices relating to the fuel cycle and siting of nuclear energy. Specifically, we are articulating policies that would advance restorative justice through the clean-up of former nuclear weapons test sites and uranium mines and mills; policies that promote procedural justice by giving communities greater authority and autonomy in decisions about whether and where nuclear technology is operated; and policies that embody distributive justice by ensuring that a more diverse set of communities have access to nuclear energy, where there is interest.

Introduction

Injustice has been a defining characteristic of the United States' legacy with nuclear energy. In the United States, communities of color and Indigenous communities have borne most of the pollution and toxic legacies of the U.S. nuclear weapons test programs and the front end of the fuel cycle for nuclear energy. Meanwhile, whiter and wealthier communities have been more likely to benefit from the clean power, well-paid jobs, and local economic benefit afforded by nuclear energy generation.[2]

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[1] U.S. Government Publishing Office, "Executive Order 14008 of January 27, 2021: Tackling the Climate Crisis at Home and Abroad" (2021), https://www.federalregister.gov/documents/2021/02/01/2021-02177/tackling-the-climate-crisis-at-home-and-abroad.
[2] River Bennett and Alex Gilbert, "Can Nuclear Energy Jobs Power a Just Transition?" Good Energy Collective, 19 January 2022,

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About Good Energy Collective: GEC is a progressive policy research organization focused on building the progressive case for nuclear energy as an essential part of the broader climate change agenda. GEC works with industry, the administration, Congress, and NGOs across the political spectrum to help shape the future of advanced nuclear technologies to contribute toward an environmentally just climate response.

If nuclear energy is to continue to play a part in a sustainable, progressive vision for our domestic energy system, the federal government and the nuclear industry must first work to address the historically inequitable balance between who has experienced the benefits and the detriments, respectively, of nuclear technology. GEC researches how the entire nuclear energy system, from the deployment of new reactors to the front and back end of its fuel cycle, can be more socially and environmentally just going forward. For example, we are exploring ways to:

1.) Secure restorative justice for communities that live in and around former nuclear weapons test sites and uranium mines and mills by increasing funding and federal commitment for clean-ups,
2.) Protect communities going forward by exploring alternative sources of nuclear fuel;
3.) Compensate former uranium workers and their families for contamination due to uranium unsafe mining and milling practices;
4.) Follow consent-based practices for the siting of nuclear plants and nuclear waste that take communities interests and concerns into account at every stage of the stakeholder process—and, critically, allow communities to decide whether these projects move forward in their neighborhoods;
5.) Establish models of community ownership for advanced nuclear projects, whose smaller and factory-fabricated nature makes them more suited to local, rather than solely utility-led, construction and ownership; and
6.) Explore ways that nuclear developers, utilities, or other energy stakeholders can distribute nuclear energy more equitably by allowing more communities (if the communities are interested) to have access to electricity from nuclear.

The WHEJAC can take several steps that could advance environmental justice as relates to nuclear energy. Below, we make three specific recommendations to the WHEJAC and provide additional feedback that we hope will be useful to the council's deliberations.

Recommendations

Communities across the country, predominantly communities of color and Indigenous communities, have waited decades for the cleanup of the yellowcake and other contaminants unleashed from the soil and that uranium companies left unremediated. These communities continue to experience devastating health impacts related to their multigenerational exposure to radon, arsenic, and other toxicants related to the front end of the nuclear fuel cycle.

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https://uploads-ssl.webflow.com/5f05cd440196dc2be1636955/61e864060e5d8dc834dbaaa4_Can%20Nuclear%20Energy%20Jobs%20Power%20a%20Just%20Transition.pdf, 8.

At the January 26-27 WHEJAC meeting, Tina Cordova, co-founder of the Tularosa Basin Downwinders Consortium, talked in her public remarks of the scars that the detonation of a nuclear device in New Mexico's desert caused at what is referred to as the "Trinity" site. These Downwinders experienced high levels of radiation, and nearby communities continue to register higher rates of diseases, including cancer, as well as fear that interacting with their land may harm their health.[3]

Also at the meeting, WHEJAC Co-Chair Richard Moore expressed an interest in the WHEJAC moving to take a closer look at addressing the impacts of "military toxics" throughout the United States, citing repeated feedback he and others have received regarding the environmental racism evident from past military activities.

Recommendation 1: Recommend to the CEQ that the following investments be explicitly included in the definition of what counts as a benefit for disadvantaged communities under Justice40 in the category of Remediation and Reduction of Legacy Pollution[4]: "Restoration of uranium mines, uranium mills, and nuclear weapons test sites."

Recommendation 2: Establish a WHEJAC working group to explore potential recommendations across the full suite of legacy waste issues in the United States, inclusive of uranium mining and uranium milling and military toxics. The working group could in part act to identify the most polluted among the abandoned uranium mines and military testing sites to help establish a list of the top-priority sites for clean-up.

As the WHEJAC has witnessed, the Biden-Harris administration is making an effort to begin weaving considerations of environmental justice into the fabric of operations across all or most federal agencies. Progress is frustratingly slow, which we continue to believe is primarily due to slow-to-change, bureaucratic procedures than to an intentional slow-walking of efforts to improve. Thankfully, progress is getting underway in certain pockets, including the U.S. Department of Energy (DOE) Office of Nuclear Energy, which is actively working to incorporate and uphold environmental justice across its programs and activities and to stand up new programming that is environmental justice-focused.

Recommendation 3: As the WHEJAC members' time allows, consider reaching out to the DOE Office of Nuclear Energy to exchange information and ideas and to hear what the department is doing to embed environmental justice considerations into the agency's work.

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[3] Atomic Heritage Foundation, "Trinity Test - 1945," 18 June, 2014, https://www.atomicheritage.org/history/trinity-test-1945.
[4] U.S. Office of Management and Budget, "Memorandum for the Heads of Departments and Agencies, 20 July 2021, https://www.whitehouse.gov/wp-content/uploads/2021/07/M-21-28.pdf, 6.

Additional Feedback

Climate Resilience Working Group

GEC noted with interest the CEQ presenters' indication that the WHEJAC would be tasked with developing a working group focused, broadly, on improving climate resilience. Improving the climate resilience of nuclear energy will be critical going forward. As is the case for other clean energy technologies, more study is needed into how nuclear energy can be hardened against climate risks that are growing in frequency and intensity, including risks from extreme heat, wildfire, drought, floods, and rising sea levels. Ultimately, nuclear energy may offer some positive attributes of climate resilience, including its smaller land requirements, and the potential of newer modular or microreactors to operate underground. As another example, one nuclear reactor could power all or most of a community that wishes to isolate itself on a microgrid during broader grid disruptions caused by extreme weather events. Mark Chambers of the CEQ discussed this "islanding" capacity as an important area of potential research going forward. The WHEJAC may want to examine nuclear energy as one component of the climate resilience working group.

Feasibility Study for Advanced Nuclear Siting in Puerto Rico

Our organization was disheartened to hear about the concerns raised by WHEJAC Member Ruth Santiago over the process of community engagement followed in the DOE-funded feasibility study into whether advanced nuclear energy could work for Puerto Rico. Any nuclear siting process must firmly uphold procedural justice, which means engaging local communities from the outset of conversations and empowering them to learn about the proposed action and to ask questions. We hope that those involved with the feasibility study will course-correct and address any shortfalls—whether real or perceived—in community engagement.

Staffing at the Council on Environmental Quality

GEC is deeply sympathetic toward the WHEJAC's frustrations with the quantity of dedicated CEQ staff working on implementing the Biden-Harris administration's environmental justice commitments. We support the WHEJAC in continuing to urge CEQ and the administration to increase funding for the CEQ and the number of CEQ staff with a portfolio specific to environmental justice in order to augment the office's competencies and ability to meet the administration's promises to support environmental justice communities.

While Slow, Progress Is Occurring

We have been encouraged by two recent actions that Congress has taken in support of a more just implementation of nuclear energy.

The first of these is the inclusion, in the Infrastructure Investment and Jobs Act enacted Nov. 14, 2021, of Section 40321, which directs the DOE to provide financial and technical support for feasibility studies determining potentially suitable locations for newer kinds of nuclear reactors in communities that are currently disconnected from a regional electric grid.[5] The infrastructure law specifies that these feasibility studies are not to occur until "robust community engagement and outreach for the purpose of identifying levels of interest in isolated communities" is performed. We are heartened that Congress carefully phrased the provision to protect the interests of local communities and ensure proper procedures of engagement are followed in gauging communities' interest in adopting advanced nuclear technology.

In addition, on Jan. 19, 2021, the House Science, Space, and Technology Committee took action to strengthen the United States' weak understanding of nuclear energy's social dimensions. Before reporting to the full House of Representatives a bipartisan bill to invigorate U.S. universities' leadership in nuclear research and development,[6] the committee adopted an important amendment offered by Rep. Jamaal Bowman (D-NY) that would enable an existing DOE program to support nontechnical academic research in the social sciences regarding nuclear energy.[7] The amendment increased authorized funding for this program by $15 million per year and defined nontechnical research in part as that which can increase community participation and confidence in nuclear energy. The House of Representatives, on Feb. 4, 2022, passed this legislation within the America Competes Act, which now awaits conferencing with the Senate.

While these legislative developments mark a positive directional change in Congress and should ultimately enhance the role of communities in nuclear siting, the federal government will need to take many additional steps to uphold environmental justice as relates to nuclear energy.

GEC believes that, if done right, nuclear energy could be an important energy source that, alongside resources like renewables, energy storage, geothermal, and hydropower, could help us reduce our carbon and criteria air pollutant emissions, as nuclear produces energy without operating emissions and takes up a smaller footprint than other clean energy sources. However, the nuclear industry must change the way it does business for nuclear to play a part in our shared vision for our communities, environment, and economy. We will continue to work to articulate policies that would foster a more community-based, progressive approach to each aspect of the nuclear energy fuel cycle and nuclear deployment.

We would be very happy to meet with members of the WHEJAC if that would serve the council's important deliberations, though we also recognize how much time and energy that members are already putting into the activities of the WHEJAC and want to be respectful of the council's time.

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[5] U.S. Congress, "Infrastructure Investment and Jobs Act," PL 117-58, https://www.congress.gov/bill/117th-congress/house-bill/3684/text.

[6] U.S. House of Representatives, "H.R. 4819 - National Nuclear University Research Infrastructure Reinvestment Act of 2021," https://www.congress.gov/bill/117th-congress/house-bill/4819.

[7] U.S. House Committee on Science, Space, and Technology, "Amendment to H.R. 4819 Offered by Mr. Bowman of New York," 18 January, 2022, https://science.house.gov/imo/media/doc/BOWMAN_033_xml.pdf.

We appreciate your consideration of our recommendations and look forward to following the continued work of the WHEJAC.

Sincerely,

Jackie Toth
Deputy Director
Good Energy Collective

City Hall Annex
Three Pond Road
Gloucester, MA 01930

CITY OF GLOUCESTER
Conservation Commission

Date: August 21, 2019
To: Mayor Sefatia Romeo-Theken, Paul Lundberg, President, Gloucester City Council
From: Rob Gulla, Chairman, Gloucester Conservation Commission
CC: Joanne Senos, City Clerk, Adrienne Lennon, Conservation Agent, Jill Cahill, Community Development Director

Re: Conservation Commission Statement Opposing Artificial Fluoridation Policy

Dear Mayor Romeo-Theken and Council President Lundberg,

The Gloucester Conservation Commission opposes the practice of artificial fluoridation of municipal water supplies. As the fluoride ion itself is a poison that can and does adversely affect biological function in plants, people, fish and animals and the bulk of the approximate 5 tons of fluoridation product purchased each year by the city ends up in our environment along with incidental other contaminants contained in that product, our opposition is in keeping with our mission to protect public interests including public and private water supplies, ground water, local flora and fauna and their habitats.

Consequently, the Gloucester Conservation Commission formally advises the Gloucester City Council to initiate the appropriate actions in order to ban fluoridation practice in our city.

Sincerely,

Rob Gulla
Chairman, Gloucester Conservation Commission

Charpentier – Cook – Feener – Gradwohl – Gulla – Jackson – Shaw-Kwasie

Environmental References

WHAT WE KNOW:

1. *We know that fluoride has an adverse impact on the migration, reproduction and mortality of salmon and trout even at 0.5 ppm in fresh water.*
2. *We know that laboratory experiments using zebra fish confirm fluoride in low concentrations such as 0.5 ppm adversely impacts the endocrine system of that animal model.*
3. *We know that approximately the same concentration has an adverse impact on some plants and aquatic life lower on the food chain.*
4. *We know that fluoride accumulates in brains of seabirds where it causes calcification.*
5. *We know that fluoride accumulation in human brains is associated with neurodegenerative conditions.*
6. *We know that industrial fluoride pollution contributes to the fluoride exposure.*
7. *We know that the fluorides added to tap water are fundamentally different compositions than the natural fluoride found in seawater.*
8. *We know that fluoridated water is harmful to many amphibians and reptiles, and consequently result in de-fluoridation of city water at zoos in order to reduce disease and mortality in these animals.*
9. *We know that fluoridated wastewater discharge from 'optimally' fluoridated communities at 0.7 ppm is frequently higher than 0.7 ppm due to a combination of factors.*
10. *We know that only 1% of fluoridation chemicals added to drinking water are consumed by people.*
11. *We know that 100% of fluoridation chemicals are contaminated with other toxins such as aluminum, arsenic, barium, cadmium, lead, etc.*
12. *We know that despite the addition of buffering chemicals to counteract the corrosive nature of fluoridation chemicals, municipal water infrastructure in fluoridated communities has an accelerated deterioration with a net result of more chemicals and toxic metals in waste water.*
13. *We know that fluoride and other toxins accumulate in plants, animals and fish and that there is no known way to reverse accumulation in environmental settings.*
14. *We know that smog results in ocean pollution that bleaches coral via the same type of poisoning that happens to developing teeth known as dental fluorosis. DF mottles the teeth of over half of U.S. teens. Our fluoridation chemicals are harvested from air pollution control systems in Shanghai, China - packaged air pollution.*
15. *We know that fluoride accumulation robs soils of nutrients by destroying microscopic life.*
16. *We know that fluoride is an enzyme poison and an EPA contaminant identified as a developmental neurotoxicant.*
    - *Until the mid 1980s, 2.4 ppm was the EPA maximum contaminant level goal (MCLG) for fluoride in drinking water. The actionable maximum contaminant level (MCL) was set at the same concentration. The MCL/MCLG was increased to 4 ppm at the behest of lobbyists for polluters and municipalities who could not afford to take steps to de-fluoridate their contaminated water. The 4 ppm MCL/MCLG was objected to by EPA scientists, but was given a provisional pass by the 1993 NRC pending research that still hasn't been done. The 2006 NRC advised that 4 ppm was not protective of human health and that the gaps in knowledge make it impossible to determine a scientifically defensible reference dose for fluoride in drinking water, but there has been no EPA action to reduce the MCL/MCLG determined as harmful.*
    - *The Safe Drinking Water Act calls for reviews of MCLGs every six years to guarantee safety.*

WHAT WE DO NOT YET KNOW:

A. *We do not yet know the long term impact of fluoridated wastewater on fresh water plants and fisheries.*
B. *We do not yet know the long term impact of fluoridated wastewater on salt water plants and fisheries.*
C. *We do not yet know the full impact of fluoridated waste water on insects, seabirds or plankton.*
D. *We do not yet know the impact of overall fluoride pollution on ocean mammals' health, migration or reproduction.*
E. *We will likely never know how to clean the fluoride out of wetlands and waters, fresh and salt, contaminated with fluoridation chemicals.*

Mankind has lived on the fertile lands and fished the bountiful waters of Massachusetts Bay for at least a thousand years. Fluoridation policy is poisoning both our land and water. Think long term.

Check for updates

A Benchmark Dose Analysis for Maternal Pregnancy Urine-Fluoride and IQ in Children

Philippe Grandjean,[1,2,*] Howard Hu,[3] Christine Till,[4] Rivka Green,[4] Morteza Bashash,[3] David Flora,[4] Martha Maria Tellez-Rojo,[5] Peter X.K. Song,[6] Bruce Lanphear,[7] and Esben Budtz-Jørgensen[8]

As a guide to establishing a safe exposure level for fluoride exposure in pregnancy, we applied benchmark dose modeling to data from two prospective birth cohort studies. We included mother–child pairs from the Early Life Exposures in Mexico to Environmental Toxicants (ELEMENT) cohort in Mexico and the Maternal-Infant Research on Environmental Chemicals (MIREC) cohort in Canada. Maternal urinary fluoride concentrations (U-F, in mg/L, creatinine-adjusted) were measured in urine samples obtained during pregnancy. Children were assessed for intelligence quotient (IQ) at age 4 ($n = 211$) and between six and 12 years ($n = 287$) in the ELEMENT cohort, and three to four years ($n = 407$) in the MIREC cohort. We calculated covariate-adjusted regression coefficients and their standard errors to assess the association of maternal U-F concentrations with children's IQ measures. Assuming a benchmark response of 1 IQ point, we derived benchmark concentrations (BMCs) and benchmark concentration levels (BMCLs). No deviation from linearity was detected in the dose–response relationships, but boys showed lower BMC values than girls. Using a linear slope for the joint cohort data, the BMC for maternal U-F associated with a 1-point decrease in IQ scores was 0.31 mg/L (BMCL, 0.19 mg/L) for the youngest boys and girls in the two cohorts, and 0.33 mg/L (BMCL, 0.20 mg/L) for the MIREC cohort and the older ELEMENT children. Thus, the joint data show a BMCL in terms of the adjusted U-F concentrations in the pregnant women of approximately 0.2 mg/L. These results can be used to guide decisions on preventing excess fluoride exposure in pregnant women.

KEY WORDS: Benchmark dose; cognitive deficits; fluoride; neurotoxicity; pregnancy; prenatal exposure

1. INTRODUCTION

The Environmental Protection Agency's maximum contaminant level goal (MCLG) of 4.0 mg/L for fluoride in drinking water was first set in 1985 to protect against chronic fluoride toxicity in the

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form of crippling skeletal fluorosis (U.S. Environmental Protection Agency, 1985). In 2006, the U.S. National Research Council (NRC) concluded that fluoride may adversely affect the brain (National Research Council, 2006). Since then, a substantial number of cross-sectional studies, mostly in communities with chronic fluoride exposure, have shown lower cognitive performance in children growing up in areas with higher fluoride concentrations in drinking water, as summarized in meta-analyses (Choi et al., 2015; Duan, Jiao, Chen, & Wang, 2018; Tang, Du, Ma, Jiang, & Zhou, 2008). Support for fluoride neurotoxicity has also emerged from experimental studies (Bartos et al., 2018; Mullenix, Denbesten, Schunior, & Kernan, 1995; National Toxicology Program, 2020). Despite the existence of recent prospective birth cohort studies (Bashash et al., 2017; Green et al., 2019; Valdez Jimenez et al., 2017), no meta-analysis has so far focused on prenatal fluoride exposure.

Fluoride is found in many minerals, in soil and thus also in groundwater (National Research Council, 2006). Since the mid 1940s, fluoride has been added to many drinking water supplies in order to prevent tooth decay (U.S. Environmental Protection Agency, 1985). Community water fluoridation is practiced in the United States, Canada, and several other countries, whereas some, like Mexico, add fluoride to table salt. Fluoridated water accounts for about 40–70% of daily fluoride intake in adolescents and adults living in these communities (U.S. Environmental Protection Agency, 2010). The fluoride concentration in drinking water roughly equals the fluoride concentration in urine (National Research Council, 2006), as also recently shown in the Canadian cohort of pregnant women (Till et al., 2018). In addition to fluoridation, some types of tea, such as black tea, constitute an additional source of exposure (Krishnankutty et al., 2021; Rodríguez et al., 2020; Waugh, Godfrey, Limeback, & Potter, 2017).

Fluoride is readily distributed throughout the body, with bones and teeth as storage depots. During pregnancy, fluoride crosses the placenta and reaches the fetus (National Research Council, 2006; World Health Organization, 2006). As fluoride is rapidly eliminated via urine, the adjusted urine-fluoride (U-F) concentration mainly represents recent absorption (Ekstrand & Ehrnebo, 1983; World Health Organization, 2006). Pregnant women may show lower U-F concentrations than nonpregnant controls, perhaps due to fetal uptake and storage in hard tissues (Opydo-Symaczek & Borysewicz-Lewicka, 2005).

For the purpose of identifying safe exposure levels, regulatory agencies routinely use benchmark dose (BMD) calculations (European Food Safety Authority, 2009; U.S. Environmental Protection Agency, 2012). As long recognized (National Research Council, 1989), fluoride is not an essential nutrient, and dose-dependent toxicity can therefore be considered monotonic. As with lead (Budtz-Jørgensen, Bellinger, Lanphear, & Grandjean, 2013), BMD results can be generated from regression coefficients and their standard errors for the association between maternal U-F concentrations and the child's intelligence quotient IQ score (Grandjean, 2019). The BMD is the dose leading to a specific change (denoted BMR) in the response (in this case, an IQ loss), compared with unexposed children. A decrease of 1 IQ point is an appropriate BMR, as specified by the European Food Safety Authority and also recognized by the U.S. EPA (Budtz-Jørgensen et al., 2013; European Food Safety Authority, 2010; Gould, 2009; Reuben et al., 2017). The present study uses data from two prospective birth cohort studies (Bashash et al., 2017; Green et al., 2019) to calculate the benchmark concentration (BMCs) of U-F associated with a 1-point decrement in Full Scale IQ (FSIQ).

2. METHODOLOGY

2.1. Study Cohorts

In the Early Life Exposures in Mexico to Environmental Toxicants (ELEMENT) project, mother–child pairs were successively enrolled in longitudinal birth cohort studies from the same three hospitals in Mexico City which serve low to moderate income populations. A full description of the cohorts and associated methods is provided in a recent "Cohort Profile" article (Perng et al., 2019). Urinary samples were collected from pregnant women between 1997 and 1999 (Cohort 2A, n = 327) and between 2001 and 2003 (Cohort 3 with calcium intervention and placebo arms, $n = 670$). Cohort 2A was designed as an observational birth cohort of lead toxicodynamics during pregnancy, while Cohort 3 was designed as a randomized double-blind placebo-controlled trial of calcium supplements. Women were included in the current study if they had at least one biobanked urine sample for fluoride analysis, a urinary creatinine concentration, complete data of adjusted covariates, and their child underwent cognitive testing at age four years ($n = 287$) and/or between ages 6 and 12 years ($n = 211$). Of the 287 participants with data on general cognitive index (GCI) outcomes and other variables, 110 were from Cohort 2A, 93 were from the Cohort 3 calcium intervention arm, and 84 were from the Cohort 3 placebo arm. Among participant in the GCI outcome, U-F data were available for all three trimesters ($n = 25$), two trimesters ($n = 121$), or one trimester ($n = 141$). Of the 211 participants with data on IQ outcomes, 78 were recruited from Cohort 2A, 75 from the Cohort 3 calcium intervention arm, and 58 from the placebo arm; U-F data for IQ outcome were available for all three trimesters ($n = 10$), two trimesters ($n = 82$), or one trimester ($n = 119$).

In the Maternal–Infant Research on Environmental Chemicals (MIREC) program, 2,001 pregnant women were recruited between 2008 and 2011 from 10 cities across Canada. Women were recruited from prenatal clinics if they were at least 18 years old, less than 14 weeks of gestation, and spoke English or French. Exclusion criteria included fetal abnormalities, medical complications, and illicit drug use during pregnancy; further details have been previously described (Arbuckle et al., 2013). A subset of children ($n = 601$) in the MIREC Study was evaluated for the developmental phase of the study (MIREC-Child Development Plus) at three–four years of age from six of the 10 cities included in the original cohort, half of which were fluoridated. Of the 601 children who completed the neurodevelopmental testing in entirety, 526 (87.5%) mother–child pairs had all three U-F samples; of these, 512 (85.2%) had specific gravity measures, while 407 (67.7%) had creatinine data, as well as complete covariate data; 75 (12.5%) women were missing one or more trimester U-F samples, and 14 women (2.3%) were missing one or more covariates.

2.2. Exposure Assessment

All urine samples from the two studies were analyzed by the same laboratory at the Indiana University School of Dentistry using a modification of the hexamethyldisiloxane (Sigma Chemical Co., USA) microdiffusion method with the ion-selective electrode (Martinez-Mier et al., 2011).

In the ELEMENT study, spot (second morning void) urine samples were collected during the first trimester ($M \pm SD$: 13.7 ± 3.5 weeks for Cohort 2A and 13.6 ± 2.1 weeks for Cohort 3), second trimester (24.4 ± 2.9 weeks for Cohort 2A and 25.1 ± 2.3 weeks for Cohort 3), and third trimester (35.0 ± 1.8 weeks for Cohort 2A and 33.9 ± 2.2 weeks for Cohort 3). The samples were collected into fluoride-free containers and immediately frozen at the field site and shipped and stored at −20 °C at the Harvard School of Public Health, and then at −80 °C at the University of Michigan School of Public Health. To account for variations in urinary dilution at time of measurement, the maternal U-F concentration was adjusted for urinary creatinine, as previously described (Thomas et al., 2016). An average of all available creatinine-adjusted U-F concentrations during pregnancy (up to a maximum of three samples) was computed and used as the exposure parameter.

In the MIREC study, urine spot samples were collected at each trimester, that is, first trimester at 11.6 ± 1.6 ($M \pm SD$) weeks of gestation, second trimester at 19.1 ± 2.4 weeks, and third trimester at 33.1 ± 1.5 weeks. Maternal U-F concentrations at each trimester were adjusted for both creatinine and specific gravity, as described previously (Till et al., 2020). For this joint analysis, however, we elected to use the U-F concentrations adjusted for creatinine to keep the urine dilution factor consistent with the adjustment procedure in ELEMENT. For each woman, the average maternal U-F concentration was derived only if a valid U-F value was available for each trimester.

2.3. Assessment of Intelligence

The ELEMENT study (Bashash et al., 2017) used the McCarthy Scales of Children's Abilities (MSCA) Spanish version to measure cognitive abilities at age four years and derive a GCI as a standardized composite score. The MSCA was administered by trained psychometrists or psychologists who were supervised by an experienced clinical child psychologist. For children aged six–12 years, a Spanish-version of the Wechsler Abbreviated Scale of Intelligence (WASI) was administered to derive FSIQ as a measure of global intellectual functioning. In the MIREC study, children's intellectual abilities (Green et al., 2019) were assessed at age three–four years using the FSIQ from the Wechsler Preschool and Primary Scale of Intelligence, Third Edition (WPPSI-III). A trained research assistant who was supervised by a psychologist administered the WPPSI-III in either English or French. In both studies, examiners were blinded to the children's fluoride exposure. All raw scores were standardized for age.

The GCI shows concurrent validity with intelligence tests, including the Stanford–Binet IQ ($r = 0.81$) and FSIQ ($r = 0.71$) from the Wechsler Preschool and Primary Scale of Intelligence (WPPSI) (Kaplan & Sacuzzo, 2010). Similarly, the FSIQ of the WASI (ELEMENT cohort) and WISC-III (MIREC cohort) is strong ($r = 0.81$) (Wechsler, 1991). The high covariance between the various measures of intellectual ability provides justification for pooling IQ scores across the two cohorts.

2.4. Covariate Adjustment

For the ELEMENT study, data were collected from each subject by questionnaire on relevant parameters, gestational age was estimated by registered nurses, and maternal IQ was estimated using subtests of the Wechsler scale standardized for Mexican adults. Covariates included gestational age (weeks), birth weight, sex, age at outcome measurement, and the following maternal characteristics: parity (being first child), smoking history (ever smoked vs. nonsmoker), marital status (married vs. other), age at delivery, IQ, education (years of education), and subcohort (Cohort 2A, Cohort 3 calcium intervention or placebo).

The MIREC study selected similar covariates from a set of established predictors of fluoride metabolism and cognitive development, including sex, city of residence, HOME score, maternal education (dichotomized as bachelor's degree or higher: yes/no), and maternal race/ethnicity (dichotomized as white: yes/no). Covariates included in the original studies (Bashash et al., 2017; Green et al., 2019) were retained in the statistical calculations in the present study. Due to a growing body of epidemiologic studies showing sex-specific effects associated with neurotoxic exposures (Levin, Dow-Edwards, & Patisaul, 2021), including fluoride (Green et al., 2019; Green, Rubenstein, Popoli, Capulong, & Till, 2020), interactions between sex and U-F exposure were examined.

2.5. Benchmark Concentration Calculations

The BMC is the U-F concentration that reduces the outcome by a prespecified level (known as the benchmark response, BMR) compared to an unexposed control with the same covariate profile (Budtz-Jørgensen, Keiding, & Grandjean, 2001; Crump, 1995). We based the benchmark calculations on regression models with p covariates in the following form:

$$\mathrm{IQ} = \alpha_0 + \alpha_1 \times \mathrm{covariate}_1 + \cdots + \alpha_\mathrm{p} \times \mathrm{covariate}_\mathrm{p} + f(c) + \varepsilon$$

where c is the urine-fluoride concentration and f is the concentration–response function, and ε is a normally distributed error term with a mean of 0 (and a variance of σ^2). To assess the linearity of the concentration-response relationship, several models were considered. In addition to the standard linear model, where $f(c) = \beta c$, we estimated a squared effect, where $f(c) = \beta c^2$, and two piecewise-linear models (or broken-stick) with breakpoints at 0.5 and 0.75 mg/L. Piecewise-linear models are useful in benchmark calculations because the slope of the concentration-response function is allowed to change linearity at the breakpoint, and in such models, benchmark calculations are less sensitive to exposure-associated effects occurring only at high concentration levels. Furthermore, to allow for the possibility of different exposure effects in boys and girls, each concentration-response model was also fitted with the inclusion of an interaction with sex.

Models were fitted separately in the two cohorts yielding analyses that were similar to those presented in the original publications (Bashash et al., 2017; Green et al., 2019) based on the original raw data and with the covariate adjustments as originally justified. Sensitivity analyses were carried out using the MIREC specific gravity-adjusted U-F values joint with the ELEMENT creatinine-adjusted U-F values as well. The Mexico study controlled for maternal bone lead stores (the primary source of prenatal lead exposure in this cohort) and blood-mercury during pregnancy, although the sample size was reduced by about one-third; the effect estimates for fluoride on child IQ increased and remained statistically significant ($p < 0.01$) (Bashash et al., 2017). Similarly, controlling for lead, mercury, perfluorinated compound, arsenic, and manganese in the MIREC study did not result in any appreciable change of the U-F estimates (Green et al., 2019). Thus, these other neurotoxicants were not included as covariates in the present calculations. Using the regression coefficients, we first calculated BMC results for each cohort and then derived joint BMCs by combining regression coefficients from the two cohorts.

Given that the BMC reduces the outcome by the BMR, a smaller BMR will result in lower BMC and benchmark concentration level (BMCL) results. For the child IQ as the outcome variable, the BMR is

1 IQ point. In our regression model, the IQ difference between unexposed subjects and subjects at the BMC is given by $f(0) - f(\mathrm{BMC})$, and therefore the BMC satisfies the equation $f(0) - f(\mathrm{BMC}) = \mathrm{BMR}$. We use concentration-response functions with $f(0) = 0$, and therefore the BMC is given by

$$\mathrm{BMC} = f^{-1}(-\mathrm{BMR})$$

In a regression model with a linear concentration-response function [$f(c) = \beta c$], we get $\mathrm{BMC} = -\mathrm{BMR}/\beta$. If the estimated concentration–response is increasing (indicating a beneficial effect), the BMC is not defined, and the BMC is then indicated by ∞.

The main result of the BMC analysis is the BMCL, which is defined as a lower one-sided 95% confidence limit of the BMC (Crump, 1995). In the linear model,

$$\mathrm{BMCL} = -\mathrm{BMR}/\beta_{\mathrm{lower}}$$

where β_{lower} is the one-sided lower 95% confidence limit for β (Budtz-Jørgensen et al., 2013). In the other models considered, we calculated the BMCL by first identifying a lower confidence limit for $f(c)$ and then finding the concentration (c) where confidence limit is equal to −BMR.

Finally, we derived two sets of joint benchmark concentrations: The MIREC results (FSIQ score) were combined with ELEMENT outcomes using either GCI or FSIQ scores for all subjects where the creatinine-adjusted U-F was available. Joint benchmark concentration results were obtained under the hypothesis that the concentration-response functions were identical in the two studies. Under this hypothesis, the concentration-response function [$f(c)$] was estimated by combining the regression coefficients describing $f(c)$. Again, using the linear model as an example, we estimated the joint regression coefficient by weighing together cohort-specific coefficients. Here we used optimal weights proportional to the inverse of the squared standard error. In a Wald test, we tested whether the exposure effects in the two cohorts were equal. We calculated sex-dependent BMC results from regression models that included interaction terms between sex and $f(c)$. The fit of the regression models was compared by twice the negative log-likelihood [$-2\ \log L$] as supplemented by the Akaike Information Criterion (AIC); the latter is provided in the tables. For both measures, a lower value indicates a better fit, but AIC-based differences below four are not considered important. For sex-dependent results, the AIC value for both boys and girls represents the fit of a model that includes an interaction between sex and exposure. As the linear model is nested in the piecewise linear model, the fit of these two models can be directly compared. Thus, we calculated the p-value for the hypothesis that the concentration-response is linear in a test where the alternative was the piecewise linear model. Here a low p-value indicates that the linear model has a poorer fit. As specific-gravity adjusted U-F values were available for an additional 105 MIREC subjects, we carried out sensitivity analyses using these data jointly with ELEMENT's creatinine-adjusted data.

3. RESULTS

Table 1 shows the regression coefficients obtained from the two outcomes (GCI and IQ score) in the ELEMENT study and the IQ score in the MIREC study. As previously reported (Bashash et al., 2017; Green et al., 2019), maternal U-F exposure predicts significantly lower IQ scores in boys and girls in the ELEMENT cohort, while it does not show a statistically significant association for boys and girls combined in the MIREC cohort. However, for the linear association, the difference between the two studies is not statistically significant and the combined data show highly significant U-F regression coefficients (Table 1). A sensitivity analysis using the larger number of observations with specific-gravity adjusted U-F did not show significant differences between the two cohort studies and yielded joint U-F effects that were significant.

Table 2 shows the BMC results obtained from the regression coefficients for each sex and for both sexes. The BMC and BMCL are presented for the MIREC study, the ELEMENT (GCI and IQ) study, and combined across the two cohorts. The AIC results did not reveal any important differences between the model fits, except that the linear slope appeared superior to the squared for the joint results that included the Mexican GCI data. For the linear models, the joint BMCL in terms of U-F (creatinine-adjusted) is approximately equal for the MIREC-ELEMENT IQ model (0.20 mg/L) and MIREC-ELEMENT GCI model (0.19 mg/L). Similarly, for the squared models, the joint BMCL in terms of U-F is approximately equal for the MIREC-ELEMENT IQ model (0.77 mg/L) and MIREC-ELEMENT GCI model (0.81 mg/L). When using the larger number of specific gravity-adjusted U-F results from the MIREC cohort, the joint analysis with the

Table 1. Regression Coefficients Adjusted for Confounders for the Change in the Outcome, for Boys and Girls Combined, at an Increase by 1 mg/L in Creatinine-Adjusted Maternal Urine Fluoride Concentration for IQ in the MIREC Study, GCI (Upper Rows) and IQ (Lower Rows) in the ELEMENT study, and a Joint Calculation. The Column to the Right (p_{diff}) Shows the p-Value for a Hypothesis of Identical Regressions in the two studies. Two Concentration-Response Models are Used, a Linear and one with the Squared Exposure Variable

	MIREC		ELEMENT		Joint MIREC-ELEMENT		
model	beta	p	beta	p	beta	p	p_{diff}
	FSIQ ($n = 407$)		GCI ($n = 287$)				
Linear	−2.01	0.16	−6.29	0.007	−3.20	0.008	0.12
Squared	−0.419	0.40	−2.68	0.02	−0.780	0.09	0.07
	FSIQ ($n = 407$)		IQ ($n = 211$)				
Linear	−2.01	0.16	−5.00	0.01	−3.07	0.01	0.22
Squared	−0.419	0.40	−2.65	0.002	−0.998	0.023	0.025

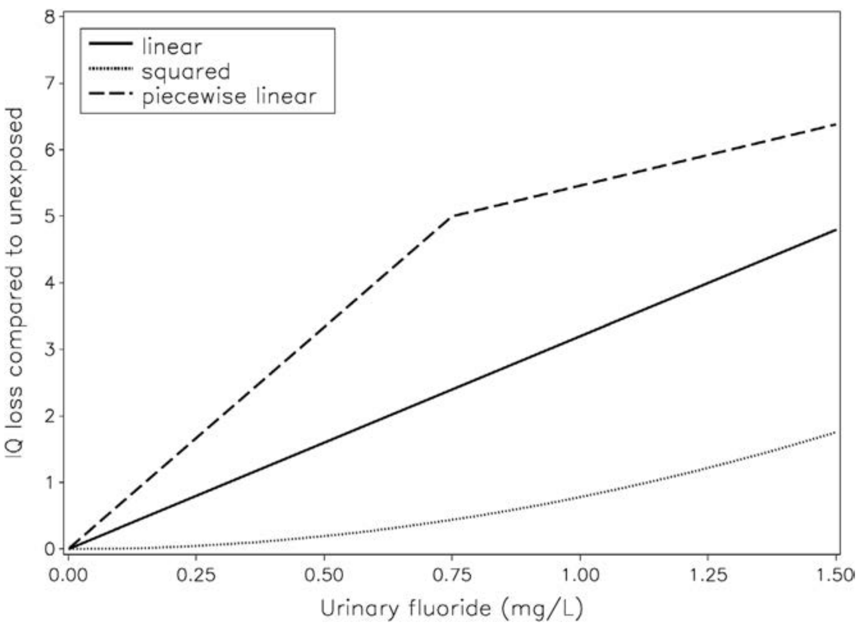

Fig 1. Association between creatinine-adjusted maternal urinary-fluoride (U-F) concentration in pregnancy and child IQ loss for the larger number of children (joint for GCI in ELEMENT and MIREC). Covariate-adjusted models are shown for the linear (solid), squared (dotted), and piecewise (dashed) linear curve with breakpoint 0.75 mg/L. The BMC is the U-F concentration that corresponds to an IQ loss of 1 (numbers shown in Tables 2 and 3).

ELEMENT data yielded results that were very close to those shown in Table 2, that is, with BMC values of about 0.19 mg/L for the linear model and about 0.63 mg/L for the squared model (data not shown).

Linear models allowing for sex-dependent effects showed a slightly better fit in the AIC mainly due to the significant interaction terms in the MIREC cohort. Although the BMCL in the MIREC cohort is clearly higher in girls than boys (0.61 vs. 0.13 mg/L), the overall BMCL for both sexes in the MIREC cohort (0.23 mg/L) is closer to the one for boys than the one for girls (Table 2). Sex-linked differences were not significant in the ELEMENT study.

Table 3 shows results using piecewise linear functions, with one breakpoint at 0.75 mg/L and one at 0.5 mg/L. A piecewise linear model is more flexible than a linear model, but AIC results showed that the joint piecewise linear models in Table 3 did not fit better than the standard linear models in Table 2. Thus, the hypothesis of a linear concentration-response relation could not be rejected: for the joint MIREC-ELEMENT IQ model, p-values for likelihood testing were $p = 0.18$ and $p = 0.15$ when the linear model was tested against models using breakpoints of 0.5 and 0.75 mg/L, respectively. For the joint MIREC-ELEMENT GCI model, the corresponding p-values were $p = 0.83$ and $p = 0.48$.

The shapes of the linear, the squared, and one piecewise concentration-response curves are shown in Fig. 1. In accordance with the BMC values, the Fig. shows that the squared model has a weaker slope

Table 2. Benchmark Concentration Results (mg/L Urinary Fluoride, Creatinine-Adjusted) for a BMR of 1 IQ Point Obtained from the MIREC Study and the Two Cognitive Assessments from the ELEMENT Study as Well as the Joint Results. Two Concentration-Response Models are used, a Linear and One with the Squared Exposure Variable. For both Models, Sex-Specific and joint benchmark Results are Provided. The fit of the Regression models was Compared by the AIC (Where Lower Values Indicate a Better Fit)

Study		MIREC ($n = 407$)		ELEMENT IQ ($n = 211$)		ELEMENT GCI ($n = 287$)		MIREC and ELEMENT IQ ($n = 618$)			MIREC and ELEMENT GCI ($n = 694$)		
Model	Sex	BMC	BMCL	BMC	BMCL	BMC	BMCL	BMC	BMCL	AIC	BMC	BMCL	AIC
Linear	Both	0.497	0.228	0.200	0.122	0.159	0.099	0.326	0.201	4770.1	0.312	0.192	5491.3
Linear	Boys	0.201	0.125	0.275	0.130	0.148	0.084	0.222	0.144	4766.7	0.184	0.125	5488.4
Linear	Girls	∞	0.609	0.160	0.091	0.169	0.087	1.098	0.275	4766.7	2.972	0.315	5488.4
Squared	Both	1.545	0.896	0.614	0.496	0.611	0.467	1.008	0.768	4768.8	1.133	0.807	5493.9
Squared	Boys	0.840	0.622	0.684	0.496	0.581	0.435	0.787	0.619	4769.4	0.761	0.601	5493.7
Squared	Girls	∞	1.262	0.576	0.449	0.642	0.434	1.637	0.866	4769.4	∞	1.040	5493.7

Abbreviations: AIC, Akaike Information Criterion; BMC, benchmark concentration; BMCL, benchmark concentration level; BMR, benchmark response; GCI, Global Cognitive Index; IQ, Intelligence Quotient.

Table 3. Benchmark Concentration (BMC) Results (mg/L Urinary Fluoride, Creatinine-Adjusted) for a BMR of 1 IQ Point Obtained from the MIREC Study and the Two Cognitive Assessments from the ELEMENT study as well as the Joint Results. Two Piecewise Linear Concentration-Response Models (with Urinary Fluoride Breakpoints at 0.5 and 0.75 mg/L) are used. For both Models, Sex-Dependent and Joint Benchmark results are Provided. The fit of the Regression Models was Compared by the AIC (Where Lower Values Indicate a Better Fit)

		MIREC ($n = 407$)		ELEMENT IQ ($n = 211$)		ELEMENT GCI ($n = 287$)		MIREC and ELEMENT IQ ($n = 618$)			MIREC and ELEMENT GCI ($n = 694$)		
Study	Sex	BMC	BMCL	BMC	BMCL	BMC	BMCL	BMC	BMCL	AIC	BMC	BMCL	AIC
Breakpoint 0.5	Both	1.751	0.092	2.688	0.431	1.004	0.042	1.073	0.139	4770.6	0.788	0.104	5495.0
Breakpoint 0.5	Boys	0.086	0.040	2.953	0.135	0.725	0.011	0.156	0.053	4766.7	0.087	0.040	5493.9
Breakpoint 0.5	Girls	∞	0.309	2.363	0.024	1.144	0.046	2.913	0.428	4766.7	3.817	0.385	5493.9
Breakpoint 0.75	Both	0.166	0.081	1.283	0.149	0.115	0.050	0.284	0.112	4769.8	0.150	0.083	5493.8
Breakpoint 0.75	Boys	0.082	0.049	1.379	0.121	0.127	0.035	0.136	0.070	4769.4	0.086	0.052	5493.6
Breakpoint 0.75	Girls	∞	0.125	1.155	0.052	0.109	0.044	1.365	0.140	4769.4	0.413	0.106	5493.6

Abbreviations: AIC, Akaike Information Criterion; BMC, benchmark concentration; BMCL, benchmark concentration level; BMR, benchmark response; GCI, Global Cognitive Index; IQ, Intelligence Quotient.

at low concentrations, while the low-concentration slope for the piece-wise association is steeper.

4. DISCUSSION

Experimental and cross-sectional epidemiology studies have provided evidence of fluoride neurotoxicity, especially when the exposure occurs during early brain development (Grandjean, 2019). As early as 2006, sufficient evidence was available to warrant further consideration of the possible brain toxicity of fluoride exposure with an emphasis on vulnerable populations (National Research Council, 2006). We now have thorough prospective epidemiology evidence on populations exposed to fluoridated water (about 0.7 mg/L) or comparable exposure from fluoridated salt and other sources. The present study is based on data from two prospective birth cohort studies (Bashash et al., 2017; Green et al., 2019) that include detailed assessment of child IQ and urinary fluoride concentrations during pregnancy. In these two studies, the mean U-F concentration (creatinine-adjusted) was similar among pregnant women living in Mexico City (0.89 mg/L) and the pregnant women living in fluoridated cities in Canada (0.84 mg/L).

Due to the brain's continued vulnerability across early development (Grandjean, 2013), early infancy may also be a vulnerable period of exposure for adverse effects from fluoride, especially among bottle-fed infants who receive formula reconstituted with fluoridated water (Till et al., 2019). Still, the effects of fetal exposure (i.e., U-F in pregnancy) in the MIREC Study remained significant when adjusting for exposure occurring in infancy. Similarly, in the ELEMENT study, the effect of maternal U-F was only marginally reduced after controlling for child U-F; fluoride exposure in school-age children showed a weaker and nonstatistically significant association with child IQ (Bashash et al., 2017). Taken together, these findings suggest that fetal brain development is highly vulnerable to fluoride exposure.

The magnitude of the fluoride-associated IQ losses is in accordance with findings in cross-sectional studies carried out in communities where the children examined had likely been exposed to chronic water-fluoride concentrations throughout development (Choi, Sun, Zhang, & Grandjean, 2012). More recent studies have shown similar results (Wang et al., 2020; Yu et al., 2018), and benchmark dose calculations (Hirzy, Connett, Xiang, Spittle, & Kennedy, 2016) relying on a large cross-sectional study (Xiang et al., 2003) showed results on the linear association similar to the ones obtained in the current analysis. These findings provide additional evidence that fluoride is a developmental neurotoxicant (i.e., causing adverse effects on brain development in early life). Given the ubiquity of fluoride exposure, the population impact of adverse effects from fluoride may be even greater than for other toxic elements like lead, mercury, and arsenic (Nilsen et al. 2020). Adverse effects of the latter trace elements are associated with blood concentrations that are about 100-fold lower than the serum-fluoride concentration that corresponds to the benchmark concentration (Grandjean, 2019).

A few retrospective studies have been carried out in communities with elevated fluoride exposure, though with imprecise exposure assessment that mostly relied on proxy variables, and without prenatal fluoride measurements (Aggeborn & Ohman, 2017; Broadbent et al., 2015). In addition to IQ outcome studies, the ELEMENT cohort found that elevated maternal U-F concentrations were associated with higher scores on inattention on the Conners' Rating Scale, an indication of Attention-Deficit/Hyperactivity Disorder (ADHD) behaviors (Bashash et al., 2018). Other studies on attention outcomes found an association between water fluoridation and diagnosis of ADHD in Canada, although data on child U-F did not replicate this association (Riddell, Malin, Flora, McCague, & Till, 2019), which is consistent with the ELEMENT study of child U-F and IQ (Bashash et al., 2017). Similarly, increased risk of ADHD was reported to be associated with water fluoridation at the state level in the United States (Malin & Till, 2015), although inclusion of mean elevation at the residence as a covariate made the association nonsignificant (Perrott, 2018).

Individual vulnerability may play a role in fluoride neurotoxicity. In the original MIREC study, boys were more vulnerable to prenatal fluoride neurotoxicity than girls (Green et al., 2019) suggesting that sex-dependent endocrine disruption may play a role (Bergman et al., 2013), among other sex-differential possibilities. Genetic predisposition to fluoride neurotoxicity may also exist (Cui et al., 2018; Zhang et al., 2015), but has so far not been verified. Other predisposing factors, such as iodine deficiency (Malin, Riddell, McCague, & Till, 2018) may contribute. For such reasons, regulatory agencies routinely use an uncertainty factor to derive safe exposure levels that are lower than the BMCL.

Both prospective studies adjusted for a substantial number of cofactors. Prenatal and early postnatal lead exposure did not influence the ELEMENT fluoride-associated IQ deficits (Bashash et al., 2017). Adjustment for other neurotoxicants or risk factors, such as arsenic and lead exposure, did not appreciably change the estimates in the MIREC study (Green et al., 2019). While BMC results were calculated for the creatinine-adjusted U-F available from both studies, U-F results adjusted for specific-gravity were available for an additional 105 MIREC women; if using the latter U-F data, slightly lower BMC results were obtained, as compared to those based on creatinine-adjusted data only. Higher results were obtained for the squared, and lower for the broken linear slopes, but neither showed a superior fit to the data when compared to the linear relationship between maternal U-F and child IQ.

The increased precision using the average maternal U-F concentration as an indicator of prenatal fluoride exposure results in stronger statistical evidence of fluoride-associated deficits, compared with using cross-sectional or retrospective studies. Still, the amount of fluoride that reaches the brain during early brain development is unknown, and even the maternal U-F concentration measurements may be considered somewhat imprecise as dose indicators. Such imprecision, likely occurring at random, will tend to underestimate fluoride neurotoxicity (Grandjean & Budtz-Jørgensen, 2010).

The prospective studies offer strong evidence of prenatal neurotoxicity, and the benchmark results should inspire a revision of water-fluoride recommendations aimed at protecting pregnant women and young children. While systemic fluoride exposure has been linked to dental health benefits in early studies (Iheozor-Ejiofor et al., 2015), these benefits occur in the oral cavity after teeth have erupted (Featherstone, 2000), thus suggesting that use of fluoridated toothpaste and other topical treatment should be considered for alternative caries prevention.

5. CONCLUSIONS

Two prospective studies examined concentration-dependent cognitive deficits associated with the maternal U-F during pregnancy; one of the studies (Bashash et al., 2017 measured child IQ at two ages and found similar results, whereas the other study (Green et al., 2019) found a fluoride-IQ effect only in boys. We explored the shape of the concentration-response curve by using a standard linear shape and compared with a squared exposure and a piecewise linear function that allowed a change in steepness at two points within the range of exposures. Comparisons between the models suggest that the standard linear function is a reasonable approximation. All of these estimates have a certain degree of uncertainty, and emphasis should therefore be placed on the joint BMC results from the two studies and involving both sexes. These findings, using a linear concentration dependence, suggest an overall BMCL for fluoride concentrations in urine of approximately 0.2 mg/L. The results of this benchmark analysis should be incorporated when developing strategies to facilitate lowering fluoride exposure among pregnant women.

CONFLICT OF INTEREST

PG has served as an expert on the hazards of environmental chemicals on behalf of the plaintiffs in Food & Water Watch v. US EPA. HH and BL served as nonretained expert witnesses (uncompensated) for the same trial, in which they offered testimony regarding the studies their respective teams on fluoride exposure and neurobehavioral outcomes. All other authors have no interest to declare.

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January 27, 2022

TO: White House Environmental Justice Advisory Council

TESTIMONY:

I am Lea Harper of FreshWater Accountability Project in Ohio and a member of Anthropocene Alliance. Thank you for another opportunity to address WHEJC again today. Normally we are too busy advocating for those who have been harmed by fracking in SE Ohio and the Ohio River Valley of Appalachia to engage as we would like, but this initiative has given us hope that our voices will be heard. Fracking has proliferated by bullying and buying its way past regulators, legislators and concerned citizens to be predatory upon those who are systemically disempowered to advocate effectively for themselves. When fracking first came to the region, people wanted to believe the industry propaganda that fracking would make the region rich and provide much-needed jobs, but now they are seeing that they were misled again. If fossil fuels would make a region rich, then SE Ohio would be the richest part of the state rather than the poorest. Already economic projections are showing that the true costs of fracking are being externalized upon the public, and all costs considered, is not worth the environmental and health harms. Just like with the thousands of abandoned oil and gas wells in the region that must be plugged and the acid mine drainage remediation from the coal industry that is being funded by public tax dollars, the fracking LLC's are poised to leave a costly legacy of toxic, radioactive contamination at our expense.

I never thought as a citizen of the United States that I would witness the human rights violations that the industry of fracking has brought, validated by the People's Permanent Tribunal at the UN. Fracking is a case study of how disenfranchised people are ignored and bullied into silence by moneyed interests. Before the industry could even get started, fracking had to be granted special environmental exemptions that no other industry in the US can have, called the Halliburton Loophole, which allows the industry to cheaply dispose of its toxic, radioactive waste as non-hazardous. It has taken many legislators and regulators to look the other way to allow the harms of fracking to continue despite the fact that scientific and economic studies show the costs are not worth it. I have personally witnessed environmental and public health harms caused by fracking that I thought could only take place in a third world country which apparently Appalachia is as a resource colony for the oil and gas industry. People are being sickened and some have died because of fracking. Recently, one of my scientific colleagues told me that he knows 10 people personally who have died as a result of the air and water pollution caused by fracking. There are many more, but unfortunately, cancer takes time and doesn't tell us its cause. As our air and water continues to be contaminated in the region, an epidemiological examination shows that cumulative air and water pollutants are already causing harmful health effects, yet more toxic polluters like frack waste processors and petrochemical plants are given permits to massively pollute on top of existing toxic loads. We have a community air monitoring program that is already showing that toxic levels of frack industry-related pollutants are associated with human health impacts, but we still have to provide the proof while the industry continues to deny its obvious harms. It appears our local, state and federal representatives care more about the short-term revenue from the industry than the long-term costs it is allowed to externalize upon us.

The fracking industry has only been able to operate as it has for so long by silencing those who are harmed through obstruction and denial or through gag orders if a family perseveres long enough to obtain a settlement. In many cases, people cannot find or afford a lawyer to represent them. We have to prove that the industry did it, and who has the money to do the air and water monitoring to assign accountability? People working hard to feed their families have little time and resources to resist, plus they have hope in false promises. The fracking industry thrives on that as whatever regulatory enforcement we have left is understaffed and underfunded as short-term profit and political favor trump good environmental and public health policy. For instance, the Ohio Air Nuisance rule was unilaterally removed by the USEPA from the State Implementation Plan when we needed it the most because of fracking, cutting off access to an important recourse. No wonder people in Appalachia become discouraged and lose hope in something better for themselves and their families.

The fossil fuel industry knows that dismantling regulations is a lucrative profit center, historically targeting legislation to dismantle important environmental regulations. The “Halliburton Loophole” in the Energy Policy Act of 2005 is a prime example. The fracking industry required exemptions from important environmental regulations to operate profitably – these are exemptions that no other industry in the US can have. Even so, it still struggles to be profitable, and the environmental exemptions have just externalized the costs upon others just like how the public is paying for acid mine drainage remediation and for the plugging of abandoned gas and oil wells. That is their playbook, and they play it very well as people in the resource colony become sicker and poorer as a result.

People living in the regions where fracking takes place are realizing that the few dangerous, toxic jobs are not worth it, and that the industry’s claim of energy freedom is a lie as product is exported overseas to higher-priced markets as our heating costs go up as a result. I never thought I would witness how people who are impoverished could be exploited with the implied consent of our own government. Now the Ohio River Valley is on yet another chopping block because of fracking to be turned into another cancer alley petrochemical corridor like the Gulf Coast for foreign interests. Fracking is bad enough, but it is leading to even greater harms like plastic and climate change. I feel sorry for those working at the regulatory agencies who are witnessing the harms and unable to do anything, often putting the blame back on the victim to do more to prove guilt of the industry that is the culprit but does not have to prove its innocence. No other industry in the US has important environmental regulations dismantled like fracking, and it is not ending well. Like Flint, Michigan, there will be congressional hearings, unfortunately, after it is too late. That is why we are hopeful that environmental injustice will be amplified and action taken before more people die just because they could be taken advantage of by living in the wrong place and working for the wrong employer without the recourse and respect given other more affluent regions of the country.

For your further information: I have copied and pasted below a recent newsletter from the Ohio River Valley Institute – I hope you read it and realize how Appalachians continued to suffer injustices and how the fracking industry propaganda machine must be stopped from continued mischaracterization and outright lies about its economic contributions: https://ohiorivervalleyinstitute.org/

Thank you for your serious consideration of our urgent plea for help to stop the further exploitation of the Ohio River Valley!

Best,

Lea Harper

Managing Director

January 27, 2022

The natural gas industry's false narratives—and false promises—have misled Appalachia for more than a decade.

Today, industry boosters would have you believe that natural gas development "buoyed" Ohio's fracking counties through the 2020 recession. That's according to a **recent Columbus Dispatch article** congratulating Monroe and Harrison counties for "posting the state's biggest economic gains in 2020."

But at the same time Ohio's fracking region was leading the state in economic output, it was **hemorrhaging jobs, shedding residents, and eking out income growth below the state average**. It's a depressing reminder that natural gas development is **structurally incapable** of generating local economic prosperity, no matter how much money is invested or how much gas is drilled.

New technologies won't save gas production, either. Increasingly, policymakers and analysts are eyeing carbon capture, use, and sequestration (CCUS) as a "silver bullet" solution to decarbonization that could prolong gas extraction in Appalachia and elsewhere. But CCUS is an economic non-starter and a poor means of decarbonization, Senior Researcher Sean O'Leary **explains**. Exorbitantly expensive, CCUS and related carbon management technologies cannot be cost-justified and have not proven their viability at scale. Moreover, the adoption of these technologies in economically struggling regions will contribute little to job growth and local prosperity and may even be counter-productive.

The route to shared prosperity and true economic growth in Appalachia is paved with effective policy, which can't happen without Black voices. Read the Black Appalachian Coalition's new **BLAC Paper** to learn more about how Black storytelling can inform sound, equitable policymaking.

HAWKINS

Corporate Office
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PRODUCT DATA SHEET

Hydrofluosilicic Acid 23-25%

PDS – 1120; REVISION 05
EFFECTIVE DATE: 02 NOV 20

General Characteristics:

Appearance:	Clear, colorless to light yellow solution
Odor:	Pungent
Chemical Formula:	H_2SiF_6
Molecular Weight:	144.09
CAS#:	16961-83-4
Freezing Point:	4° F
Boiling Point:	222.5° F
Shelf Life:	730 days
Storage Recommendations:	In accordance with AWWA B703- Fluorosilicic Acid

Standard Specifications:

COMPONENT	SPECIFICATION
Assay, wt.%	23.0 – 25.0
Free Acid (as HF), wt.%	≤ 1.00
Fluorine (F), wt.%	18.00 – 21.00
Lead (Pb), ppm	≤ 50
Arsenic (As), ppm	≤ 50
Iron (Fe), ppm	≤ 100
P_2O_5, wt.%	≤ 0.5

PHYSICAL PROPERTIES	
Specific Gravity, 60° F	1.210 – 1.240

Additional Information:

AWWA Standard: Product meets AWWA Standard B703 for Fluorosilicic Acid.

Bioterrorism Act of 2002: All appropriate Hawkins, Inc. facilities are registered with the FDA per the Public Health Security and Bioterrorism Preparedness and Response Act of 2002.

Country of Origin: Product is manufactured in the United States.

NSF Certification: Certified to NSF ANSI/Std. 60 with a maximum use level of 6 mg/L.

Notice for Product Numbers: 1100, 1102, 1125, 1135, 1145, 1160, 32665, 34124, 41868, 57273 ("Product(s)")

Hawkins, Inc. ("Hawkins") presents the information in this Product Data Sheet ("Information") in good faith and believes the Information to be accurate as of the Effective Date. Hawkins warrants only that when Hawkins ships the Product, it will meet published specifications. Other than this warranty, **HAWKINS MAKES NO OTHER REPRESENTATION OR WARRANTY, EITHER EXPRESS OR IMPLIED, FOR COMPLETENESS, ACCURACY, MERCHANTABILITY, FITNESS FOR A PARTICULAR PURPOSE, OR ANY OTHER NATURE WITH RESPECT TO THE INFORMATION, OR TO THE PRODUCT TO WHICH THIS INFORMATION REFERS.** Hawkins will not be responsible for damages of any nature whatsoever resulting from the use of, or reliance upon, the Information or the Product to which the Information refers.

January 15, 2022

TO:
National Environmental Justice Advisory Council nejac@epa.gov
Public Comment for Mtg 1/5/2022: EPA-HQ-OA-2021-0848

White House Environmental Justice Advisory Council whejac@epa.gov
Public Comment for Mtg 1/26-27/2022: EPA-HQ-OA-2021-0683

Dear NEJAC and WHEJAC Members,

I am a healthcare professional with several years experience as a maternal-child public health nurse for the New Hampshire Department of Health and Human Services. I write to inform your councils about the risks of water fluoridation and the need to reverse course.

I support quality dental care for all people. This includes access to professionals who can monitor oral health and intervene as needed.

Misguided public officials have supported water fluoridation with the hope this practice will decrease caries among people who do not have dental coverage. This is an unacceptable policy. The risks associated with fluoridation, including developmental neurotoxicity, preclude its consideration as a fair and effective policy for the general population. Environmental justice means access to public water untainted by fluoridation chemicals.

Please work to end water fluoridation. You will find ample support in Dr. Christine Till's excellent video summarizing the history of water fluoridation and what the scientific research is telling us (*Fluoride and the developing brain*. 28 September 2021. Dr. Christine Till Presentation to Rotary Club of Calgary.mp4 on Vimeo).

Dr. Till joined Dr. Bruce Lanphear and Dr. Linda Birnbaum in an op-ed published in Environmental Health News and found here: Op-ed: It is time to protect kids' developing brains from fluoride (7 October 2020). Of note is Dr. Birnbaum's former role as Director, National Institute of Environmental Health Sciences and National Toxicology Program of the National Institutes of Health.

Sincerely,

Katie Lajoie, BSN, BA, RN
429 Wheeler Rand Road
Charlestown, NH 03603
603-826-4803
jlje2316@gmail.com

D559

Ms. Sharice Davids

Member of Congress (D-KS)

Member Ho-Chunk Nation of Wisconsin

Co-chair Native American Caucus

Cc: Legislative directors of all Caucus Members

RE: FINDING A US FEDERAL GOVERNMENT CHAMPION FOR NATIVE CANADIANS HARMED BY CONSTRUCTION OF MEGA DAMS/FINDING A LEGAL REASON WHY A UNIT OF THE US GOVERNMENT CAN REFUSE TO PURCHASE CANDIAN HYDRO ELECTRICITY ON HUMANITARIAN GROUNDS

Dear Congress Woman Davids:

I am a grandmother with a degree in health planning residing in New York city since 1994. I was born in Detroit, Michigan on the Canadian border which we crossed frequently to purchase groceries and visit relatives. My father was born in Montreal and my mother in Jaffa Israel when it was under British mandate. I raised my children in Wisconsin your ancestral land. I am 100% European Jewish and my granddaughters are probably 20% indigenous. How a country treats its minorities is a human rights issue. You don't have to be a minority to redress injustice toward minorities. But if you are a minority, it is easier to put yourself in the place of other minorities. I think that is what is happening as I continue to learn about the way Canadian Hydro is made.

I am writing the Native American Caucus for help discovering whether US law or US agreements under international law could provide a rationale for any unit of the US government to decline to purchase electricity from Hydro Quebec on purely humanitarian grounds. I am prompted to write you now by the prospect of one final hearing by our State Regulatory system followed by a green light for a transmission line and a contract to bring hydro power from Canada into Astoria, New York City.

The current way Canada produces electricity distresses me. First destroy huge swaths of wilderness ignoring Canadian treaties with tribes living on the land. The land is often flat, so to amass sufficient water, flood an area that could be the size of New York State. Do not ask permission of the tribe or refer to previous treaties. Let the dying trees emit c02 adding to global warming. Leave the people on what was once land to starve or die from methyl mercury poisoning. Prevent them from practicing their traditional way of life and passing on valued culture to their children.

Put in climate activist terms. the construction of the dams creates environmental justice communities. But what is the legal framework by which the US deals with these communities and protects them/prevents them from occurring? Right now, it appears morality stops at the border.

Climate activists in New York have been trying to prevent a transmission line called CHPE which would bring hydro power from Canada into New York City. Our regulators have announced a final hearing. The transmission line is paired with a contract with Hydro Quebec. This has prompted me to write the Native American Caucus for help locating a legal framework that could justify halting the purchase on purely humanitarian grounds.

If this Canadian "mining" created climate justice communities in New York City perhaps our new state climate law would provide a rationale for not doing business with Hydro Quebec. But the injustice to native peoples is happening in Canada. If the people working on the dams in Canada were the ones suffering, then perhaps some UN or other international labor treaty could be invoked. But the harm is done to populations living in the construction zone. This harm includes starvation, inability to hunt farm or fish, poisoning of the water with methyl mercury and inability to pass on way of life/culture to one's children. This really hits home with me. **How can this be legal when it is done in the name of clean renewable energy and a solution to climate change?**

I wonder if the Native American Caucus has any official charge from our federal government to concern itself with this injustice. If so, please let me know where this charge is documented. If the caucus has no mandate to look into these problems that start beyond our borders, I suspect your social contacts throughout the world have made you well aware of this Canadian practice.

Many of us climate activists believe our only recourse would be a member of congress assigned to look at trade, but I am having trouble locating the committee.

Many of us tried to find a US official to halt the transmission line's trade permit, but we have not found a champion in the Department of Energy. Perhaps your caucus can find that champion and or amend a current law that would declare creation of environmental justice communities *anywhere* grounds for rejection by the potential US government purchaser.

Congress Woman Davids, I hope you can not only share this letter with your legislative staff but also circulate it among the Native American Caucus. Ideally, more than one caucus member has information about possible champions who could help end this old-fashioned exploitation of native peoples now out of place in the twenty first century.

I hope to receive a reply from you and other members of the Native American Caucus identifying laws, potential lawmakers, potential champions within the legislative or executive branch of government.

If you need further information. I would be happy to provide it.

Sincerely,

Rachel Makleff

Mba Health Planning University of Wisconsin Madison

212 316 0673 cell: (for texting) 646 671 2115

320 Riverside Drive apt 1-H

New York, NY 10025

Public Comment to the White House Environmental Justice Advisory Committee

Enlarged and re-submitted after the sessions of

Wednesday January 26, 2022

Rachel Makleff PhD in the Humanities

Mba in Health Planning

Member of Grow Renewable New York: No Canadian Hydro

Volunteer for prominent national environmental organizations

Dear WHEJAC and staff:

Thank you for outstanding work on behalf of Environmental Justice Communities in the United States. Please send on my gratitude to Vice President Kamala Harris for *her* skillful work in this field and to President Biden for having our country rejoin the Paris Agreement and carefully crafting US staff design to support the many aspects of the international effort.

I am especially grateful today for the Executive Orders that created WHEJAC and the public comment period which gives me a voice. But I am sadly disappointed in a gap I see in US responsibility to clean up a situation our own country is creating.

The lover guards the beloved

May wickedness be diminished

(Traditional Jewish morning prayer)

Dear Fellow Americans:

I sang these words this morning during a zoom prayer service. And I took them to heart. There ***is*** a way for wickedness to be diminished in our import regulations. We need more guardrails.

Currently, if workers in a factory outside our borders are harmed by working conditions or if consumer use of the product in the United States will harm our citizens **<u>it is understood that</u>** **<u>trade should be forbidden</u>**. But if people living on foreign soil ***near a factory beyond our borders*** are harmed by the manufacturing process there is no required lifting of a trade permit. This is happening over and over again in the importation of hydroelectricity into the United States from Canada. The manufacturing process floods large swaths of land resulting in deforestation and release of carbon dioxide – the exact opposite of what this planet needs. It destroys ecology and poisons first nations through the creation of methyl mercury. It starves first nation peoples who did not give permission for this despoliation of nature they depend on as a food supply and as a way to pray to their creator. The rights of native peoples to live on their land and pass on traditions to the next generation are halted.

We are rejoining the United Nations through the Paris Agreement while creating global warming beyond our borders and denying the rights of native peoples described in the UN Rights of Man. We are creating environmental justice communities beyond our borders. <u>This has to stop.</u>

I am lodging this protest based on a situation I have personally experienced in New York City where we know this is happening with no moral insight by the Department of Energy which reports to the President.

For at least ten years, mainstream activists in the Sierra Club have sought to stop a transmission line which would import hydroelectricity into New York City. If this one additional transmission line is built there is no guarantee that the Canadian government would stop building their dams. We have tried to interest our members of Congress but have had no success. A Presidential permit for the transmission line was issued under President Trump. Now with the election of President Biden and our re-entry into the Paris agreements we felt we had a chance to go directly to the President and his Executive Departments to rescind the presidential permit.

One of our members began petitioning the federal Department of Energy asking it to rescind the presidential trade permit including on humanitarian grounds. She was told, "This is not our purview, this is international."

- Reform of trade regulation is needed to consistent with the suite of Executive Orders that created WHEJAC and which looked forward to a more generous global role for the United States as leader of climate policy.

In looking at the description of WHEJAC duties I am uncertain as to whether WHEJAC can advise on this international policy of the trade permits. I call upon President Biden to authorize increased power of WHEJAC to work with all relevant departments in his cabinet to fix this *gap in common sense justice*. Increasing EJ communities and greenhouse gas at the same time by turning a blind eye doesn't fit with America's role as a leader within the Paris Agreement.

- Oversight and implementation of this reform of trade regulations should be added to WHEJAC responsibilities
- Because of experience, training, and values WHEJAC is unusually well suited for this new role

While not trained in this field, I have been looking at how US trade policy could prevent this kind of situation in general. I belong to several fair-trade groups as a grassroots supporter and none to date have shown any interest in studying this problem. I realize this inaction might simply be caused by lack of resources on the part of staff. But here is WHEJAC under the gaze of the White House. Perhaps there is a way to lend staff to some of the fair-trade associations so they can advise.

Congress could prohibit creation of EJ communities abroad within our US definition of fair trade with a statement. But there is a catch. As Lori Wallach used to point out in her webinars –many producers are given an exception and considered as if their product was "made in America" thus creating a loophole.

In fixing this loophole, I suggest staff from the office of our Trade Representative attend meetings with the experts at WHEJAC. I also suggest that the Labor Department experts on NAFTA should be asked to help WHEJAC look for regulatory solutions.

Interim Reform:

I am asking President Biden to announce that reforming the whole system that allows for creation of these EJ communities outside our borders is needed.

I am asking the President to further announce that either his office or his executive departments should have the ability to deny or rescind the presidential trade permit on purely humanitarian grounds starting now. Preliminary emergency guidelines for making this decision should be drafted. Records should be kept on how often this is happening and where. WHEJAC and all interested departments should be given copies of these records.

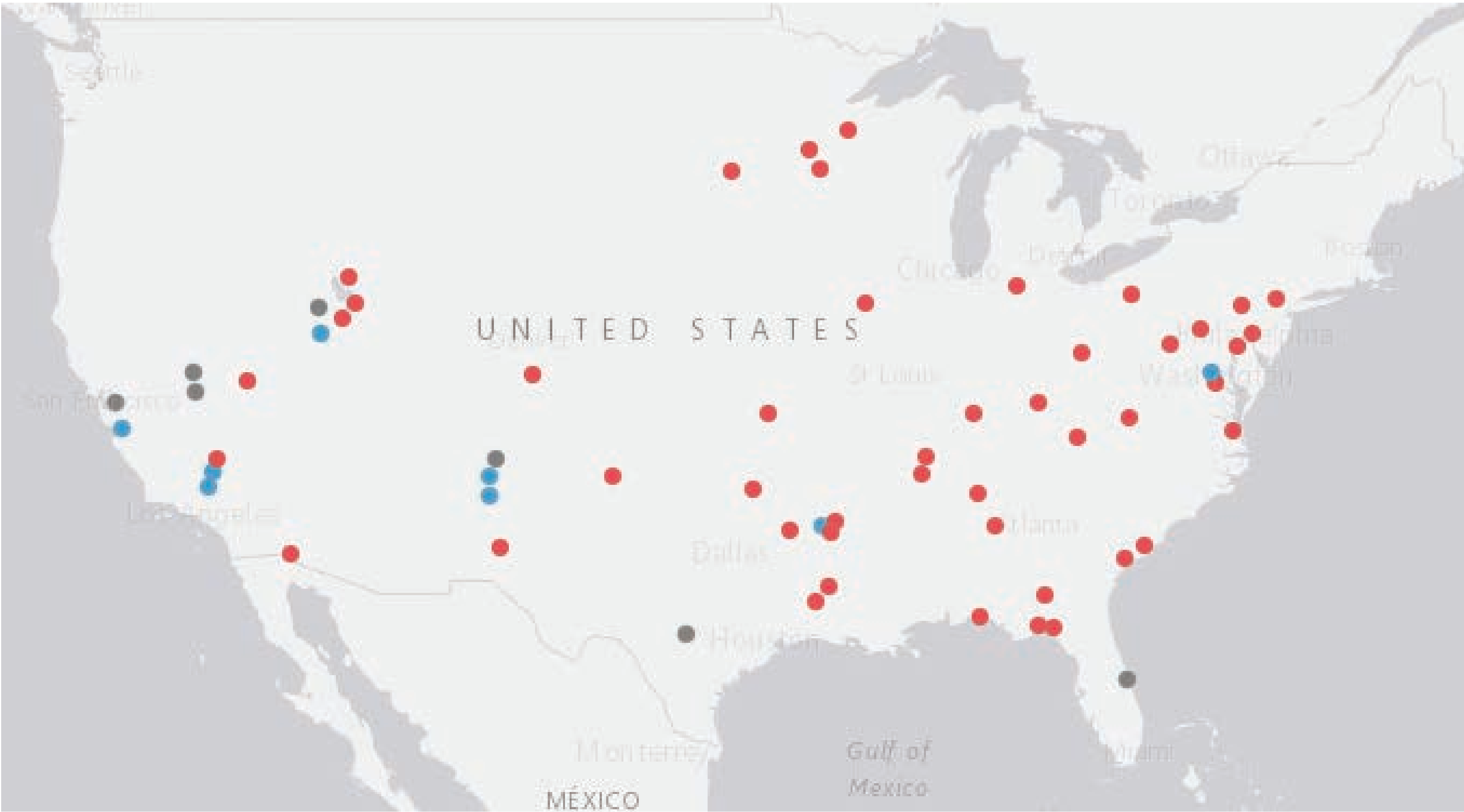
As an example of the harm to EJ communities in Canada that needs to be stopped, I am sending via url a news article written by a first nation Canadian on how his life changed when a mega dam was built near his home.

Thank you for the opportunity to share my experience with you.

"The lover guards the beloved

May wickedness be diminished"

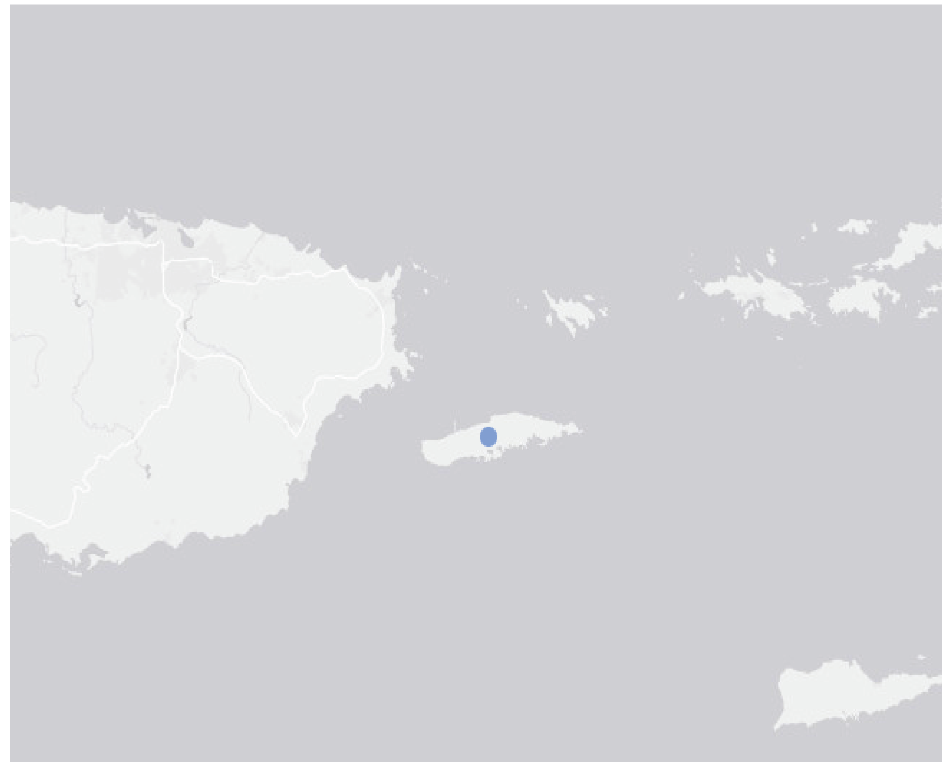
Map of Active Open Burn/Open Detonation Facilities in the U.S.



- Low-income community and greater than 50% people of color population.
- Low-income community.
- Either no data available or not a low-income community.

Note: Andersen Air Force Base in Guam and Former Atlantic Fleet Weapons Training Area in Puerto Rico are pictured on the following page.

Andersen Air Force Base, Yigo, Guam*



Former Atlantic Fleet Weapons Training Area, Vieques, Puerto Rico

**The demographic data for communities near the open burn/open detonation sites is based on Census data from the American Community Survey generated through EPA's EJScreen. The Census Bureau does not conduct the American Community Survey in Guam so demographic data for the community around this facility is not available.*

To: U.S. Customs and Border Protection Re: Scoping: Supporting Environmental Assessment Prefer Full EIS; Opposing Rio Grande Valley Border Barrier and Related System Elements
From: Sarah Bishop Merrill, M.S., Ph.D., January 24, 2022 (see photos and video attached)

This memo serves as my comment on the scope of the planned environmental review of our fragile eco-systems here in the Lower Rio Grande Delta, especially along the Brownsville Ship Channel. Not only do we have hard-working climate refugees and asylum-seekers trying to help us with our labor shortages in many fields from agriculture and health care to construction, but we also are home to the seriously threatened habitats of several other endangered species, e.g. the Aplomado Falcon, Ocelot, Sea Grasses, other birds, oysters, shrimp, fish, and the Manatee who have recently been spotted near the Bahia Grande, the largest freshwater restoration in the Northern Hemisphere. Bahia Grande restoration was completed in 2003 when I moved here from the Chicago area to teach Philosophy, --especially Applied Ethics, Social and Political Philosophy, Logic, etc. I am now retired, and thus able to observe and volunteer more, e.g., on State Park Friends (Estero Llano Grande) Board. I have studied environmental and social issues here for 20 years. I am a former Fellow of the Center for Women in Government and Civil Society, Rockefeller College, SUNY.

I see the scope of this Environmental Review as including the environmental justice required to maintain a delicate stream of human workers as well as listed species protection. The continued mistreatment of asylum seekers and climate change refugees needed for low paying jobs in our fields and construction projects is a serious concern. These immigrants should not be feared, given their important economic contributions. So both eco-system services and human capital are within your proper scope. Now that NEPA again includes, we hope, **cumulative environmental effects**, I urge you to consider environmental justice as well as the loss of habitat and access to the river which are threatened by more barriers. The removal of riverine habitat and new oil and gas projects also contribute to Climate Change.

I am glad that our EJ units of government are aware not only of the cumulative negative effects of the oil and gas proposed and existing projects on the TX Gulf Coast, but also of the cumulative **positive effects and future** of the real and growing diplomacy like that of our distinguished Vice President Harris, through cross—cultural projects, turning the Wall into graphic art displays and bi-cultural art projects, and through a regulated flow of labor and commerce across our border. In addition to the privilege of working on the transport team of the Angry Tias and Abuelas with my former colleague at South Texas College, Joyce Hamilton, I have been supporting progress toward protecting this precious wild area and its lovely Upland Lomas (TV personality, Richard Moore, funded by SAVERGV through the Hershey Foundation. See transcript or link below.) It is threatened by 2 LNG plants, though we stopped ANNOVA already. Also dangerous and polluting, and greenwashing the carbon footprint with their so-far unproven and ineffective "Carbon Capture Sequestration," are the Jupiter Project and oil and gas pipelines. The environmental integrity of our fragile coastal region is also at risk from Border Wall blockage of access to the river for many species, including many land-owners whose families have been here before Texas was in the United States, holding old land grants from the King of Spain, and living below the Wall. The Treaty of Guadalupe Hidalgo (1848) had established ownership and access that is now being stopped. And our friends the Native American tribe of Carrizo Comecrudo have sacred sites in this region as well. They also oppose the Wall, and gathered with us when French bank activists and journalists visited here.

The huge amounts of money spent on these barriers should be spent instead to restore, where possible, damaged habitat, fishing and shrimping access (the LNG tankers will block the Port of Brownsville for many hours), and to improve our air quality. Many cars have leaky mufflers and people cannot afford to repair them. Help us preserve what we have left, and prevent the worst effects of Climate Change, with more 100+ degree days each year.

As you see from this powerful photograph taken from the top of an upland loma, a wall or barrier on the land is a silly, counter-productive, and unecological project, since those wishing to enter the U.S. illegally can easily come in by boat or plane. The tunnel vision of those wishing to engineer more Wall focuses mainly on profit for a few contractors, as if the coastline and airport access did not exit. We have various small airports in Cameron County, on the Gulf Coast east of Brownsville, Harlingen, San Benito, Olmito, and La Feria. Beaches are accessible to cars and trucks here in Texas, where our State Constitution guarantees citizen access to beaches. So our Border Patrol is covering a huge area and does a great job. Quite a few of my thousands of former students here have married or are themselves Border Patrol officers. They deserve more credit for what they do in saving lives at the border, and promoting proper legal entry for the many people seeking jobs and an escape from the collapsed social contract of their Central American nations.

Our region remains peaceful and productive, a network of diplomatic and mutually beneficial economic, social, and environmental exchanges. At the Citizens' Forum of the International Boundary and Water Commission, (U.S. State Department) we have an open exchange of information and planning with the Mexican engineers on the Commission, which is tri-national (US, Canada, and Mexico), and we deal with the crucial issues of flood prevention, erosion control, water quality, and maintenance, access to, and security of, the levees. Several sections of the Wall were built right ON the levees a few years ago, after we notified Senator Hutchison that we needed levees for flood prevention, not more Wall.

I am so thankful that the Biden Administration understands how precious and fragile this region is, and how our riverine habitats need preservation, not only with an EA, but a full EIS, concerning the Border Wall construction, and other highly toxic oil and gas projects which have a cumulative effect of a huge Carbon and Water Footprint. We have been fighting for this against the invasion of those wishing to build numerous oil and gas facilities here, although LNG (Methane, the worst GHG there is!) is *not* a "transitional fuel." These planned projects, along with the Super Heavy Rocket test launches by SpaceX who built before the permitting process was conducted, have already ruined large sections of this coastal region, with declining air quality, and continuing debris and run off from spaceship launch failures, pipeline and other construction troubling most of the local residents. Boca Chica has been taken over by Elon Musk and Company, closing roads and beaches to which Texans have a state constitutional right. Methane (LNG) traps heat worse than CO_2. The worst thing that can happen besides the LNG toxicity and danger of catastrophic (Methane) Vapor Cloud Explosions, is more Border Wall construction. We fought so it would not take the Santa Ana National Wildlife Refuge, the Butterfly Center and the historic La Lomita Mission, with thousands of people coming out to demonstrate opposition to more Wall. Birds who need darkness to hunt cannot tolerate bright lights, by the way, and are dying in unprecedented numbers, or avoiding this region altogether, resulting in far lower numbers in our bird counts.

Our main business is Eco-Tourism, e.g., on South Padre Island, etc. Our Wildlife Corridor of preserved lands and protected habitats is still incomplete. Proposed LNG projects (also undergoing merely an EA and not a full EIS due to FERC's former conditional approval last November). But tourists don't like to see or experience air and noise pollution, closed beaches, and heavy machinery tearing up and building unpermitted facilities on land along Rts. 48 and 4. Given the proximity of the border, and their clearly significant negative environmental impact on the region, construction of LNG and Border Barrier facilities will ruin the birding, shrimping, and fishing which so many have loved, and come here to experience. Our local economy is rather well-integrated with that of our Mexican cousins and grandparents: our population, from my more than 19 years of teaching experience here, is peaceful and multi-cultural, respectful of human rights, and of the eco-system services we can never replace. The degradation and effects of extremes from Climate Change is irreversible.

Here is another reason to keep our mutual access with Mexico open: We may need help with catastrophes, just as they came to help us after Hurricane Dolly. Our uniformed services will not be able to deal with the catastrophe of a Vapor Cloud Explosion, or VCE, which can result from a small fire below igniting an invisible pool of Methane in the air above. Methane, being lighter than other components of air, rises above them and traps in heat at rates far higher than CO_2 does. It is highly flammable, and causes fires so intense that the whole sky burns for 25 miles around, throwing cars a kilometer away with its force. It may be necessary to have assistance from our Mexican friends when a predictable disaster occurs here. Our hospitals and fire-fighters have no way of fighting such a huge VCE, and as happened after deadly hurricanes here in the past decade, Mexicans have often come to our assistance. We have an active cultural diplomacy in progress, with mutual economic benefits, health care, educational and art collaboration: unless it is an open Wall of Artworks, (see recent books documenting some wonderful bi-cultural art projects and recent Spectrum 1 News reports) the wall is useless. The LNG companies have still not released their emergency plans, and no funding for training the uniformed services exists. These are unacceptable risks.

For more information, please see the FB site of SAVERGV, and see our many filings and comments through FERC, TCEQ, FAA, and other agencies. Below is a photograph of the Upland Lomas we are trying to protect, a link to Moore's video clip, and a transcript. I also attach some emails containing research and disturbing facts on SpaceX & the Rio Grande and Texas LNG proposals, Rio Bravo, & Valley Crossing Pipelines -- all more complicated issues than I address here.

Thank you for your service to our nation, our neighboring nations, and our planet's future.

((____))

"Richard Moore Outdoor Report: Threatened Lomas," 2 minutes 27 seconds, Channel 23 Valley Central News, https://www.valleycentral.com/richard-moore-outdoor-report1/richard-moore-outdoor-report-threatened-lomas/

HARLINGEN, Texas **(ValleyCentral)** — They are one of the rarest habitats in the world, perhaps the best wildlife habitat remaining in the Rio Grande Valley and among the most threatened.

These verdant Lomas or little hills visible from Highway 4 as you travel to Boca Chica Beach are unique geological formations found in only three locations in the world, Australia, Africa, and southernmost Texas. Formed over centuries from wind-blown silt or clay particles deposited by the Rio Grande, the prevailing south wind over time formed small rises which eventually became covered with vegetation. Acre for acre, these scattered Lomas are perhaps the most diverse habitat in South Texas. Benito Treviño, renowned naturalist and plant expert, recently visited the Lomas for the first time and was astounded at the abundance of plant life, likely exceeding 2,000 plants per acre. "I am very impressed with this habitat, Richard, it is like…I have never seen quite anything like this," said Treviño. There are only some 12 of these Lomas north of Highway 4, and most are on Brownsville Navigation District property, managed under a lease agreement with the United States Fish and Wildlife Service forming the current 4,600 acre Loma Ecological Preserve. This protective lease agreement expires in 2023, and thus far port officials have shown little inclination to renew Loma protection. This should be done as part of a clear region-wide policy that protects our security environmentally as well as socially. Security is more than a barren region cleared for a useless wall, a 13th Century technology which is obsolete and wasteful, destroying habitat and access to water for many species.

With the sprawling development of Space X and rapid expansion of Port of Brownsville, including a new road linking the port to Highway 4, the historic Lomas are threatened. Dr. Chris Gabbler, Assistant Professor with the University of Texas Rio Grande Valley Biology Department, is hopeful the rare Lomas can be saved as more than 95 percent of the Rio Grande Valley's native habitat has vanished. "People often think that nature and development are decidedly at odds, and that is not true. We can do this the right way. We can continue to grow our economy, grow our region, while we still respect and protect these last few gems," said Dr. Gabbler.

And the lovely Upland Loma (former planned Annova LNG site--defeated), looks like this:

Military is Open Burning PFAS, with EPA's Permission

In communities across the U.S. and its territories, the Departments of Defense and Energy routinely open burn and open detonate (OB/OD) countless tons of hazardous munitions wastes in the open air. It continues to be the "standard method for disposal because it is a technically simple method of disposal that is frequently the least expensive and easiest to perform." Despite the commercial availability of safer technologies, this devastating practice continues.

In addition to PFAS, toxic emissions from OB/OD include explosives, elemental metals (e.g., arsenic, cadmium, chromium, cobalt, lead and mercury), volatile and semi-volatile organics, polycyclic aromatic hydrocarbons, chlorinated dioxins and furans, and perchlorate. (Source: National Academy of Sciences, *Alternatives for the Demilitarization of Conventional Munitions,* 2019)

What are PFAS?
Per- and polyfluoroalkyl substances (PFAS) are a group of man-made chemicals that includes PFOA, PFOS, GenX, and many other chemicals. PFAS are used to make fluoropolymer coatings and products that resist heat, oil, stains, grease, and water. The best-known fluoropolymer is polytetrafluoroethylene or Teflon.

Why do some military munitions contain PFAS?
PFAS are added to improve the performance and stability of military explosives and munitions.

What happens to PFAS when subjected to open air burning?
PFAS are not destroyed in an open fire and are therefore dispersed to the air and the surrounding environment where they accumulate in people, as well as fish and wildlife. At higher temperatures, poisonous hydrogen fluoride gas may be generated. Hydrogen fluoride is a listed hazardous air pollutant subject to regulation by U.S. EPA and authorized states under the Clean Air Act.

What health risks are associated with exposure to PFAS?
PFAS have been shown to affect growth and development, reproduction, thyroid function, the immune system, injure the liver and increase risk for certain cancers.

EPA and Tennessee state regulators are both permitting the military to open burn munitions wastes containing as much as 15% PFAS at Holston Army Ammunition Plant.

CASE STUDY: Open Burning PFAS in Kingsport, Tennessee

Every year, Tennessee's Holston Army Ammunition Plant is permitted to open burn **1,250,000 pounds** of munitions wastes that may contain as much as **15% PFAS** by weight.

Why is the Army allowed to open burn PFAS and other toxic waste at Holston?
Both the U.S. EPA and Tennessee regulators recently re-issued permits allowing open air burning of wastes that contain PFAS and other toxic compounds. This burning has been going on for decades.

Can regulators prohibit the burning of PFAS and highly toxic wastes?
YES! At other military sites like the Blue Grass Army Depot in Kentucky, the military is prohibited from burning PFAS and dozens of other toxic wastes. Both Blue Grass and Holston are in EPA Region 4.

PFAS Content of Munitions Permitted for Open Burning
at Holston Army Ammunition Plant – Partial List

Munitions Item	PFAS (Fluoropolymer)	Percentage by weight	Principal Explosive Ingredient
HDX-106	Teflon ®	1.4	RDX
LX-04	Viton-A ®	15	HMX
LX-07	Viton-A ®	10	HMX
LX-10-2	Viton-A ®	5.4	HMX
LX-17	Kel-F ®	7.5	TATB
PBX-9502	Kel-F ®	5	TATB
PBXN-7	Viton-A ®	5	TATB/RDX
PBXN-5	Viton-A ®	5	HMX
PBXN-6	Viton-A ®	5	RDX
PBXW-14	Viton-A ®	5	HMX/TATB
unspecified	OXY-461 ®	*unspecified*	*unspecified*

Kel-F® is also called Neoflon®. RDX = Royal Demolition eXplosive. HMX = High Melting eXplosive. TATB = 1,3,5-Triamino-2,4,6-trinitrobenzene. OXY-461™= vinyl chloride-chlorofluoroethylene copolymer.

Matthew Tejada April 27, 2015
Director
Office of Environmental Justice
USEPA Headquarters
William Jefferson Clinton Building
1200 Pennsylvania Avenue, N. W.
Mail Code: 2201A

Washington, DC 20460

Dear Director Tejada:

The undersigned representatives of Ohio communities and environmental organizations are writing to you to ask your Office to intervene to prevent what we believe are clear environmental justice abuses cognizable under Executive Order 12898 being committed in our state.

These abuses are being committed in low income areas of Ohio under the authority of federal law contained in the Safe Drinking Water Act's ("the Act") Class II Underground Injection Control ("UIC") Program for injection wells disposing of oil and gas wastes through US EPA's remarkably inadequate delineation and oversight of the Class II UIC program delegated to the Ohio Department of Natural Resources ("ODNR"). In particular, U.S. EPA is tolerating undeniably inadequate public participation and enforcement in low-income, rural Appalachian areas of Ohio where injection wells are being sited rapidly and recklessly by ODNR pursuant to this federal delegation.

OVERVIEW OF THE SECTION 1425 CLASS II UIC PROGRAM IN OHIO

I. The Profoundly Weak Federal Requirements for Class II Injection Well Delegation under Section 1425 of the Safe Drinking Water Act.

Ohio shares the unfortunate status with twenty-two (22) other states[1] of having a delegated UIC program under the negligibly defined program created by Section 1425 of Part C of the Safe Drinking Water Act. Section 1425 was adopted when the Act was amended in 1980 and created a sweetheart program for the oil and gas industry that allows states to receive federal delegation under a streamlined program with minimal safeguards. However, U.S. EPA's errors and neglect in implementing the Section 1425 program have greatly multiplied Congress's lapse in enacting Section 1425 initially.

These implementation failures are plainly evident in the fact that US EPA has *never adopted regulations* governing the delegation process under Section 1425 in the thirty-four (34) years since its enactment. In the absence of any rules, US EPA has instead ostensibly implemented the program pursuant to a minimally defined guidance document that was

[1] http://www.epa.gov/ogwdw/uic/pdfs/Delegation%20status.pdf

adopted in 1983.[2] This simplistic guidance has *never been amended* in the thirty-one (31) years since its adoption. We are not aware of any current efforts at US EPA to either adopt regulations to implement Section 1425 or to update the 1983 guidance.

The Agency's failure to provide basic regulatory definition for Section 1425 delegation at the federal level has not been remedied by its state level implementation in Ohio. Unlike the more rigorous, initial form for federal program delegation under Sections 1421 and 1422 of the Act, the Section 1425 program allows states to regulate Class II wells using their own program requirements rather than following minimum EPA regulations. Ohio received its Class II UIC delegation in 1983[3] and this delegated program is being implemented pursuant to a simplistic, twelve-page Memorandum of Agreement between Region V and ODNR that was signed in March, 1984. That agreement has not been substantially amended in the thirty (30) years since its initial adoption.

US EPA's failure to responsibly implement Section 1425 is evident from the mere recitation of the facts in the preceding paragraphs. This history of failure has no parallel in any other program administered by US EPA. The dismay and shock of Ohio citizens facing environmental harm from Class II injection wells when they first become aware of these elemental facts regarding this so-called "regulatory program" under Section 1425 is almost impossible to describe to you.

II. The Federal 1425 Program Has Ignored the Fundamental Changes in the Injection Well Program Caused by Horizontal Shale Wells, also known as "Fracking."

As recounted above, the federally delegated program requirements for Class II underground injection control in Ohio have been frozen in place for over three decades at a nominal level. As you are no doubt aware, the scope and environmental risk of oil and gas field waste disposal has certainly not stayed remotely as static.

In Ohio, horizontal shale drilling, better known as "fracking," has completely transformed the oil and gas industry in the last four years. In that time, Ohio has gone from no horizontal "fracked" wells to 584 in production by the end of the second quarter of 2014.[4] The amount of oil and gas production wastes generated by horizontal fracked wells is dramatically greater than previous production wells with each fracked well requiring an average of 6.5 million gallons of water and chemicals to frack initially.[5] It also appears that most, if not all, of these wells will be "re-fracked" one or more times to remain productive with a corresponding generation of millions more gallons of highly contaminated, toxic fracking wastewater for each well. Estimates of the amount of this contaminated water that "flows back" to the surface as waste that must be then be disposed range from 30% to 70%.[6]

[2] http://www.epa.gov/ogwdw/uic/pdfs/guidance/guide_uic_guidance-19_primacy_app.pdf
[3] 49 FR 46896
[4] http://oilandgas.ohiodnr.gov/production
[5] Figure generated from data in Fracfocus, at http://fracfocus.org/.
[6] U.S. Department of Energy, Office of Fossil Energy and National Technology Laboratory, *Modern Shale Gas*

All of this disposal in Ohio takes place in injection wells authorized pursuant to the token Section 1425 delegation scheme.

To meet this radically increased disposal volume, the number of Class II injection wells permitted in Ohio has grown from 144 to 240 over the past four years. Over that same time, the quantity of wastewater disposed in Ohio injection wells has increased from 12,597,115 barrels in 2011 to 24,911,564 barrels in 2014.[7] This quantity will significantly increase as horizontal well drilling matures in Ohio through current efforts to increase pipelines and mid-stream facilities.

It is not this radical increase in overall volumes of wastes disposed in Ohio Class II wells alone that confronts Ohioans, but also the greater toxicity of the waste fluids caused by the mass use of chemicals in the hydraulic fracturing process. Nothing in US EPA's Section 1425 program addresses the unique public health and environmental risks of the fracking chemicals being injected here. Also, Ohio does not even require the chemicals used to be disclosed until sixty (60) days after drilling operations are completed, see Ohio Revised Code 1509.10(A). As a result, these chemicals will not be publicly disclosed until after the contaminated flowback wastewater has been injected, which leaves the affected community with absolutely no opportunity for knowing what is being injected through and beneath their water supplies. Further exacerbating this lack of public health disclosure under Ohio law, the General Accounting Office recently reported[8] that Ohio is the only state of eight major oil and gas producing states studied that requires absolutely no testing of the chemical contaminants in oil and gas wastes to be injected before permitting injection wells.

Compounding this problem are radical and unprecedented "trade secrecy" protections for fracking chemicals. In Ohio, a well owner now unilaterally chooses whether it will declare any of its chemicals as "proprietary" and does not even have to disclose the chemical's risks to any office of the state government, thereby effectively concealing the risks presented by these chemicals, see Ohio Revised Code 1509.10(I). Due to these state provisions - which no federal requirement in the delegation process even addresses - the federally approved Class II program in Ohio utterly fails to address these massive amounts of haphazardly identified toxic chemicals. Historically, such miserable or corrupt policy decisions at the state level have been prevented by minimum federal requirements. However, that fundamental safeguard is simply absent in the 1425 program in Ohio.

In addition to chemical wastes, flow back from these deep wells is also routinely contaminated by radioactive materials that present an entirely new set of serious environmental and public health dangers. Nothing in the Section 1425 program addresses these radioactive wastes as well.

Development in the United States: A Primer, DE-FG26-04NT15455, April 2009, p. 66, http://fossil.energy.gov/programs/oilgas/publications/naturalgas_general/Shale_Gas_Primer_2009.pdf.

[7] Numbers compiled from fee reports filed by injection well operators with ODNR.

[8] GAO-14-857R, Drinking Water: Characterization of Injected Fluids Associated with Oil and Gas Production, Sept. 23, 2014.

As the federal Section 1425 program has been frozen in place for three decades and has not seen the slightest change in response to any of these new dangers caused by horizontal shale drilling, we believe you can readily understand why the public distrust of the federally delegated Class II program is deep and red hot in this state.

Because of the lack of program definition and modernization, the Class II injection well programs delegated by US EPA under Section 1425 are open invitations to persistent environmental injustice. As the next section explains, that open invitation has been fully exploited in Ohio's Class II UIC program and has caused disproportionate impacts on low-income rural Appalachian communities in our state.

ENVIRONMENTAL JUSTICE IMPLICATIONS

The Class II UIC Section 1425 program in Ohio has public participation requirements so inadequate that they would be comical but for the profound injustice they cause, provides unreliable and inadequate information to the public on the environmental risks inherent in the waste injection program, and has a long-standing history of federal oversight that is too weak and indifferent to ensure meaningful enforcement at the state level where low-income communities are put at risk. These considerations are directly related to your Office's responsibilities under Executive Order 12898. Also, since the sole federal 1425 delegation guidance document of 1983 and the Memorandum of Agreement of 1984 have both remained stagnant since their adoption, these policies in no way reflect the environmental justice considerations that were adopted in Executive Order 12898 in 1994. Given their outdated, neglected status, it is no surprise that the Section 1425 program does not address the disproportionate impacts of Class II injection well disposal on Ohio's low-income, rural Appalachian communities.

I. Disproportionate Impacts on Ohio's Low-Income Appalachian Communities.

The federal Appalachian Regional Commission has officially recognized thirty-two (32) Ohio counties[9] as being part of Appalachia due to their history of geographical isolation and economic depression. These thirty-two counties comprise 36% of Ohio's eighty-eight counties. Based on the most recent (July, 2009) figures from the Appalachian Regional Commission,[10] these 32 Appalachian counties are the home for 17.4% of Ohio's total population.

Based on the Data Sheet of December 1, 2014, prepared by the ODNR's Division of Oil & Gas Resources Management, Ohio, copy attached, ODNR has permitted 237 total Class II injection wells statewide, of which 199 are currently injecting waste. Of these 199 active wells, 149 – or 74.9% of the total - are located in Ohio's thirty-two Appalachian counties.

[9] Adams, Ashtabula, Athens, Belmont, Brown, Carroll, Clermont, Columbiana, Coshocton, Gallia, Guernsey, Harrison, Highland, Hocking, Holmes, Jackson, Jefferson, Lawrence, Mahoning, Meigs, Monroe, Morgan, Muskingum, Noble, Perry, Pike, Ross, Scioto, Trumbull, Tuscarawas, Vinton, and Washington. See http://www.arc.gov/counties and also Ohio Revised Code 107.21

[10] http://www.arc.gov/reports/custom_report.asp?REPORT_ID=40

As for the remaining thirty-seven (37) permitted but not-yet active wells, these are identified in ODNR's December 9, 2014, map entitled "Class II Brine Injection Wells of Ohio," copy attached. This map identifies an additional fifteen wells in Ohio currently being drilled and an additional twenty-two (22) wells in Ohio that have permits but have not been drilled. Of the fifteen wells being drilled, all of them, or 100%, are in Appalachian Counties (Ashtabula, Trumbull, Mahoning, Tuscarawas, Guernsey, Monroe and Meigs Counties) and 9 of the 22 permitted wells are in Appalachian counties for 40.9%% (Trumbull, Mahoning, Tuscarawas, Harrison, Muskingum, Guernsey, Noble and Washington Counties). For total wells permitted in Ohio (237), 173 are in Ohio's 32 Appalachian counties, or 73%.

As the Appalachian 17.4% of Ohio's population (and 36% of counties) possess almost three-quarters (74.9%) of Ohio's active Class II injection wells and 73% of all permitted wells, it is clear that ODNR's injection well program has a disproportionately high and adverse environmental impact on Ohio's low-income communities. ODNR's Class II injection well program is therefore well within the parameters of Executive order 12898 which requires federal agencies to develop environmental strategies to address disproportionately high and adverse health and environmental impacts on low-income populations.

This disproportionate impact should be a particular concern to your office. As you are aware, the EPA Office of Environmental Justice defines environmental justice as the:

> "fair treatment of people of all races, cultures, income and educational levels with respect to the development, implementation and enforcement of environmental laws, regulations and policies. Fair treatment implies that no population of people should be forced to shoulder a disproportionate share of the negative environmental impacts of pollution or environmental hazards due to a lack of political or economic strength." Federal Actions to Address Environmental Justice in Minority Populations and Low-Income Populations (59 FR 7269, February 16, 1994).

To meet this goal, the Order imposes the mandate that:

> "[E]ach Federal agency shall make achieving environmental justice part of its mission by identifying and addressing, as appropriate, disproportionately high and adverse human health or environmental effects of its programs, policies, and activities on minority and low-income populations in the United States and its territories..."

Environmental justice considerations are a particularly appropriate inquiry in the permitting and enforcement of Class II injection wells because Ohio's low-income Appalachian communities are disproportionately subject to serious environmental risks from these injection wells. These risks range from spills of toxic (including radioactive) fracking fluids, contamination of water supplies by fracking fluids through inappropriate geological siting or well failures, earthquakes, and the leakage or exposure to harmful volatile chemicals from open pools storing fracking waste. Nearby communities experience the brunt of these risks while receiving no economic benefit.

Appalachian communities also possess comparatively fewer emergency response forces, many of whom are volunteers with inadequate training and equipment for the largely unknown risks involved. As noted above, large scale fracking waste disposal involves exotic new chemical compounds of untested toxicity and whose identity is frequently concealed by Ohio's adoption of an extremely one-sided law in 2012 that singled out the fracking industry to obtain nearly unrestrained concealment of the identity of these chemicals under the guise of "proprietary information," see Ohio Revised Code 1509.10(I).

These serious local problems are all exacerbated by US EPA's failure to implement the Section 1425 delegation program effectively. When US EPA approved Ohio's primacy over Class II injection wells, it is apparent that it failed to consider the undue burden that would ensue to low income Appalachian communities. In the twenty years since Executive Order 12898 was adopted, those policies were never amended to address the environmental justice implications of the weak Section 1425 program. With the advent of widespread horizontal shale drilling, the impacts from that series of defaults are now greatly magnified and pose a direct threat to Ohio's low-income Appalachian communities which US EPA has yet to recognize or address.

As provided in Executive Order 12898, US EPA must adopt strategies designed to insure that these Ohio communities are provided with:

1) ample access to relevant and reliable information on injections wells,
2) meaningful opportunities for public participation in their permitting process, and
3) effective enforcement of this federally delegated programs.

As the following sections make clear, none of these environmental justice goals are being met in the Appalachian areas of Ohio that are being disproportionately impacted by the Section 1425 injection well program.

II. Inadequate Public Notice and Public Participation

Although adequate public notice and public participation are hallmarks of the Federal Government's commitment to environmental justice, they are barely present in the formal Section 1425 program and even that marginal situation is deteriorating rapidly in Ohio. US EPA's guidance barely addresses these issues (at pages 21-22), but even those minimal requirements have been all but ignored by ODNR without consequence.

The public notice and participation provisions in Ohio are contained in Ohio Administrative Code ("OAC") Section 1501:9-3-06(H). Paragraph (H)(1) provides that public notice of the filing of an injection well permit shall be provided by the publication in a newspaper of general circulation in the affected county for not less than five consecutive days and in a weekly circular available to county engineers. A copy of that notice is also to be provided to all "owners or operators of wells" within either a one-quarter or one-half mile radius around the proposed injection well – although to no members of the public living within that radius. Paragraph (H)(2) then provides that comment may be provided by the public *on the application* (i.e., not on the draft permit) *within 15 days* of the last day that the notice was published in the local newspaper. If any such comments are made, then the chief of the ODNR division must rule on the "validity" of the comment, and in the event he finds it valid, the individual commenter alone (not the public in general) may attend a hearing with the chief on the objection. While the Ohio Administrative Code provides for publication in a newspaper of general circulation in the affected county, this did not hold true in a recent permit issued by ODNR for an injection well in Monroe County, Ohio. The well was being converted from a production (dry) to a class II injection well and even though the well is located in Monroe County the public notice appeared in Washington County.

This process clearly does not constitute "meaningful" public notice and participation as required in Executive Order 1298. Indeed, this system has no practical use at all to the people of Ohio, especially in light of how it has been implemented at ODNR. This is because:

First, the strongest part of the system, the newspaper notice, is unlikely to result in effective communication of the pending application to the people of the affected region. It is considerably weaker than the state's own system of public notice of oil and gas production well permits in urban areas where notice must be provided to any homeowner within 500 feet of the well as well as to local mayors and township trustees, see Ohio Revised Code 1509.06(A)(9).

Second, there is no reliable method by which a potential commenter can obtain the application before the expiration of the token 15 day comment period. While the newspaper notice is to indicate how more information can be found from the applicant or ODNR, there is no mechanism for insuring that the application will be provided in a timely fashion. No duty is placed upon the applicant to provide the application and it is the experience of the undersigned that ODNR is currently taking over two months to respond to public records requests. Most potential commenters would arrive at the expiration of the 15 day period with nothing in hand to comment upon.

Third, the only record available during the comment period might be the application but there would be no permit to give context to the application. Issues for comment generally arise once a potential commenter sees how the state is addressing potential problems in an actual permit document. Accordingly, under this system, comments are reduced to guesswork and the chance that the chief would subsequently find those comments "valid" for convening a hearing becomes a slim possibility – even if the chief approached those determinations in good faith, see below. Indeed, this system is the only federal program that we are aware of where comments are to be provided when a draft permit is lacking and it appears designed to make those comments immaterial.

Fourth, a fifteen day comment period is grossly inadequate on its face in light of the complex geologic, hydrogeologic, and engineering issues presented by injection well permits. A commenter would be required to hire professional experts to analyze the application which cannot be done in such a restricted timeline. Also, the ODNR regulation allows no discretion for an extension of this minimal period. No other program delegated by US EPA has a 15 day comment period.

Fifth, in practice, the ODNR chief is routinely not finding comments "valid" and is therefore routinely refusing hearings requested by the public. The most notorious example is from Athens County in late 2013 where over a hundred comments were submitted to ODNR requesting a public hearing, a request even reiterated by the local county commission. The request was denied without explanation – even though the well application involved was proposing the largest disposal volume in Ohio. The Athens County Commission subsequently convened its own public hearing on the injection well at which over a hundred people attended in an attempt to cover US EPA's delegated agency's default. To our knowledge, no public hearing has been conducted by ODNR on an injection well anywhere in Ohio.

Sixth, ODNR's public "participation" process with the chief allowing a public hearing only after unilaterally screening comments and finding them "valid" without explanation, is far weaker than the system outlined in US EPA's 1983 guidance document. Paragraph e.2.A on page 22 provides that "The State program should provide opportunity for a public hearing if the Director finds, based upon requests, a significant degree of public interest." If this standard were in place in Ohio, a public hearing as requested by the people of Athens County described in the previous paragraph would certainly have been held. However, ODNR has not been challenged by US EPA for this significant variation from the guidance that has led to public participation in Ohio becoming illusory. This incident raises a serious question whether this guidance, that forms the only basis for delegation of the Section 1425 program, is taken seriously at all within the implementing agencies, including by U.S. EPA itself

Seventh, ODNR also refuses to follow the 1983 guidance – and again without any challenge from US EPA – by ignoring Paragraph e.3 on page 22 that "The final State action on the permit application should contain a 'response to comments' which summarizes the substantive comments received and the disposition of the comments." To our knowledge, only a nominal number of responsiveness summaries have ever been prepared by ODNR, perhaps only one.

Eighth, ODNR has within the past year unilaterally changed its permit issuance program for Class II injection wells in a very fundamental way that clearly exposes that agency's contemptuous disregard for meaningful public participation. Until approximately a year ago, ODNR issued a single permit to each injection well to which the public notice by newspaper notice and its vestige of a potential public hearing applied, and most importantly, to which appeal rights for affected persons attached. However, in 2010, the Ohio legislature abrogated long-standing Ohio law that universally allowed permit appeals in another sweetheart statute for the oil and gas industry by exempting drilling permits for production wells in Ohio Revised Code 1509.06(F), later confirmed by the Ohio Supreme Court in *Chesapeake Exploration, LLC v. Oil & Gas Commission, et al.,* 135 Ohio St.3d 204, 985 N.E.2d 480, 2013-Ohio-224 (Jan. 30, 2013).

At some unknown point within the past year, ODNR – without any public announcement and without even changing its permit regulation in Ohio Administrative Code 1501:9-3-06 – surreptitiously altered its permit scheme into a bifurcated system with an initial drilling permit, which it now claims is not appealable pursuant to Ohio Revised Code 1509.06(F), and a second and clearly ministerial permit that authorizes the commencement of actual disposal after the well passes a fifteen minute pressure test.

The artifice behind this new system is that, because the Supreme Court case expressly held in the *Chesapeake Exploration opinion* that "injection well" permits were appealable, ODNR calls this second ministerial permit the appealable "injection well" permit while the far more important provisions of the now purportedly unappealable initial permit, where all the critical geological and engineering determinations are found, is to be immune from public review. In taking this position, ODNR is acting as if it is oblivious to the facts that the second permit has no public notice or comment opportunities whatsoever, and that, as the issuance of the second permit is never publicly noticed to the affected public, the public cannot as a practical matter appeal the permit before the jurisdictional thirty (30) day appeal deadline in Ohio Revised Code 1509.36 expires. On June 12, 2014, the Ohio Oil & Gas Commission that hears these permit appeals upheld this new, bifurcated system and handed ODNR its goal of immunity from public accountability in its Section 1425 program when it dismissed an appeal of the critical "initial" permit, copy of opinion attached. That regrettable decision is now under appeal, but the scenario dramatically demonstrates the extent and underhanded nature to which ODNR is willing to go in order to avoid meaningful public participation and accountability and perpetuate environmental injustice to the disproportionately affected, low-income residents of Appalachian Ohio.

Although Region V has been made aware of this change, it has made no public effort to restore the public's rights to meaningfully participation in injection well permitting decisions in their communities. This situation reveals that the environmental justice goal of meaningful public information and participation is openly scorned in the implementation of Ohio's Section 1425 program. The success of this artifice to date is only possible because the 1983 guidance does not insure public appeal rights in the delegated program, instead only providing the vacuous statement on page 21 that: "It is assumed that most States already have legislation that governs public participation in State-decision making and defines such processes as appeals, etc." Allowing critical program components to be determined merely by content-free statements like this underscore the previous observation in this letter that the Section 1425 program is an open invitation to environmental injustice.

III. Inadequate US EPA Oversight of a Failed State Enforcement Program

ODNR's enforcement program has no priority, is minimal in effort, and is clearly inadequate to deter violations at the state's Class II injection well sites. Its principal civil enforcement tool is contained in Ohio Revised Code 1509.04(G) which authorizes a referral to the Ohio Attorney General or local prosecuting attorney for injunctive relief and, pursuant to Ohio Revised Code 1509.33, for civil penalties ranging from $2,500 to $20,000 per violation. Ohio Revised Code 1509.99 adds criminal penalties to the possible penalties following a formal request from ODNR. However, no such requests have been made by ODNR for either civil or criminal prosecution under this authority. The ODNR program does not have authority to impose administrative fines independently.

Ohio Revised Code Section 1509.04 also provides ODNR with wide-ranging authority to suspend or revoke injection well permits. We are aware of only a single incident where this authority has been used by ODNR against an owner or operator of an injection well which was the novel episode of an injection well owner in Youngstown who was criminally prosecuted by U.S. EPA for illegal waste dumping into a sewer

The only enforcement related activity that we are aware of is that inspectors may identify violations at injection well sites and record the fact in their inspection reports, but their only action taken in regard to these violations is to notify the operator and ask them to remedy the violation with no sanctions attached. Whether the violations are resolved is seldom documented in Department files. We also have noted a pattern that injection well sites where local citizens or newspapers actively follow a well's status are inspected periodically, but sites without focused public interest (which are the vast majority of sites) are likely to be ignored by ODNR inspectors. Such a minimal enforcement effort is incapable of deterring violations and has created a climate where violations are tolerated and their significance studiously minimized by the political leadership at ODNR.

There are two particular circumstances that highlight this phantom enforcement program at ODNR that leaves Ohio's Appalachian communities disproportionately at risk. A frequently noted violation at injection well sites is that the annulus, or open space within the well casing surrounding the injection tube, is not kept at a pressure greater than the pressure in the injection tube itself. The purpose of this requirement is that the greater annulus pressure will assist in limiting any leak in the injection tube and force the leaked fluid down into the injection zone. A simple review of ODNR's on-line database, copy attached, shows that this requirement is routinely violated with 3,944 formally recorded incidents of annulus pressure being less than injection pressure since 2001. No enforcement action has been taken for any of these nearly 4,000 violations. As a result, Ohio's Appalachian communities are being exposed to a preventable risk of contamination.

Another chronic area of non-compliance is ODNR's avoidance of its obligation to require the closure and plugging of non-functioning or abandoned wells of all kinds (including production wells that are not governed by the UIC program, but which allow a pathway for contamination from leaking injection wells). ODNR is required to obtain the plugging of these wells after they are taken out of use to limit their potential for rapidly spreading groundwater contamination. However, ODNR appears to be avoiding this important obligation through a de facto policy of declaring that only wells "incapable of use" due to deterioration or damage, are to be considered abandoned, even if they have not been used for an extended number of years and there is no intent ever to use them again. Also, even when "plugging orders" are issued by ODNR, they are not followed up upon to enforce compliance but rather are reissued indefinitely or the matter is just dropped.

This lack of enforcement was documented in a June 1, 2014 story in the *Columbus Dispatch* titled "Oil, gas wells often keep operating despite violations," which found that many wells cited for violations have remained in violation for years without any follow-up action. The Department's explanation for its inaction is noteworthy: that many of these violations are "a low priority" because "they are idle wells that need to be permanently closed." In other words, they are wells that the state should be requiring to be plugged but the Department concedes it has simply given up on that duty. The Department justifies this stance by saying simply that these open wells "probably pose no imminent environmental threat." This failure to enforce the plugging requirement is also a concern for injection wells, because according to ODNR's December 9, 2014, map of injection wells, only two are classified as plugged, even though many of these wells have been in operation for decades and are aged.

One clear reason for the inadequate enforcement program is that the ODNR program is notoriously understaffed. ODNR's Oil and Gas program, of which the UIC program is a part, has suffered devastating staff decreases since 1987 when it had 124 full-time equivalents to a low of 35 in the late 2000's before the fracking boom in Ohio. The program's enforcement program during the 1980's was handled by a specialized unit organized specifically to meet the unique demands of enforcement; this unit was eliminated during these cuts and has not been restored leaving ODNR with no oil and gas program staff dedicated exclusively to enforcement. During this same period, the UIC program staff was reduced from 20.85 full-time equivalents to just 3.18. The current staffing needs of the UIC program seem to be met by sharing staff from the larger program rather than having its own dedicated staff.

The larger oil and gas program has slowly restaffed over the past four years to approximately 120 staff members. However, only 50 of those positions are for inspectors that are critical for maintaining an enforcement program. Even ODNR admitted publicly over two years ago that it needed 90 inspectors, or almost double its current number, see *Cleveland Plain Dealer*, May 10, 2012, "Drilling Inspectors needed: Ohio looks to hire as shale play spreads to more counties." In this article, ODNR announced it would meet this staffing goal by early 2013, but it has added only twenty additional inspectors since then, not the sixty it said are needed. ODNR further admitted in this article that it had only inspected 18% of the state's operating wells (of all kinds) in 2011, leaving more than 50,000 unchecked that year.[11] This rate was the lowest of four major producing states reviewed in the article (Pennsylvania, Texas, Colorado and Oklahoma).

[11] This number cited in the *Plain Dealer* refers only to actively operating wells and does not include inactive wells that are almost never inspected. There are approximately 150,000 such inactive wells in Ohio.

According to ODNR, only four inspectors are currently dedicated to the UIC program statewide. Most of the inspectors' time is spent on functions related to serving the industry's needs related to permitting, such as observing cement jobs and mechanical integrity tests, so that little time remains available to them for enforcement related activities. Other environmental justice related components of the ODNR program remain starved of staff resources and are given an even lower priority than inspectors, such as staff to respond to public records request within a reasonable time frame or to conduct the now non-existent public hearings or responding to public comments.

As will be discussed below, US EPA's periodic reviews of the Ohio Section 1425 program have flatly ignored these enforcement deficiencies and even the state's history of debilitating staffing problems. Accordingly, the environmental justice goal to "promote enforcement of all health and environmental statutes in areas with minority populations and low-income populations" is being ignored by US EPA in regards to Ohio's Section 1425 program.

IV. Regulatory Capture of Ohio's 1425 Program by the Oil and Gas Industry

The significance of the information above cannot be fully appreciated without your Office's consideration of a recent incident that exposes the underlying bias of Ohio's Class II UIC program as being implemented hand-in-hand with the oil and gas industry with an explicit goal of freezing out all expressions of legitimate public concern. The incident was the disclosure in February, 2014, of a ten-page "Communications Plan" dated August, 20, 2012, (copy attached) drafted by ODNR to influence Ohio public opinion to favor the Department's partnering with the oil and gas industry to "proactively open state park and forest land to horizontal drilling/hydraulic fracturing . . ." (p. 1).

A stated goal of this plan is to overcome "zealous resistance by environmental activist opponents, who are skilled propagandists," who are subsequently called "eco-left" pressure groups, see p. 1. These groups are later identified by name on page 5 under the heading "opposition groups" and includes virtually every group that works with Ohio communities in their concerns over fracking, including long-standing national organizations such as the Sierra Club and the Natural Resources Defense Council.

This public relations strategy presumes that absolutely all expressions of concern over the environmental risks created by fracking are illegitimate and unworthy of the Department's consideration. The strategy also identified the "allied" groups that ODNR will work together with on this campaign against the "eco-left" which includes the oil and gas industry's main lobbying and umbrella groups, the Ohio Oil and Gas Association and America's Natural Gas Alliance, and even singles out the Halliburton corporation as a cooperating partner with ODNR (p. 5). Subsequently disclosed documents also established that this one-sided, industry-friendly and public-adverse public relations program was discussed multiple times within the Governor's office itself.

Seldom does the public, or the responsible authorities at US EPA, obtain such a deep and utterly candid insight into the inmost motivations and mindset of its state agents responsible for implementing a federally delegated program. This document establishes ODNR's explicit intent to identify itself unreservedly with the oil and gas industry while placing itself in unyielding, reflexive opposition to citizen concerns with fracking. All expressions of concerns about the fracking industry or with ODNR's programs are, as a matter of express policy, to be dismissed as illegitimate "propaganda" and ruthlessly countered.

ODNR has confirmed for you that it will not consider the environmental concerns of Ohio's disproportionately impacted communities as worthy of consideration. The Department's policies to repress public information and public participation, and avoid exposing the industry to accountability for violations, are direct extensions of this underlying policy. Usually your Office must infer the presence of environmental injustice from surrounding circumstances, but ODNR's public relations strategy document has established its intention to systematically deny environmental justice to Ohio's Appalachian communities as an admitted fact.

V. US EPA's Oversight of ODNR's Section 1425 Program is Woeful

The two main documents for US EPA oversight of this state of affairs in an annual report from ODNR to Region V and periodic audits by the Region conducted approximately every five years. Attached is ODNR's annual report for federal fiscal year 2010 covering its actions to meet its grant commitments. It is less than a page and a half. It provides the barest summary of the program's enforcement activity, makes no reference to any public participation or outreach activities of any kind, and makes no reference to the overriding program limitation of the UIC's staffing level at that time (barely over 3 full-time equivalents). This report was filed before the advent of fracking in the Midwest that subsequently caused the UIC programs' permitting activities to radically increase. It effectively communicates the superficial level of accountability that Region V required of ODNR's Section 1425 program.

The audits conducted by Region V of ODNR's program were even more superficial, however. Attached are the last two audits conducted in 2005 and 2009 of some dozen pages each. About all your Office needs to know about these audits is that it is readily apparent that the 2009 audit is a mere cut-and-paste from the 2005 audit with negligible changes in wording and none at all in conclusions and in effusive, fact-free praise. There is no critical attitude expressed at any point in these audits nor is there any statistical analysis to justify its overwhelmingly rosy assessments. There is not a statement in either audit that ODNR would feel the slightest discomfort with or any need to contest.

From an environmental justice perspective, the audits show no awareness of the objectives of Executive Order 12898. Only the 2009 audit contains a reference to public participation activity which is a single public meeting in Ashtabula County which does not even appear related to a permitting process. The 2005 audit mentions only a single odd incident of the state responding to a citizen complaint but nonetheless both audits jump to a conclusion that the program's responsiveness to the public is "effective." The enforcement discussion praises the Division, especially for its use of computerized record keeping and "field presence" (2005 audit, p. 6), but glosses over the fact that the Division has never made an enforcement referral and only uses its weakest forms of enforcement through mere violation notices issued by inspectors with questionable deterrent effect. To cap off the audit's efforts to avoid even the mention of unpleasant facts, only the 2005 audit even mentions the program's critical staffing crisis in stating that its "suggests DMRM consider hiring additional support staff and entry-level technical staff to assist the UIC program" (p. 12) without even mentioning that the program's staffing had been slashed from 20 to 3; the 2009 audit ignores this fundamental problem entirely.

A cursory review of the audits is all that is needed to demonstrate their superficial, unrestrained "gushy" quality and their failure to reflect the environmental justice considerations of Executive Order 12898. These audits were before the advent of fracking in Ohio that radically increased the Ohio public's demands for accountability from the UIC program. These audits demonstrate US EPA's failure to comply with Executive Order 12898's requirements for environmental justice in regards to the Section 1425 UIC program in Ohio.

One final episode will provide the capstone for demonstrating Region V's feckless lack of oversight of its delegated UIC program in Ohio. Hearing the frustration from Ohio citizens arising from the utter lack of public participation in ODNR's injection well program, the Buckeye Forest Council, the Ohio Sierra Club and the Center for Health, Environment and Justice sought to compensate by holding public hearings in Portage and Athens Counties to allow citizens to bring forth their complaints on dealing with ODNR and the injection wells in their communities. At the end of the two public hearings, these groups sent all comments and accompanying documents to US EPA, Region V in September, 2013. To date, no response whatsoever has been received from Region V on this information that corresponds to the very heart of the environmental justice considerations embodied in Executive Order 12898.

CONCLUSION

The undersigned representatives of the Ohio environmental community ask your Office to investigate US EPA's failure to protect the nation's environmental justice goals for Ohio's low-income, Appalachian communities facing a disproportionate risk from Class II injection wells. To be very frank, Ohioans need your help desperately to install the values of environmental justice into Ohio's Section 1425 UIC program. We will cooperate with your Office in this investigation and urge you to conduct it promptly.

We are confident that your investigation will result in findings documenting US EPA's failings and recommendations to bring the implementation of this rogue program back within its federally required duties and obligations to low-income Ohioans. Your assistance in insuring that the people of Appalachian Ohio are accorded their full portion of human respect and protection envisioned by Executive Order 12898 is most appreciated by ourselves and by countless thousands of our fellow Ohioans.

Thanking you in advance,

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On behalf of the following

Greg Pace - Guernsey County Citizens Support on Drilling Issues. Guernsey County, Ohio
Christine Borello - IEL Superfund Site & Stark County Concerned Citizens, Stark County, Ohio
Rev. Monica Beasley-Martin - Defenders of the Earth Outreach Mission. Mahoning County, Ohio
Betsy Cook - Southeastern Ohio Fracking Interest Group. Washington County, Ohio
Heather Cantino - Buckeye Forest Council. Statewide
Joanne Gerson - Southwest Ohio No Frack Forum. Hamilton County, Ohio
Andrea Reik - Athens County Fracking Action Network. Athens County, Ohio
Carol Apacki - Licking County Concerned Citizens for Public Health & Environment. Licking County, Ohio
Diana Ludwig- Frackfree America National Coalition. Mahoning County, Ohio
Susie Beiersdorfer -Frackfree Mahoning Valley
Lynn Anderson - Guardians of Mill Creek Park. Mahoning County, Ohio
John Williams - We Are Not Expendable. Trumbull County, Ohio
Vanessa Pesec - Network for Oil and Gas Accountability and Protection. Lake County, Ohio
Nick Teti - Coshocton Environmental and Community Awareness. Coshocton County, Ohio
Jill Hunkler - Concerned Barnesville Area Residents. Belmont County, Ohio
John Morgan - Ravens Rock, Inc. Belmont County, Ohio
Susan Jennings - Arthur Morgan Institute for Community Solutions. Green County, Ohio
Loraine McCosker - Appalachian Ohio Sierra Club. Athens County, Ohio
Mike and Ruth Partin - Concerned Citizens of Sycamore Valley. Monroe County, Ohio
Gwen Fisher - Concerned Citizens Ohio. Portage County, Ohio
Mary Greer- Concerned Citizens Ohio-Shalersville. Portage County, Ohio
Rachael Belz - Ohio Citizen Action. Hamilton County, Ohio
Caitlin Johnson - Communities United for Responsible Energy. Cuyahoga County, Ohio

Cc: Interagency Working Group on Environmental Justice, Washington D.C.
Gina McCarthy, Director, US EPA
Susan Hedman, US EPA Region V

Munitions Constituents as Emerging Contaminants and IRIS Update

Anita K. Meyer DABT

Toxicologist

Environmental & Munitions CX

Huntsville Engineering and
Support Center

April 9, 2014

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US Army Corps of Engineers
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Emerging Contaminant Watch List

- 1,4-dioxane
- **Antimony**
- *Cadmium and Compounds*
- *Cobalt and Compounds*
- Decabromodiphenyl ether
- Diisocyanates
- **2,4-Dinitroanisole (DNAN)**
- **Dinitrotoluene (DNT)**
- Dioxins
- *Manganese and Compounds*

Emerging Contaminants Watch List (cont.)

- Nanomaterials – carbon
- Nanomaterials-metals
- *Nickel*
- N-nitrosodimethylamine (NDMA)
- **5-Nitro-1,2,4-triazol-3-one (NTO)**
- **Perchlorate**
- Perfluorooctane Sulfonic Acid (PFOS)
- Perfluorooctanoic Acid (PFOA)
- N-Propyl Bromide
- Trichloroethylene (TCE)
- *Tungsten and Alloys*
- *Vanadium*

Emerging Contaminants Action List

- **Lead compounds**
- Chromium VI
- Beryllium
- Naphthalene
- **Cyclotrimethylenetrinitramine (RDX)**
- Sulfur Hexafluoride (SF6)
- Phthalate Esters

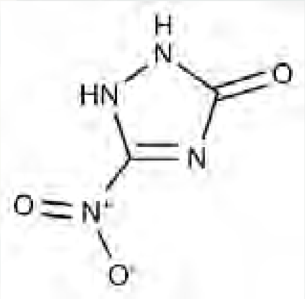
DNAN and NTO; Basis for Inclusion on EC Watch List

- Insensitive munitions replacements for RDX and TNT, for example IMX 101, IMX 104, OSX-12, PAX 21, and PAX 48
- Currently not highly regulated
  - TSCA limits discharge
  - Army Institute of Public Health has recommended occupational exposure levels (OEL)

Studies Ongoing by DOD to Understand Toxicity, Fate & Transport

Initial findings:

- DNAN likely less toxic than TNT
- NTO -
  - Male reproductive impacts
  - Low bioaccumulation potential
  - Has capacity to migrate



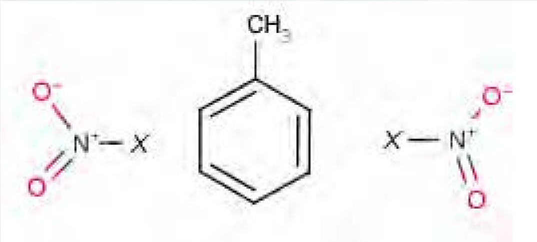
Phase I Impact Assessments

- DNAN will be performed fourth quarter FY14
- NTO Results:
  - DoD is using NTO and insensitive munitions containing the compound responsibly.
    - Health and safety protocols are in place at production and Load-Assemble-Pack (LAP) facilities
    - Waste streams carefully controlled.

Phase I Impact Assessment

- Identified risks associated with NTO:
  - Improve wastewater treatment systems to accommodate greater nitrogen load from increased production
  - Occupational – current environment exposure well below the proposed OEL of mg/m^3, but as operations move to full-rate production, there may be increased exposure

NTO remains on the Watch List.



Dinitrotoluene

- New screening provisional peer reviewed toxicity value issued by EPA for technical grade DNT
- The mixture is assumed to be 76% 2,4-DNT and 19% 2,6-DNT, the remaining 5% of the mixture is assumed to be a mixture of 2,3-, 2,5-, 3,4- and 3,5-DNT

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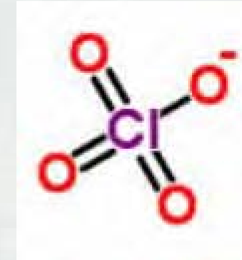
Dinitrotoluene

- EPA Regional Screening Levels for technical grade DNT are 3x higher than 2,6-DNT
- Values not published for 2,3-, 2,5-, 3,4- and 3,5-DNT separately; screening level applied to total DNTs
- DNTs are regulated as totals by State of Wisconsin: 0.05 µg/L

Perchlorate



- Moved from EC Action List to Watch List several years ago
- Reference dose published in EPA's Integrated Risk Information System (IRIS) 2005
- Several States have MCLs; Federal MCL under development
  - ►CA MCL is 6 ppb; in 2012 released a public health goal of 1 ppb

Perchlorate MCL Timeline

- 2005 - National Research Council: *Health Implications of Perchlorate Ingestion,* establishes reference dose (RfD) for perchlorate
- Oct. 2008 – EPA published preliminary determination not to regulate perchlorate
- Jan. 2009 – EPA published interim health advisory level of 15 µg/L
- Aug. 2009 – EPA published health reference levels for various life stages of 1 to 47 µg/L for comment
- Feb. 2011 – EPA published final determination to regulate perchlorate

Perchlorate MCL Timeline

- May 2012 – EPA Science Advisory Board (SAB) publishes advice on development of the MCL
- Sept. 25, 2012 – EPA shares information regarding analytical methods and treatment technologies in public meeting
- ~~Feb. 2013~~; ~~Dec. 2013~~ – Statutory deadline to publish MCLG and MCL proposal
- ~~Aug. 2013~~; Aug. 2014– Statutory deadline to publish final MCL

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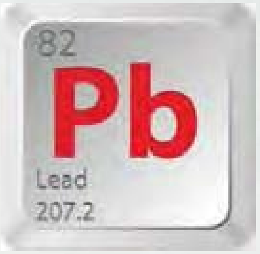
Major Conclusions in Draft SAB Advisory Report

- Sensitive Life Stage
  - ► Change from NRC 2005: From pregnant women with hypothyroidism to hypothyroxinemic pregnant women (and their fetus/infant)
- Epidemiology and Biomonitoring Data
  - ► Insufficient but meta- or pooled analysis might provide important information
- PBPK Modelling
  - ► Integrate mode of action

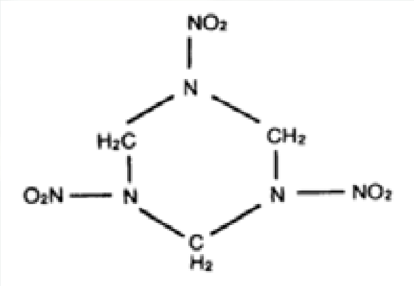


Lead

- Center for Disease Control (CDC) now uses a reference level of 5 µg/dL blood lead level (BLL). Is based upon upper 2.5% tile of 1-5 year olds tested in the U.S.
- Previously 10 µg/dL BLL was a "level of concern"
- EPA may adopt the new value; associated soil level may be 150 mg/kg

RDX

- DoD invested in RDX toxicity research that has been published and will be used in the ongoing IRIS reassessment of its toxicity.
- Drinking water level of concern 0.61 µg/L based upon cancer effects. Newer data suggest that a cancer value may not be warranted.



RDX

- Data supporting a change in the cancer toxicity value
  - ► Large body of evidence suggest it is not genotoxic or mutagenic
  - ► Two year rat study was negative
  - ► No human data link RDX to cancer
- Neurotoxicity or prostate inflammation will likely drive the noncancer reference dose

IRIS Update

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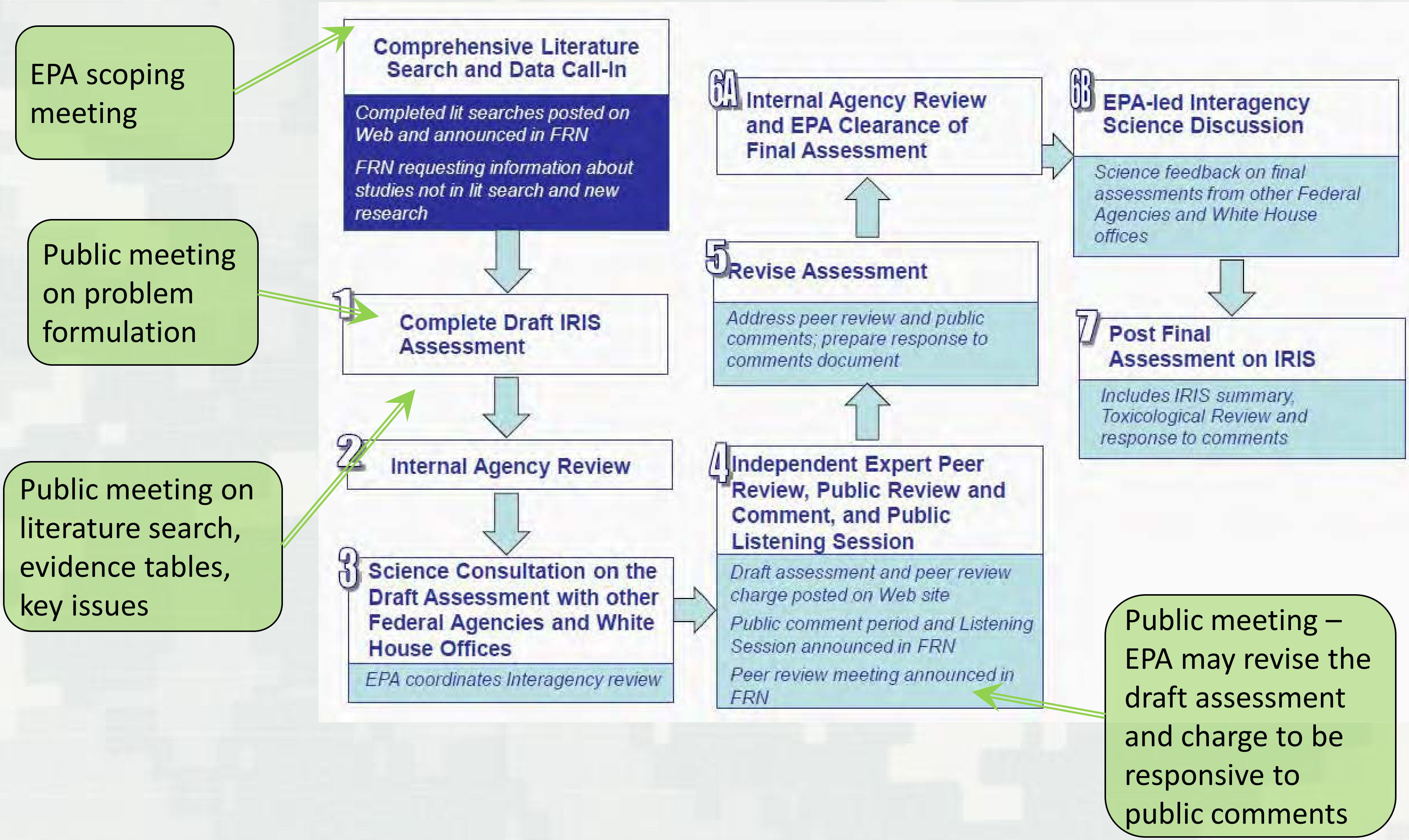
IRIS Process Steps with Enhancements



D607

Current Chemicals of DoD Interest Undergoing Assessment

- RDX
- Diethyl Phthalate
- Hexabromocyclododecane (HBCD)
- Inorganic Arsenic
- Benzo(a)pyrene (BaP)
- Hexavalent Chromium
- Trimethylbenzenes

B(a)P Undergoing Public and Peer Review

- New dermal slope factor, noncancer oral and inhalation toxicity values; existing oral slope factor was modified
- Inclusion of dermal SF decreases soil RSL by 28x
- New inhalation RfC yields an RSL equivalent to the 10^{-6} -based RSL, includes a developmental endpoint.

Risk-Based Screening Levels*		
	Existing	Proposed
Resident Soil (mg/kg)	0.015	0.1
Resident Water Use (µg/L)	0.003	0.002
Resident Air (µg/m^3)	0.00087	0.002

*Using EPA Regional Screening Level Calculator; basis 10^{-6} risk goal.
Basis of existing air RSL CalEPA inhalation unit risk.

Health Effects Documents for PFOA and PFOS

Developed by EPA Office of Water

- Chronic noncancer reference doses developed for each chemical.

Basis for values:

- PFOA - Increased liver, kidney and spleen weight; developmental effects in rodents
- PFOS - Liver and developmental effects in monkeys and rodents
- Associations seen in human epidemiological data for some effects

Risk-Based Screening Levels		
	Water (µg/L)	Soil (mg/kg)
PFOA	0.31	1.2
PFOS	0.47	1.8

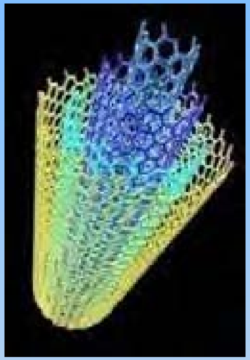
Current Provisional Short-Term Health Advisories:
PFOA – 0.4 µg/L
PFOS – 0.2 µg/L

Perfluorooctanoic acid (PFOA), Perfluorooctane sulfonate (PFOS)

Managing Chemical & Material Risks

DoD Emerging Contaminants Program
-Emerging Risks-





Briefing for EMDQ Workshop

Paul Yaroschak, P.E.
Deputy for Chemical & Material Risk Management
Office of the Deputy Under Secretary of Defense
(Installations & Environment)

D611

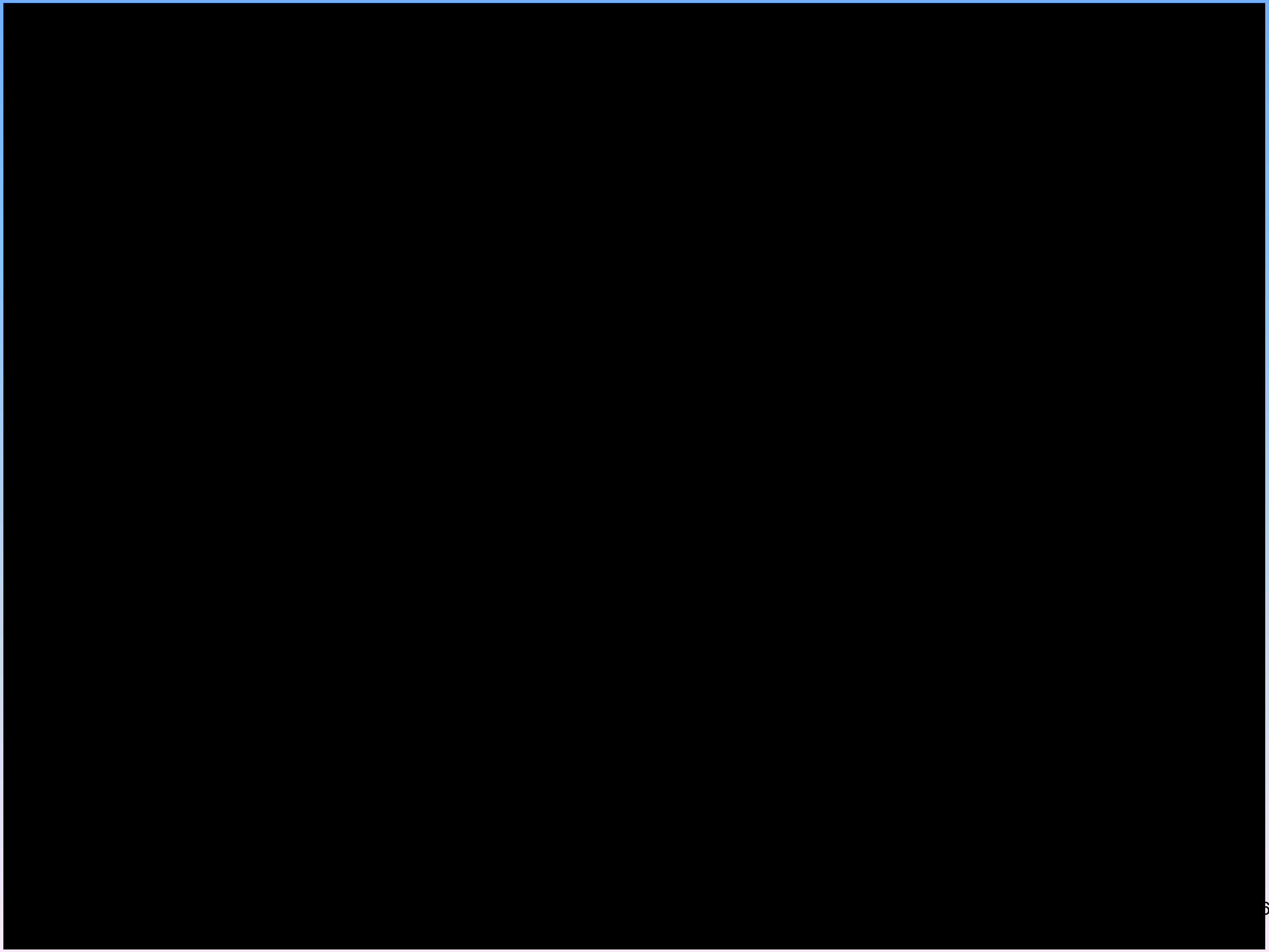
Part 1 – Context, Trends, & Emerging Contaminants (ECs) Program Refresher

Trends

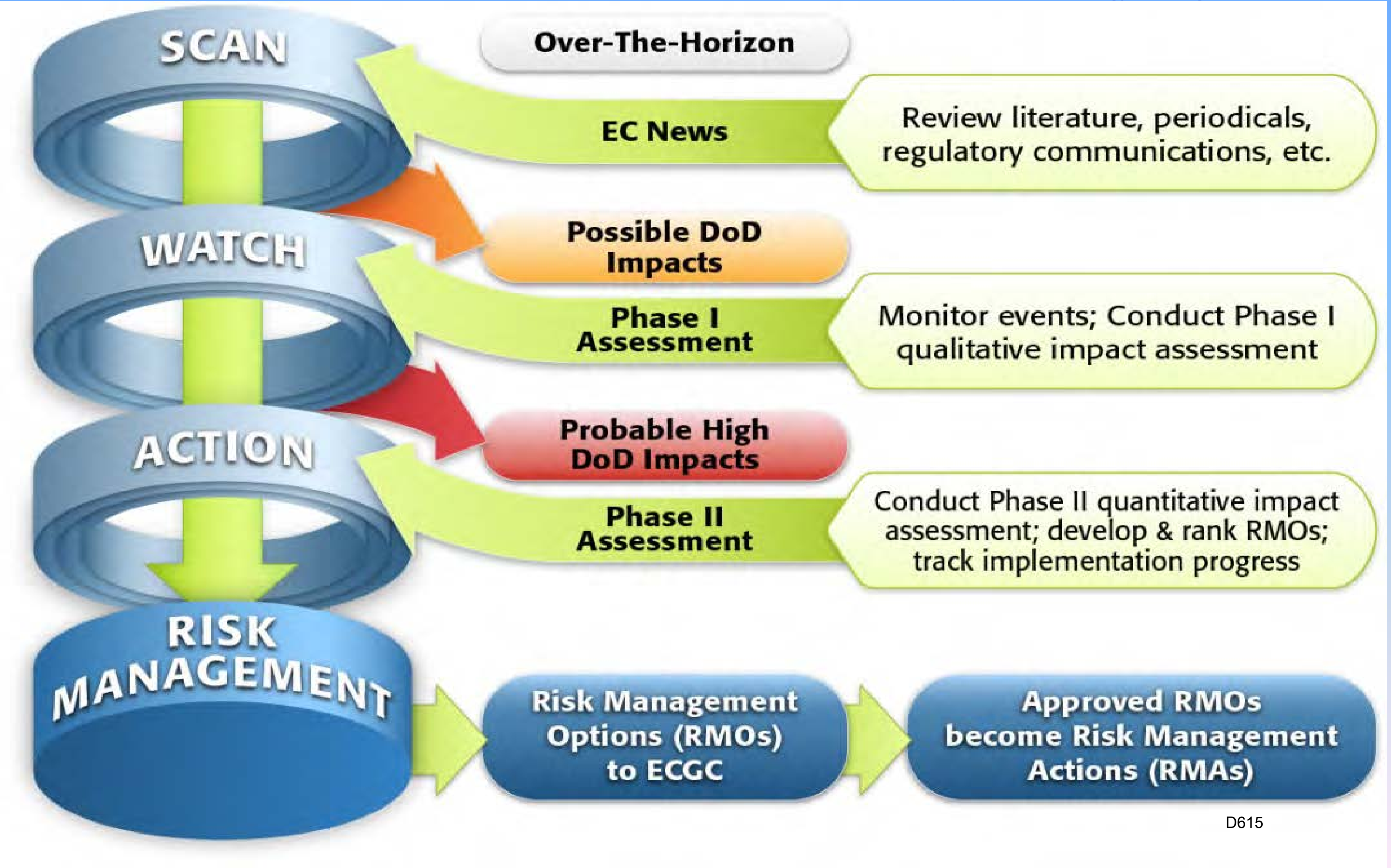
- **Use of Precautionary Principle**
  - Must understand health & environmental effects before using chemicals
- **Biomonitoring – What's showing up in humans?**
  - Centers for Disease Control's national bio-monitoring & California voluntary program
- **Evolving Risk Assessment Science & Process**
- **Strict Chemical Management & Green Chemistry**
  - Cradle to grave
- **International, Federal, & State Toxic Substances Laws**
  - Restrictions or banning of chemicals/materials (e.g., flame retardants)
  - California Green Chemistry Law & Proposition 65 Litigation
  - EU's REACH[1]
  - Pending TSCA[2] reform

[1] Registration, Evaluation, Authorization & Restriction of Chemicals
[2] Toxic Substances Control Act

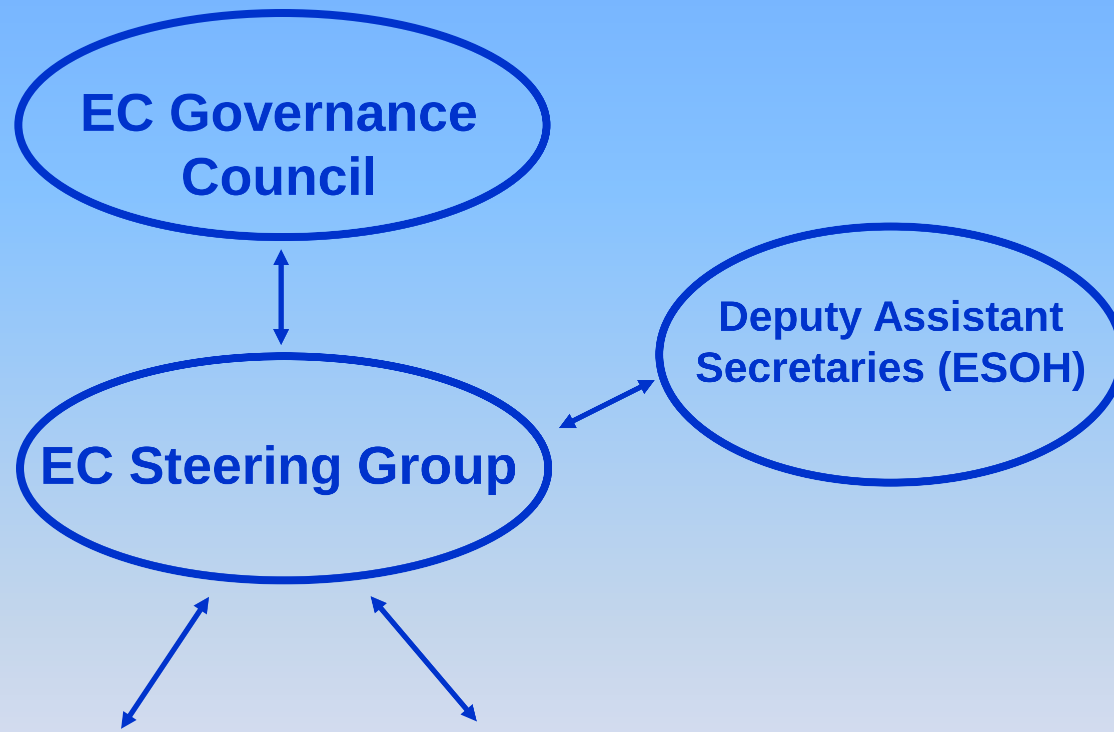
Automobile Industry Perspective (video)



EC "Scan-Watch-Action" Process



Program Governance

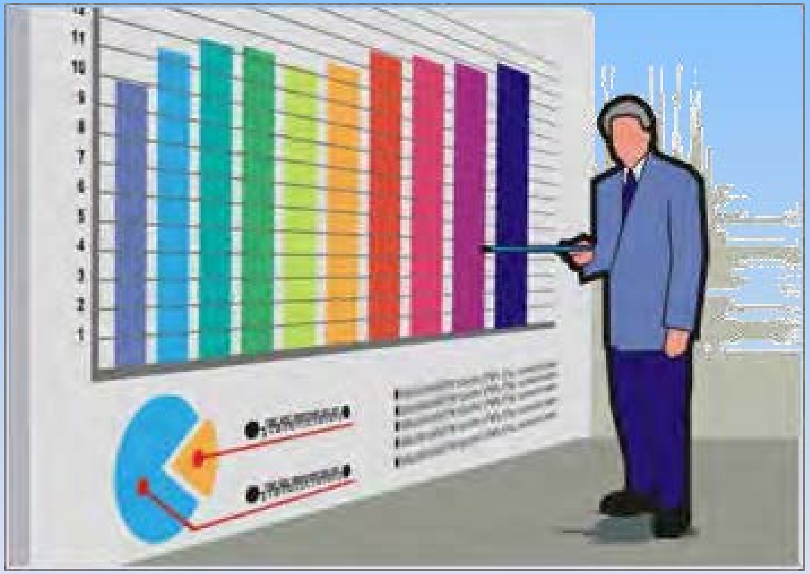


EC Subject Matter Experts, Working Groups & MERIT

D616

MERIT= Materials of Evolving Regulatory Interest Team – a virtual DoD-wide team

Part 2 – Progress Report

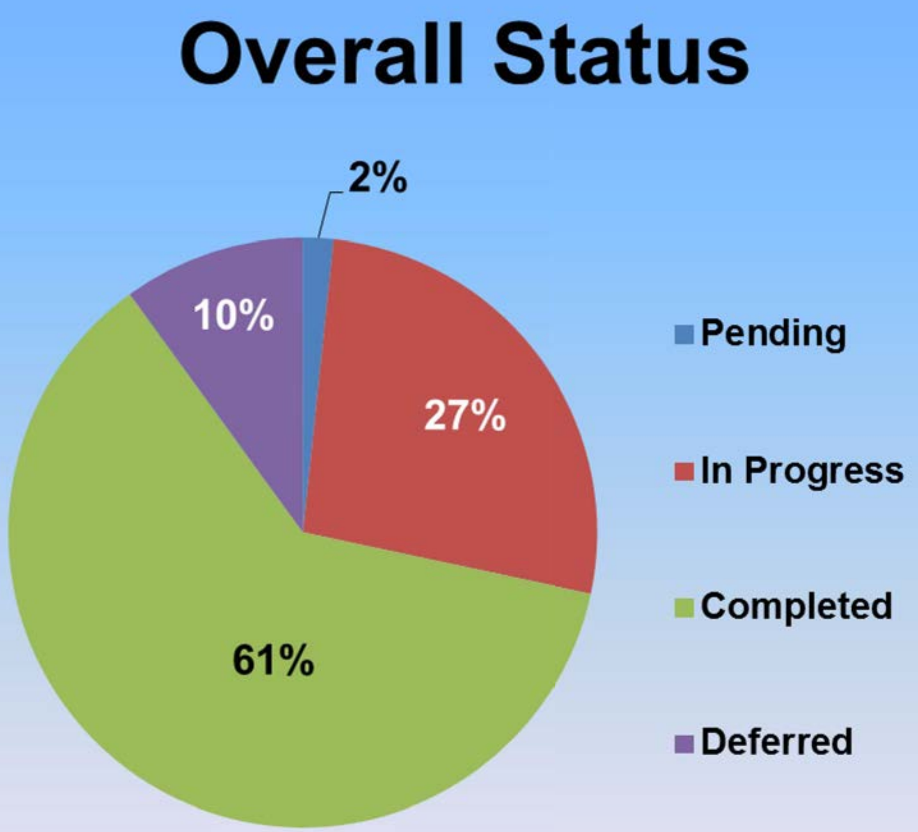


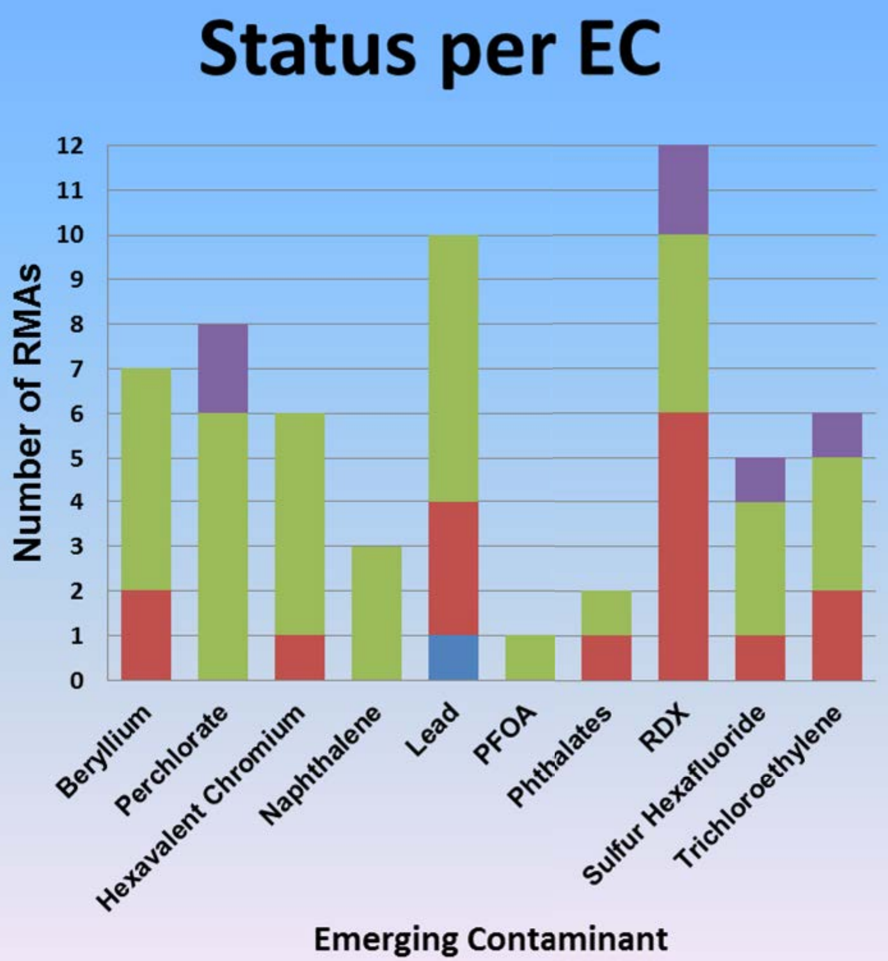
D617

Program Scorecard – Cumulative

- **Potential ECs screened --- over 500**
- **Phase I Impact Assessments completed --- 36**
- **Phase II Impact Assessments completed --- 10**
  - All current/former action list chemicals completed.
- **60 Risk Management Options (RMOs) developed & turned into Risk Management Actions (RMAs)**

Status-Risk Management Actions





D619

EC Watch List – April 2014

- ü Tungsten/alloys
- ü 1,4-dioxane
- ü Metal Nanomaterials
- ü Carbon Nanomaterials
- ü PFOS
- ü PFOA
- ü DNT
- ü Nickel
- ü Cadmium
- ü Manganese
- ü Dioxin

ü **Phase I Impact Assessment completed**

- Cobalt
- Antimony
- ü Diisocyanates
- ü TCE …moved from action list
- ü Perchlorate …moved from action list
- ü decaBDE
- ü Vanadium & compounds
- ü NDMA
- ü DNT
- • DNAN
- ü NTO

Energetic Compounds

Notes:

- Di-nitrotoluenes (DNT)
- Perfluorooctanoic acid (PFOA)
- Perfluorooctyl sulfonate (PFOS)
- decabromodiphenyl ether (decaBDE)
- 5-Nitro-1,2,4-triazol-3-one (NTO)
- N-Nitrosodimethylamine (NDMA)
- Trichloroethylene (TCE)
- 2,4 dinitroanisole (DNAN)

D620

EC Action List – April 2014

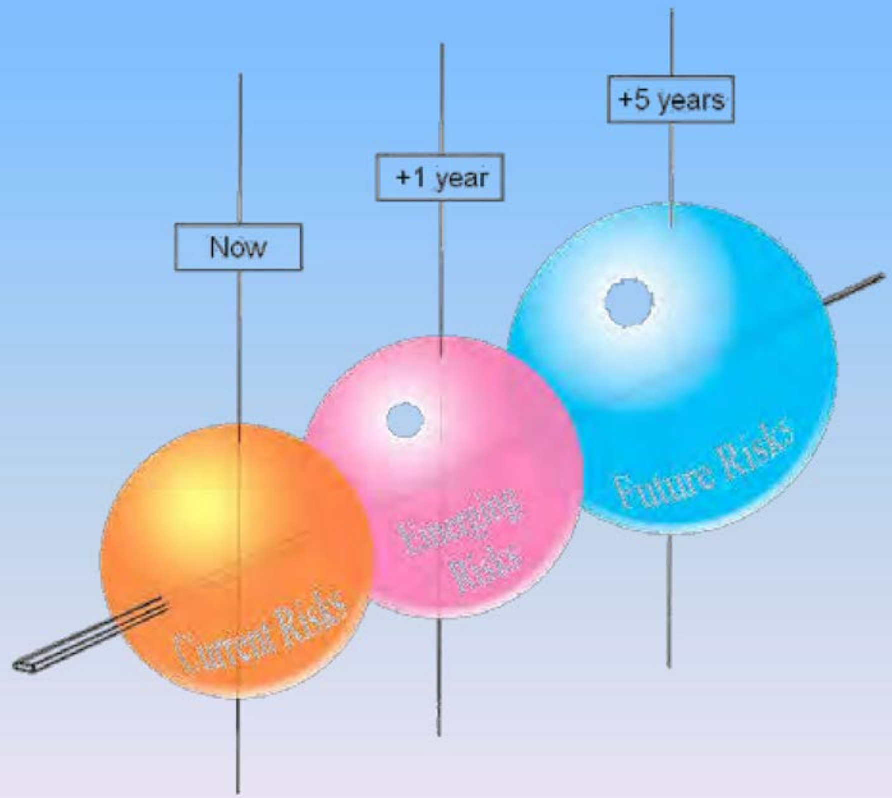
- Ü Royal Demolition eXplosive (RDX)
- Ü Hexavalent Chromium (Cr6+)
- Ü Naphthalene ...pending downgrade to watch list
- Ü Beryllium
- Ü Sulfur Hexafluoride (SF6)
- Ü Lead
- Ü Phthalates
- 1-Bromopropane ...pending ECGC approval

ü **Phase II Impact Assessment completed.**

RDX = Cyclotrimethylenetrinitramine

D621

Part 3 – Risks & Risk Management Actions



Key Risk Management Actions Completed

- Perchlorate RDT&E; Congressional Mythbusters brief
- Hexavalent chromium RDT&E; USD(AT&L) policy; Defense Federal Acquisition Regulation
- SF6[1] policy on capture & recycling
- Beryllium life cycle study
- Development of innovative naphthalene dosimeter for fuel handlers
- RDX[2] toxicological studies
- Coordination with PEO/PM for chem/bio protection equipment related to phase-out of phthalates

[1] Sulfur Hexafluoride [2] Cyclotrimethylenetrinitramine

Lead – Why on the Action List?

- **Evolving science & regulations may pose a risk to personnel & range operations…most munitions contain lead**

Range Instructor

- **Lead-free electronics pose a risk to DoD supply chain…short-circuiting in components**

D624

Lead Risk Management Actions

- **RDT&E on lead free munitions**
- **DoD-Industry Consortium on lead-free electronics**
  - **Develop technologies to detect lead-free circuit boards**
  - **Develop viable lead-free solders**
- **National Academy of Sciences Study**
  - **Concern: Lead exposures to personnel such as small-arms range instructors given new human health science**
  - **Conclusion:** **"**A review of the epidemiologic and toxicologic data allowed the committee to conclude that there is overwhelming evidence that the OSHA standard provides inadequate protection for DOD firing-range personnel and for any other worker populations covered by the general industry standard."
- **Underway: Development of DoD BLL standards**
  - **Surveillance & action levels**

Evolving Risks/Issues

- **New science on human health effects of ECs**
  - NAS[1] study for lead DoD concludes: "OSHA standard is inadequate to protect personnel"
  - NAS study has implications for range personnel; work group developing DoD-specific Blood Lead Level standards
  - Most munitions use lead; changes in air and groundwater standards have implications for range management

- **New explosive compounds are ECs (e.g., DNAN[2])**
  - Toxicology & fate/effects not fully understood; presents risks to ranges due to residual contamination
  - EC program conducting Phase I Impact Assessments to assess and mitigate risk

[1] National Academy of Sciences [2] 2,4-Dinitroanisole, an energetic compound replacement for TNT

Evolving Risks/Issues

- **Serious chemical availability issues with potential impacts on readiness…hard to quantify impacts**
  - Flame retardants: used in systems, platforms, equipment; phased out prematurely due to EPA pressure on manufacturers
  - Sulfur hexafluoride (SF6): a global warming gas, used in mission critical systems (e.g., AWACs aircraft; MK-50 torpedo; fire control systems); likely SF6 ban/restrictions; no substitutes on horizon for DoD applications
  - Phthalates: used as plasticizers; being phased-out; risks for DoD is use in DoD Chem/Bio protection equipment and munitions

- **Lack of supply chain visibility for chemicals/materials**
  - Difficult to assess risk and pinpoint risk management actions

D627

Department of Defense Emerging Contaminants Program

INNOVATIONS IN AMERICAN GOVERNMENT AWARD

Harvard University – Ash Institute for Democratic Governance & Innovation

D628

Backup Slides

Emerging Contaminants Program Genesis

- **~2004 – Perchlorate[1] detections in groundwater & drinking water cause national concern**
  - Disputes between DoD and regulators over response actions
  - Training/testing on 2 ranges curtailed
- **2005/6 – DoD forms EC Work group with EPA & Environmental Council of States**
  - EC Definition agreed & three policy papers developed
- **2008 – DoD creates EC funding line in FYDP**
- **2009 – DoD issues EC policy instruction**

[1] An oxidizer chemical found in munitions, pyrotechnics, and rockets

What is an Emerging Contaminant?

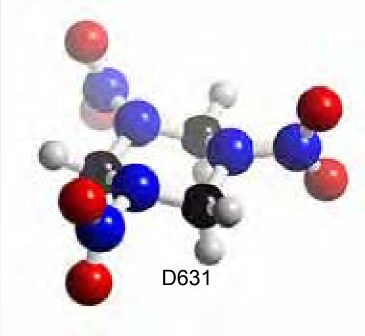
- Chemicals & materials that have pathways to enter the environment and present real or potential unacceptable human health or environmental risks…

and either

  - do not have peer-reviewed human health standards

or

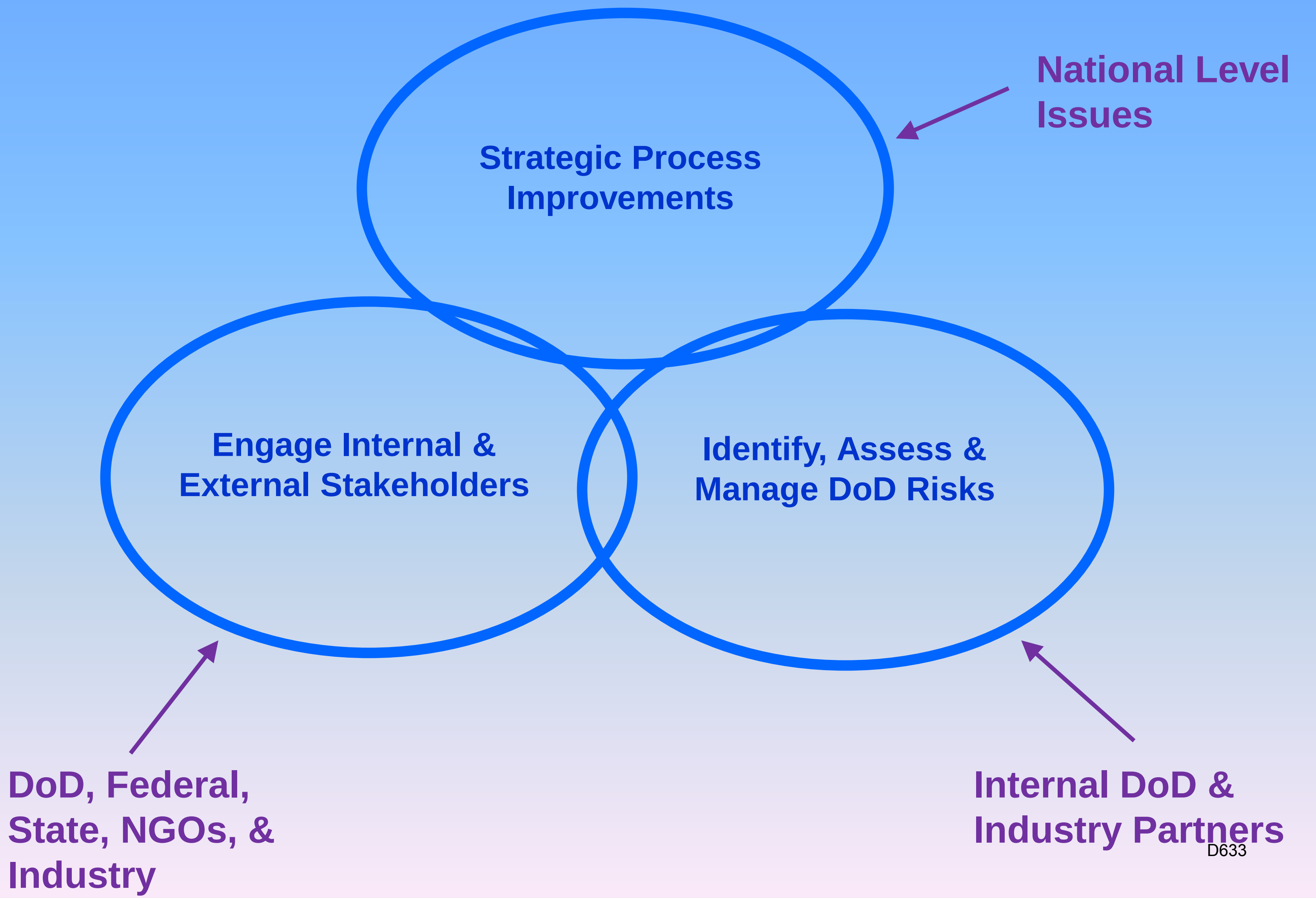
  - Standards/regulations are evolving due to new science, detection capabilities, or pathways.



How Can ECs Affect DoD?

- **Cause adverse health effects on operating forces, DoD employees, and/or public**
  - Human health protection paramount
- **Reduce training/readiness**
  - Restrictions on use of ranges
- **Restrict availability and/or cost of materials or chemicals**
  - Adverse impact on mission-critical applications & industrial base community
- **Increase O&M and/or cleanup costs**
  - Resource drain from mission needs

Program Strategic Priorities



Phase I Impact Assessment Completed

- ü **Perchlorate**
- ü **Hexavalent Chromium (Sept 2006)**
- ü **Naphthalene (Sept 2006)**
- ü **Trichloroethylene (TCE) (Oct 2006)**
- ü **1,2,3-Trichloropropane (TCP) (Nov 2006)**
- ü **n-Nitrosodimethylamine (NDMA) (Nov 2006)**
- ü **1,4-Dioxane (Dec 2006)**
- ü **Dinitrotoluenes (DNT) (Dec 2006)**
- ü **Perfluorooctanoic Acid (PFOA) (Jan 2007)**
- ü **Perfluorooctyl Sulfonate (PFOS) (Jan 2007)**
- ü **Polybrominated Diphenyl Ethers (PBDEs) (Jan 2007)**
- ü **Dioxins (Feb 2007)**
- ü **Tetrachloroethylene (PCE) (Feb 2007)**
- ü **Beryllium (Mar 2007)**
- ü **Lead (Mar 2007)**
- ü **RDX (Royal Demolition eXplosive) (Mar 2007)**
- ü **Tungsten (Mar 2007)**
- ü **Nickel (May 2007)**
- ü **Hexavalent Chromium (Jul 2007)**
- ü **Tungsten Alloy (Dec 2007)**
- ü **Sulfur Hexafluoride (SF6) (Jan 2008)**
- ü **Naphthalene (Apr 2008)**
- ü **Cadmium (May 2008)**
- ü **Lead (July 2008)**
- ü **Cerium (May 2009)**
- ü **Cadmium (Sept 2010)**
- ü **Dinitrotoluenes (DNT) (Jan 2011)**
- ü **Nanomaterials (Metal-Based) (Feb 2011)**
- ü **Manganese (May 2011)**
- ü **Diisocyanates (Jun 2011)**
- ü **Phthalate Esters (Jun 2011)**
- ü **Nanomaterials (Carbon-Based) (Nov 2011)**
- ü **Decabromodiphenyl Ether (Apr 2012)**
- ü **Vanadium and Compounds (Oct 2012)**
- ü **1-Bromopropane (1-BP) (Jan 2013)**

D634

This summary is for chemicals on which all three parts of a Phase I Impact Assessment were completed.

Phase I Impact Assessment Results Summary

Recommended for Watch List

- Cadmium and Compounds
- Cerium ***
- Cobalt and Compounds
- Decabromodiphenyl Ether (deca-BDE)
- Diisocyanates
- Dinitrotoluenes (DNT)
- 1,4-Dioxane
- Dioxins
- Manganese and Compounds
- Nanomaterials (Metal- and Carbon-Based)
- Nickel
- Perfluorooctyl Sulfonate (PFOS)
- Tetrachloroethylene (PCE) ***
- Tungsten
- Tungsten Alloy
- Vanadium and Compounds

Dropped After Phase I

- Dichlorobenzenes
- n-Nitrosodimethylamine (NDMA)
- Polybrominated diphenyl ethers (PBDEs)
- 1,2,3-Trichloropropane (TCP)

Recommended for Phase II / Action List

- Beryllium
- Hexavalent Chromium
- Lead
- Naphthalene
- Perchlorate *
- Perfluorooctanoic Acid (PFOA) **
- Phthalate Esters
- RDX
- Sulfur Hexafluoride (SF6)
- Trichloroethylene (TCE) **
- 1-Bromopropane (1-BP) *(proposed)*

Future Assessments (anticipated date)

- 1,4-Dioxane (Inhalation only) (TBD)
- n-Nitrosodimethylamine (NDMA) (TBD)
- 2,4-Dinitroanisole (DNAN) and 5-Nitro-1,2,4-triazol-3-one (NTO) (components in insensitive explosive formulations) (TBD)
- Cobalt (pending IRIS review) (TBD)

Determining Need for Phase I Assessment

- Antimony

D635

* Demoted to Watch List in September 2010
** Subsequent Phase II Impact Assessment recommended delisting from the Action List and adding to the Watch List
*** Regulatory developments supported delisting from the Watch List

Sulfur Hexafluoride (SF6) Background

- A non-flammable, non-toxic gas – no human health concerns
- Extremely stable, with excellent dielectric properties (electrical insulation and arc-quenching)
- A high global warming potential – 22,800 times more potent than carbon dioxide (CO_2) – long lasting in the atmosphere
- Average global SF6 concentration has increased by about 7 percent per year during the 1980s and 1990s

SF6 Commercial Uses

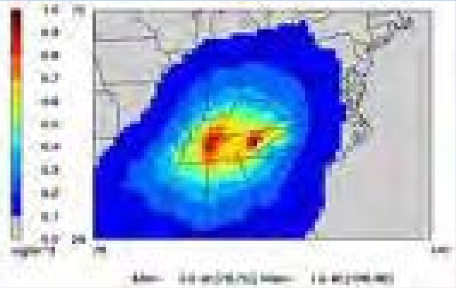
- High-voltage electrical switchgear & transformers
- High-energy imaging equipment
- Research - atomic particle tandem accelerators

D637

SF6 Military Uses

- Pressurization/dielectric for aircraft targeting pods/avionics -- Airborne Warning and Control System (AWACS) radar (e.g., E-3 Aircraft)
- Waveguide pressurization for shipboard targeting radar (e.g., MK 92 Fire Control System)
- Comprehensive Nuclear Test Ban Treaty monitoring and nuclear event detection
- MK-50 Torpedo propulsion






D638

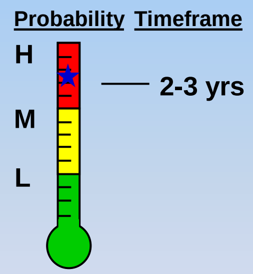
SF6 Phase I Impact Assessment

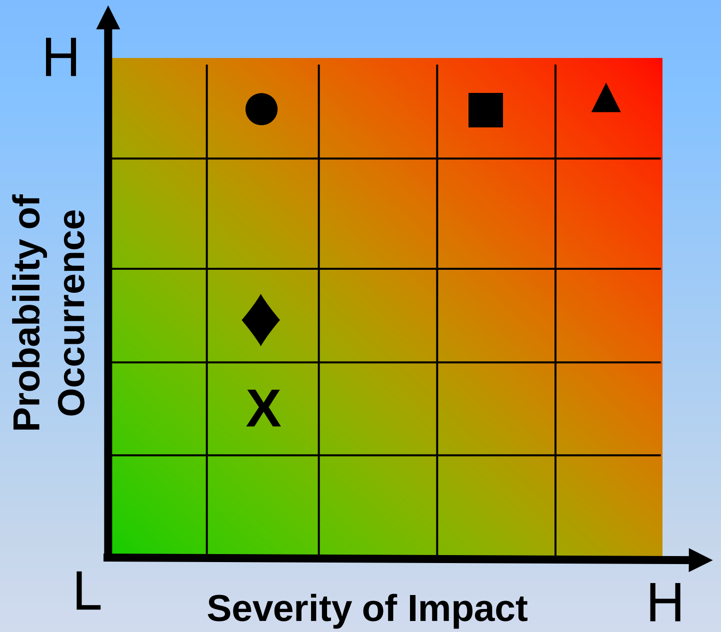
Completed January 2008

Sulfur Hexafluoride (SF6) is used in radar systems (e.g., AWACS aircraft); helicopter rotor-blade leak tests; discharge testing in fire suppression systems; electrical switch gear; and propulsion systems for specific weapons (e.g., MK-50 torpedo) in service and under design.

Likelihood of Toxicity Value/ Regulatory Change

1. **Probability that Greenhouse Gas emission initiatives will restrict use/availability of SF6**





- ♦ **ES&H**
- ■ **Training & Readiness**
- ▲ **Acquisition/RDT&E**
- ● **PO&MD of Assets**
- X **Cleanup**

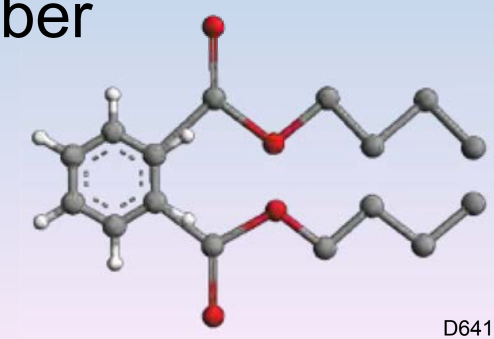
D639

SF6 Risk Management Actions

- **DoD Policy issued on SF6 capture & recycling**
- **RDT&E on substitutes for mission critical applications**
- **Coordination with Electric Power Research Institute on substitutes for electrical infrastructure**

Phthalates Background

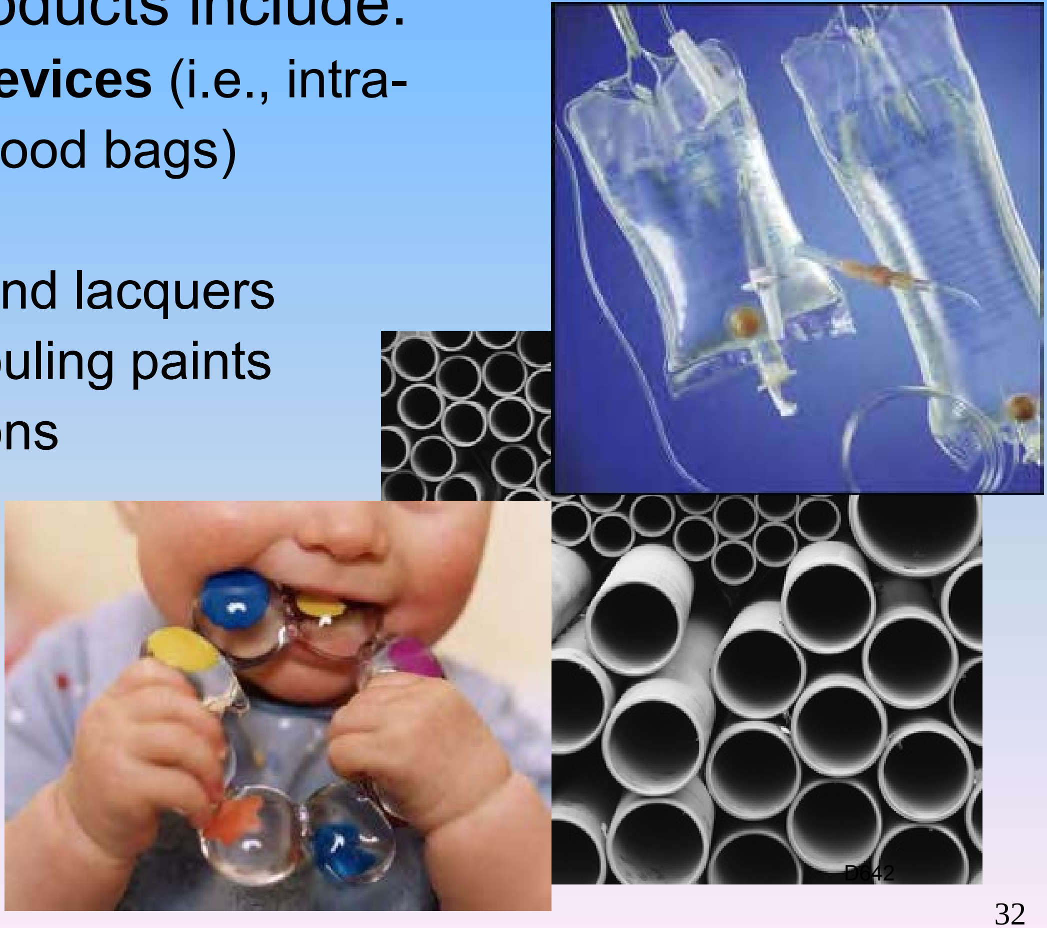
- Organic compounds derived from petroleum…phthalates are esters of phthalic acid
- Main uses:
  - Plasticizers to increase flexibility, durability and transparency of plastic products and to soften polyvinyl chloride (PVC) products
  - Solvents for oil-based dyes and nitrocellulose-based lacquers and coatings
- Due to their universally beneficial qualities, phthalates have found their way into a wide variety of consumer products
- Widespread human exposure…a number of phthalates appear in human biomonitoring surveys



D641

Phthalates Commercial Uses

- The specific phthalates in a product depends on the properties the phthalates impart and their cost
- Phthalate-containing products include:
  - **Medical supplies and devices** (i.e., intra-venous (IV) tubing and blood bags)
  - Dental materials
  - Paints, wood varnishes and lacquers
  - Anti-corrosion and anti-fouling paints
  - Wire and cable applications
  - Sealing compounds
  - Vinyl tile
  - Textiles and textile inks
  - Cosmetics
  - **Food packaging**



Phthalates Military Uses


- Solid rocket fuel binder
- Rocket motors & cartridges
- Plastics, rubber and vinyl components
- Wire insulation

- CBRN equipment (protective masks, gloves, boots, hoods) ??


D643

Phthalates Risk Drivers

- CPSC[1] assessments and/or EPA Chemical Action Plan (CAP) for phthalates may result in requirements to label, restrict, or ban specific phthalates
- Three phthalates[2] included on the *REACH Authorisation List* (Annex XIV) cannot be placed on the market or used after 21 July 2015 without authorization
- Bottom line: Production of certain phthalates discontinued in U.S.…additional suppliers may stop producing specialty phthalates critical to DoD applications
  - Time/cost intensive RDT&E needed for phthalate substitutes
  - Items made with new materials may require re-qualification

[1] Consumer Product Safety Commission

[2] BBP—Butyl benzyl phthalate; DEHP—Di(2-ethylhexyl) phthalate; DBP—Dibutyl phthalate

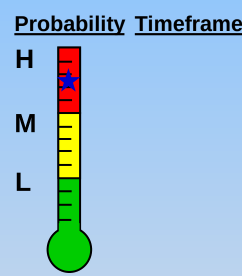
Phthalates Phase I Impact Assessment

Completed June 2011

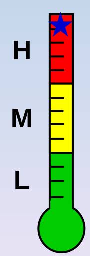
Phthalates are used as a plasticizer to create preferable physical properties in plastic products. Critical DoD phthalate-containing items include CBR equipment (protective masks, gloves, boots, hoods), propellant mixtures used in numerous munitions products, and a variety of sealers, paints, and resins.

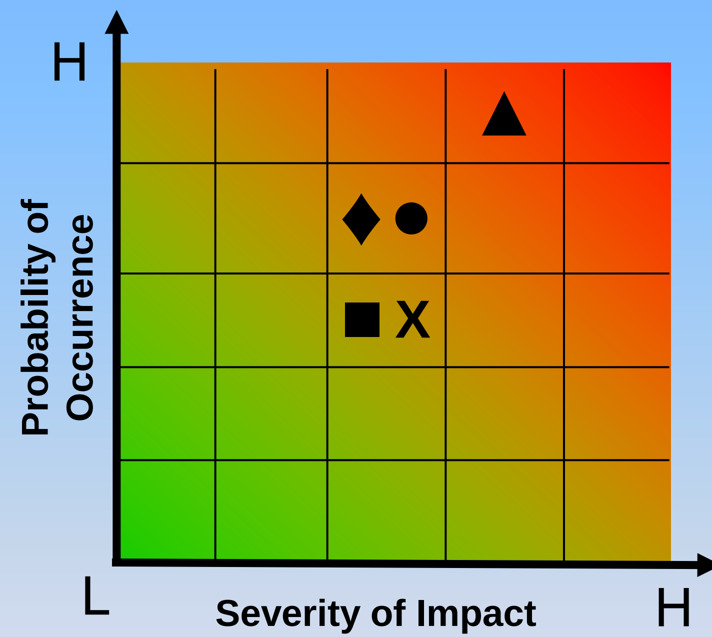
Likelihood of Toxicity Value/ Regulatory Change

1. **Probability that USEPA TSCA chemical management regulations will restrict use/availability of phthalates**



2. **Probability that EU REACH chemical management regulations will restrict use/availability of phthalates.**





- ♦ **ES&H**
- ■ **Training & Readiness**
- ▲ **Acquisition/RDT&E**
- ● **PO&MD of Assets**
- X **Cleanup**

D645

Phthalates Risk Management Actions

- **Issued early Risk Alert**
- **Completed Phase II Impact Assessment**
- **Worked with Joint Program Executive Office for Chemical & Biological Defense (JPEO-CBD) to minimize risk to protective equipment**
- **Issued Risk Memo to DoD Acquisition Executives**
  - **Locate critical applications requiring phthalates**
  - **Take risk management actions (e.g., testing substitutes)**

EC "Scan-Watch-Action" Process

Over -the- horizon

EC News

Review literature, periodicals, regulatory communications, etc.

Possible DoD impacts

Phase I Assessment

Monitor events; Conduct Phase I qualitative impact assessment

Probable high DoD impacts

Phase II Assessment

Conduct Phase II quantitative impact assessment; develop & rank RMOs

Risk Management Options (RMOs) to ECGC

Approved RMOs become Risk Management Actions (RMAs)

D647

decaBDE[1]
An Example of Material Availability Risk

- A **flame retardant** used in electronics, wire and cable insulation, textiles, automobiles & aircraft
- EPA: "Studies have shown that decaBDE persists in the environment, potentially causes cancer and may impact brain function."
- EPA & companies agree to **phase-out production** & sales for most uses 31 Dec 2012 & end all uses by end of 2013
- DoD, FAA, NASA, and industry believe phase-out is premature; substitutes not fully verified for performance or health risks

[1] decabromodiphenyl ether

Flame Retardants in Aerospace Products Have Increased Survivability

- **Assures safety in flight, if fire occurs**
- **Assures ability to escape, if aircraft crash occurs**
- **Meets FAA requirements**
  - 14 CFR Part 25 regulations:
    - Section 25.853, Compartment Interiors
    - Section 25.855, Cargo/Baggage Compartment
    - Section 25.856, Thermal/Acoustic Insulation
    - Section 25.869, Wire Flammability
    - Appendix F, Detailed Test Requirements
      - Materials and parts must successfully pass test/s in order to show compliance
      - Nine (9) different tests specified; some materials/parts must pass multiple tests
      - Variations of configurations require individual testing

2008 Continental Airlines 737
0 fatalities, 115 survivors

DecaBDE has become integral to meeting stringent aviation safety requirements

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DecaBDE is Used in Many Applications in Most Boeing Products

- Adhesives and Tapes
- Composites
- Ducting & Molded Parts
- Electrical/Electronics
- Emergency Equipment
- Fabrics & Films
- Insulation
- Interiors
- Sealants

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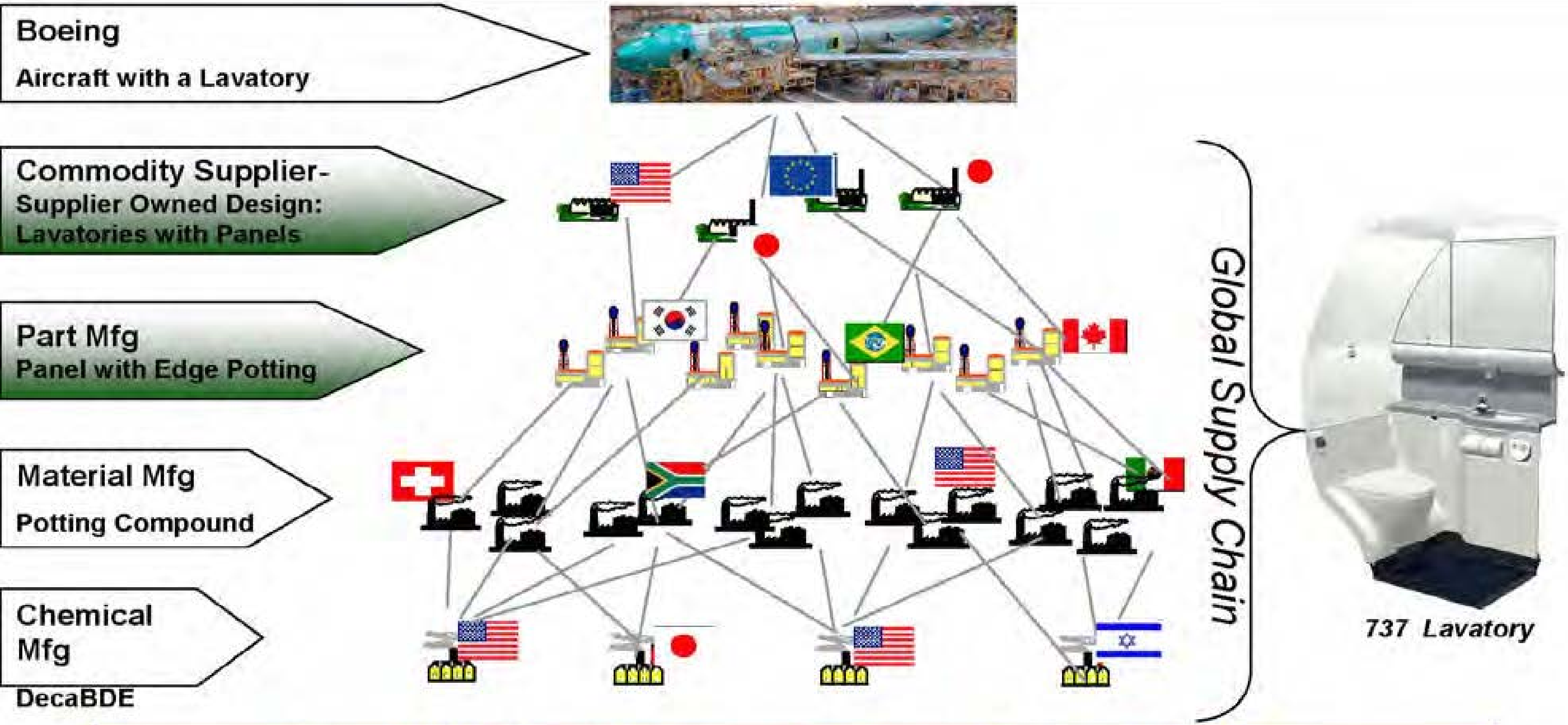
Boeing has a Global, Multi-Tiered Supply Chain



DecaBDE alternative manufacturers & compounders drive the replacement timetable

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The Boeing Company – Distribution to OIRA – 16 February 2011

D651

decaBDE Risk Management Actions

- **Issued "EC Risk Alert" to DoD & prime contractors**
  - **Difficult to locate decaBDE in supply chain**
- **Working with OMB, EPA, FAA, Aerospace Industries Association on reducing risk and evaluating substitutes**
  - Commented on EPA proposed rule on flame retardants
  - Possible joint testing on substitutes
- **Convened stakeholders roundtable on larger public policy issue of chemical phase-out process**

D652

Maxxam

Success Through Science®

PFOS, PFOA and Other Fluorinated Compounds:

Overcoming Sampling and Analytical Challenges

Author: Terry Obal, Ph.D., C.Chem.

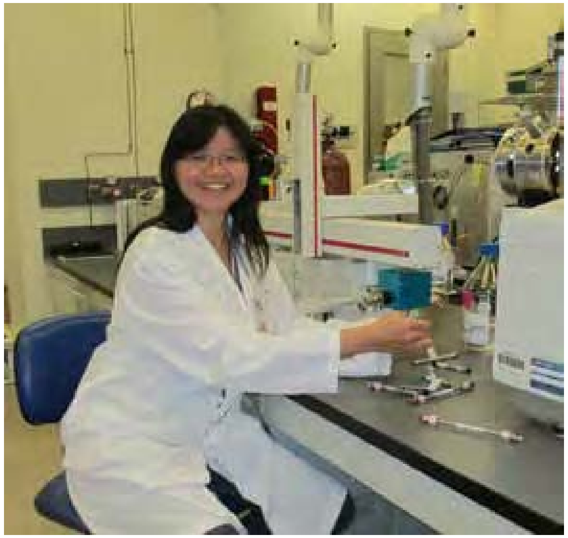
Date: 2014 DoD Environmental Monitoring Data Quality Workshop April 8-10, 2014

D653

Acknowledgments

Success Through Science®

Adam Robinson



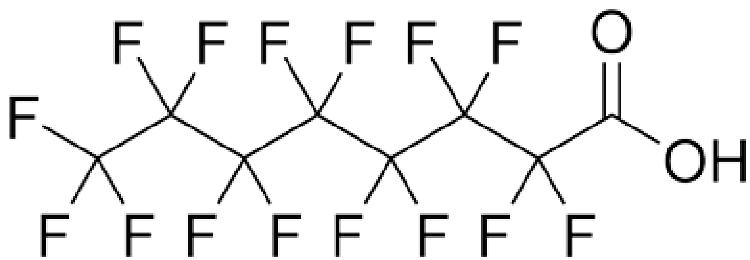
Sin Chii Chia

Outline

- Overview
  - History
  - Environmental Significance

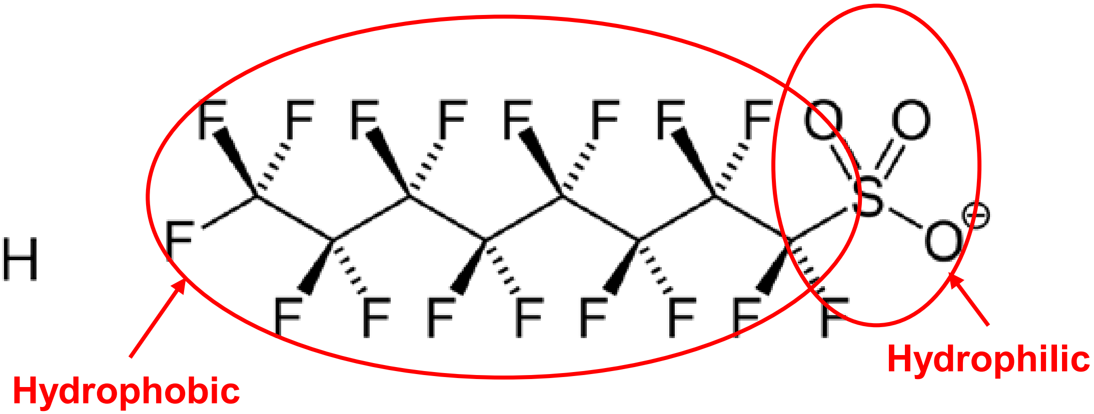
- Sampling, Analytical and Reporting Considerations
  - Sampling Challenges
  - Analytical Methods
  - "Understanding" the results

What are they?



Perfluorooctanoic Acid (PFOA)

Teflon®



Perfluorooctane Sulfonate (PFOS)

Scotchguard®

Chemical and Physical Properties

Success Through Science®

- Very stable and persistent – do not degrade
- Low volatility
- Soluble in water
- Readily bind (sorb) to variety of materials (hard to predict partitioning)
- Bioaccumulation
- Ubiquitous (“they’re everywhere”)

Fluoropolymers vs. Fluorinated Telomers

Success Through Science®

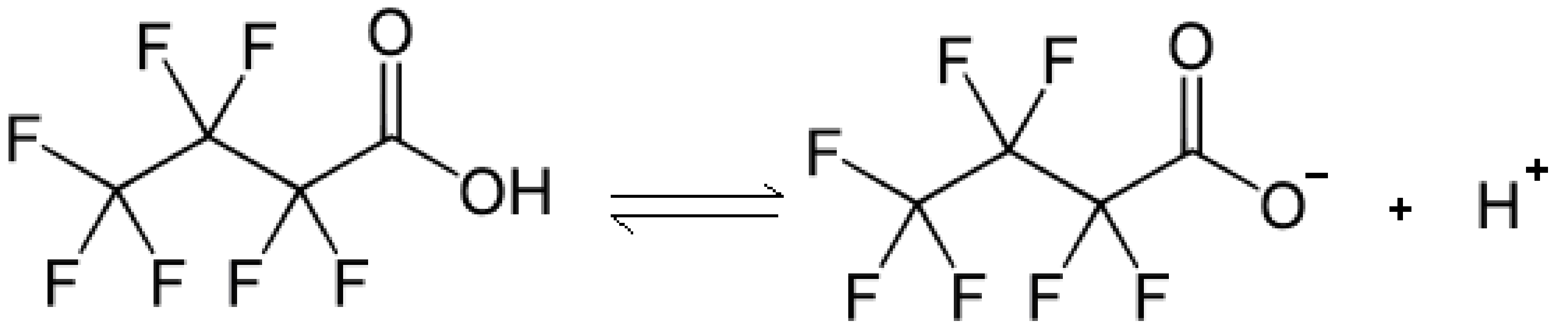
Fluoropolymers

- Long chain molecules
- Fire resistant
- Repel oil, grease, stains and water
- Used to provide non-stick surface in cookware and waterproof breathable membranes for clothing
- Hundreds of other uses in virtually all industry segments (e.g. aerospace, automotive, chemical, electronics, textile, etc.)

Fluorinated Telomers

- Surfactants and surface treatment chemicals in many products (e.g. repellent coatings on textiles, leather and paper)
- High performance surfactants in products that need to flow evenly (e.g. paints, coatings, fire fighting foams, engineering coatings in the manufacture of semi-conductor coatings)

PFC Naming Conventions



Perfluorobutanoic Acid
aka
Perfluorobutyric Acid

Perfluorobutanoate

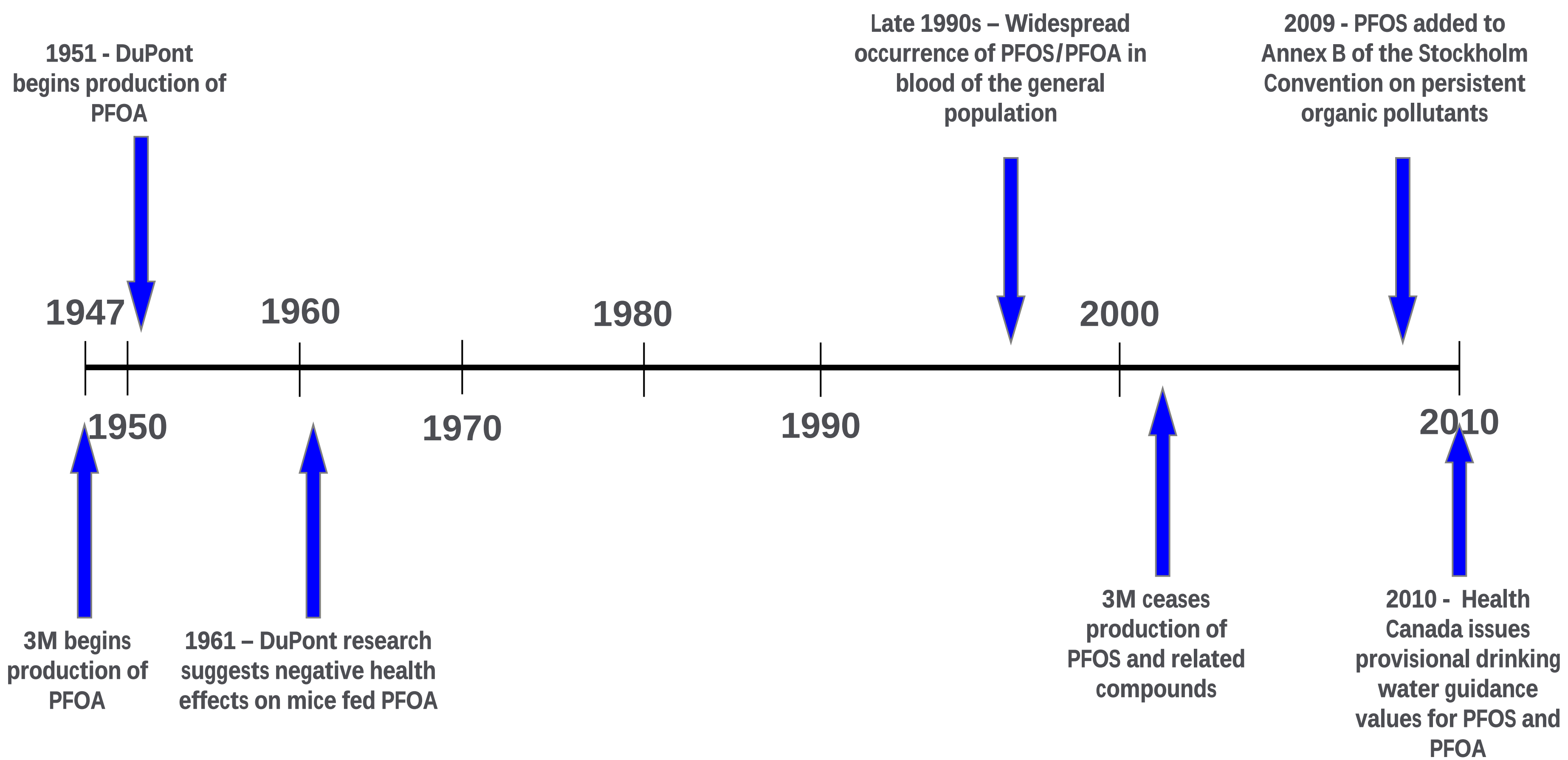
Naming Conventions

Success Through Science®

Abbreviation	Acid	Conjugate
PFBA	Perfluorobutanoic Acid	Perfluorobutanoate
PFBS	Perfluorobutanesulfonic Acid	Perfluorobutanesulfonate
PFPA	Perfluoropentanoic Acid	Perfluoropentanoate
PFHxA	Perfluorohexanoic Acid	Perfluorohexanoate
PFHxS	Perfluorohexanesulfonic Acid	Perfluorohexanesulfonate
PFHpA	Perfluoroheptanoic Acid	Perfluoroheptanoate
PFOA	Perfluorooctanoic Acid	Perfluorooctanoate
PFOS	Perfluorooctanesulfonic Acid	Perfluorooctanesulfonate
PFNA	Perfluorononanoic Acid	Perfluorononanoate
PFDA	Perfluorodecanoic Acid	Perfluorodecanoate
PFDS	Perfluorodecanesulfonic Acid	Perfluorodecanesulfonate
PFUdA	Perfluoroundecanoic Acid	Perfluoroundecanoate
PFDoA	Perfluorododecanoic Acid	Perfluorododecanoate
PFTrDA	Perfluorotridecanoic Acid	Perfluorotridecanoate
PFTeDA	Perfluorotetradecanoic Acid	Perfluorotetradecanoate
PFOSA	Perfluorooctanesulfonamide	Perfluorooctanesulfonamide

Key Dates for PFOS and PFOA

Success Through Science®



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Environmental Pathways for PFC Exposure

Success Through Science®

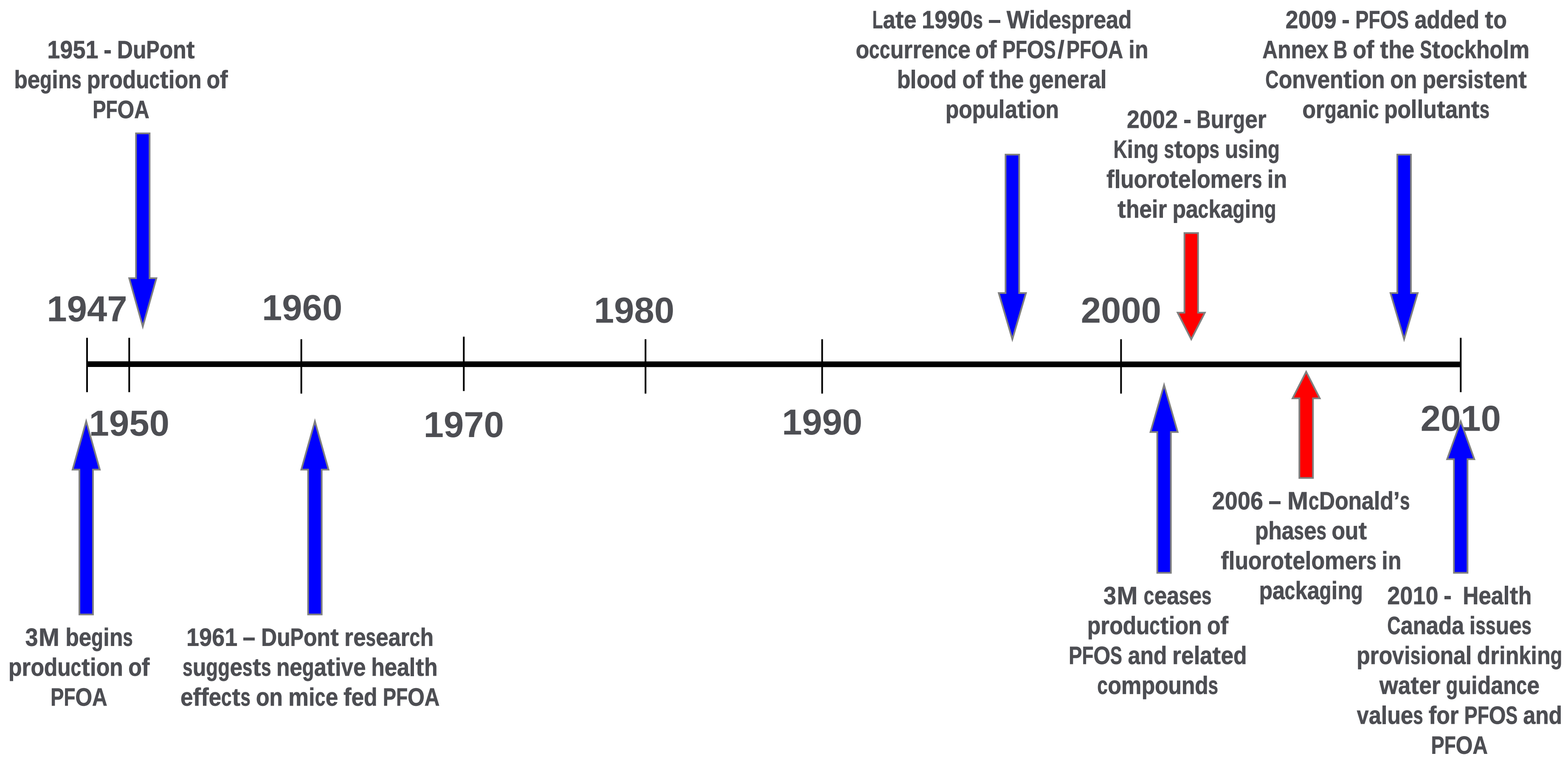
- Commercially used perfluorinated compounds (PFCs) have been widely detected in humans, but the sources of human exposure are not fully characterized

- Suggested sources of exposure
  - Drinking Water
  - Dust/Ambient Air
  - Food

How are PFC finding their way into people?

Success Through Science®

Key Dates for PFOS and PFOA



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Where are they being found?

Success Through Science®

- Although PFOS, PFOA and other PFCs are likely to be found anywhere, at differing concentrations, typical areas where these are compounds of concern, at elevated concentrations, are:
  - AFFF
  - Airports
  - Run-off from incidents of fire
  - Landfill leachate
  - WWTP Effluent

…and now they're making the news

Success Through Science®

THE KINGS COUNTY ADVERTISER
THE KINGS COUNTY REGISTER

The Register/Advertiser > News

Contaminated water in Greenwood

Nancy Kelly
Published on March 25, 2010

Chemical found in 14 Wing well

Environment International 39 (2012) 19–26

Contents lists available at SciVerse ScienceDirect

Environment International

journal homepage: www.elsevier.com/locate/envint

Highly elevated levels of perfluorooctane sulfonate and other perfluorinated acids found in biota and surface water downstream of an international airport, Hamilton, Ontario, Canada

S.R. de Solla [a,*], A.O. De Silva [b], R.J. Letcher [c]

[a] Wildlife and Landscape Science Directorate, Environment Canada, 867 Lakeshore Road, Burlington, ON, Canada
[b] Water and Science Technology Directorate, Environment Canada, 867 Lakeshore Road, Burlington, ON, Canada
[c] Wildlife and Landscape Science Directorate, Environment Canada, Ottawa, ON, Canada

Documents show widespread PFOS pollution at Hamilton's airport

The toxic firefighting chemical polluting Hamilton's airport has been found at more than 10 airports and military sites across Canada, according to a federal petition response.

Documents provided in a response to a petition filed with the auditor general's office by local biologist Joe Minor also say Transport Canada is still deciding whether it will help Hamilton deal with perfluorooctane sulfonate (PFOS) pollution at the city airport, which was once federally owned.

…nada is spending millions of dollars cleaning up historic pollution at a municipal airport in British Columbia even as it …me help to Hamilton.

…udying PFOS (perfluorooctane sulfonate) contamination at an old firefighting training pad at its airport, which Transpor… Canada owned until 1996. The toxic chemical has also been found downstream in fish and turtles in the Binbrook reservoir (http://www.thespec.com/news/local/article/…-low-level-pollution-riddles-reservoir-bottom).

Toxicology

Success Through Science®

- Still being evaluated- a range of toxicological effects have been reported in a variety of species
- Several agencies around world have developed aquatic risk values for PFOS and PFOA, and terrestrial risk values are being developed
- Not yet confirmed as human carcinogen
- Readily absorbed and accumulates in all tissues, especially target organs (e.g. liver)
- Not metabolized and eliminated slowly
- PFOS exposure has been associated with many health problems including some cancers
- Several agencies have derived human toxicity reference values – all suggest high potential human toxicity

Regulatory Status in Canada (PFOS Focus)

Success Through Science®

- Risk management strategies seek to have environmental PFOS concentrations as low as possible, prevent re-introduction to market, and address remaining uses (restrictions, exemptions, BMP etc.)
- PFOS still in AFFF (allowed at 0.5 ppm); AFFF with PFOS >0.5 ppm is <u>prohibited</u> on and after May 30, 2012 (certain military operations are excluded)
- On and after May 29, 2013, manufacture, use, sale and import of PFOS and PFOS-containing products is prohibited in Canada
- Other PFCs (polyfluorotelomer sulphonates) are used in newer AFFF formulations
- January 2013: EC Consultation document describes existing RM actions in Canada and globally, requests input on exemptions, and sets next steps and timelines

Regulatory Status

Success Through Science®

- 2010/2015 PFOA Stewardship Program:
  - Eight (8) global suppliers agree to:
  - 95% reduction in PFOA (and related precursor) levels in emissions relative to 2000 levels
  - Total elimination of PFOA (and related precursor) levels in emissions
- September 30, 2013 – USEPA Significant New Use Rule (SNUR) relating to perfluoroalkyl sulphonates (PFASs)
- SNUR related to perfluoroalkyl carboxylates (PFACs) (in-process)

Regulatory Limits – Drinking Water

Success Through Science®

Jurisdiction	PFOA (ug/L)	PFOS (ug/L)	PFBA (ug/L)	PFBS (ug/L)
Canada – Health Canada	**0.7**	**0.3**	N/V	N/V
U.S.A - EPA	**0.4**	**0.2**	N/V	N/V
U.S.A. – Minnesota	**0.3**	**0.2**	**7**	**7**
U.S.A. – New Jersey	**0.04**	N/V	N/V	N/V
U.S.A. – North Carolina	**2**	N/V	N/V	N/V
Europe – United Kingdom	**10**	**0.3**	N/V	N/V
Europe - Germany	**0.1 (PFOA and PFOS)**		N/V	N/V

Regulatory Limits – UCMR3

Success Through Science®

Compound	Minimum Reporting Level (ug/L)
Perfluorooctane sulphonate (PFOS)	0.04
Perfluorooctanoic Acid (PFOA)	0.02
Perfluorononanoic Acid (PFNA)	0.02
Perfluorohexanesulfonate (PFHxS)	0.03
Perfluoroheptanoic Acid (PFHpA)	0.01
Perfluorobutane sulphonate (PFBS)	0.09

Provisional Soil Levels - PFOS

Success Through Science®

Jurisdiction	Residential (ug/g)	Commercial (ug/g)	Industrial (ug/g)
Canada – Health Canada[1]	**0.7**	**1**	5
U.S.A – EPA Region IV	**6**	N/V	N/V

1) CCME Guidelines for PFOS anticipated 2014/2015

Problem Statement(s)

Success Through Science®

- Inconsistent approaches to analysis for PFCs in simple and complex environmental matrices
- High risk of sample cross contamination due to ubiquitous nature of PFCs and the tendency to be found at high concentrations
- Sample matrices range from simple to complex
- Data comparability between laboratories is difficult
  - High variability
  - Lack of confidence in the results
  - Inability to make supportable remedial decisions

Success Through Science®

Sampling, Analytical and Reporting Considerations

Sample Containers

Success Through Science®

Teflon®

Sampling: Best Practices

Success Through Science®

- Sampling and field quality assurance plans must address the concern for false positives
- Sampling methods MUST address potential sources of PFOS, PFOA and other PFCs:
  - No Teflon®
  - No Gore-tex®
  - No water proof field note books
  - No glass sampling containers
- Water for blanks MUST be PFC free.

Maxxam

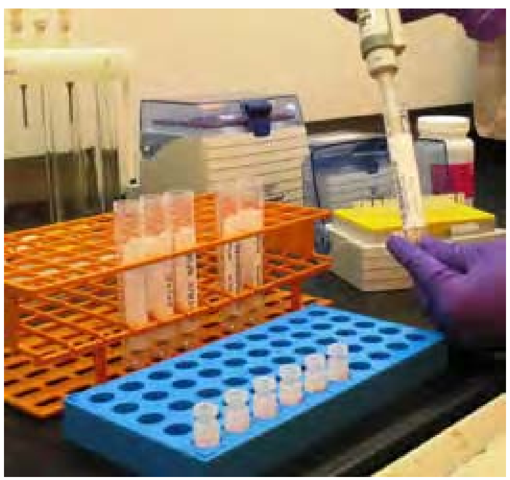
D676

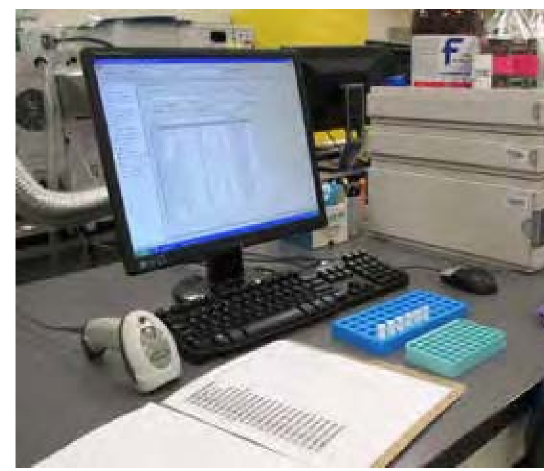
Analytical Methods
The "Basics"

Success Through Science®









Importance of Isotope Dilution…

- If the isotopically labeled analogue is added to the sample at the very beginning of the analytical process (i.e. before any sample homogenization, extraction, clean-up etc.), it enables exact compensation for variances at all stages of the analysis, from sample preparation through to the final instrumental measurement.

- IDMS provides greater accuracy than other calibration methods because it compensates for any matrix effects that may suppress recovery of the parameters being measured.

Importance of Isotope Dilution…

Success Through Science®

- Simply put…

  …the recovery of the labeled compound, which is not naturally present in the sample, is an exact representation of the recovery of the native compound which is present in the sample.

- Using PFOS as an example, the $^{13}C_4$-labelled analog of PFOS is used to correct for varying recovery of the native (^{12}C) PFOS from the sample. So if the recovery of the labeled PFOS is 60%, the recovery of the native PFOS being measured will also be 60%.

Branched (technical) vs. Linear PFOS

Success Through Science®

- When interpreting PFOS data, it is important to understand if it is being quantified as the linear or branched chain isomers
- Technical PFOS is a mixture of linear and branched chain PFOS isomers
- Linear PFOS is typically pure
- Branched chained PFOS contains at least 3 isomers
- This is an important differentiation because if linear PFOS is used as the calibration standard, the quantitative results on real-world samples (containing a mix of linear and branched PFOS) can be off by as much as 40%

Limits of Quantitation (LOQs)

Success Through Science®

Compound	LOQs	
	Water (ug/L)	Soil (ug/kg)
Perfluorobutanoic Acid (PFBA)	0.02	0.1
Perfluorobutanesulfonic Acid (PFBS)	0.02	0.1
Perfluoropentanoic Acid (PFPA)	0.02	0.1
Perfluorohexanoic Acid (PFHxA)	0.02	0.1
Perfluorohexanesulfonic Acid (PFHxS)	0.02	0.1
Perfluoroheptanoic Acid (PFHpA)	0.02	0.1
Perfluorooctanoic Acid (PFOA)	***0.02***	***0.1***
Perfluorooctanesulfonate (PFOS)	***0.02***	***0.1***
Perfluorononanoic Acid (PFNA)	0.02	0.1
Perfluorodecanoic Acid (PFDA)	0.02	0.1
Perfluorodecanesulfonate (PFDS)	0.02	0.1
Perfluoroundecanoic Acid (PFUdA)	0.02	0.1
Perfluorododecanoic Acid (PFDoA)	0.02	0.1
Perfluorotridecanoic Acid (PFTrDA)	0.02	0.1
Perfluorotetradecanoic Acid (PFTeDA)	0.02	0.1
Perfluorooctanesulfonamide (PFOSA)	0.02	0.1

Sampling Protocols and Analytical Methods: Key Areas for Consideration

Success Through Science®

- Sample Collection Protocols:
  - Low flow, bailers, passive samplers
  - Cross-contamination
  - Minimize exposure of samples/extracts to potential sources of PFCs
  - Fill containers completely
- Minimize transfer of sample aliquots
  - (i.e. as much as is possible, avoid subsampling prior to addressing adsorption)
- Sample Preparation:
  - Homogenization/Filtration

Sampling Protocols and Analytical Methods: Key Areas for Consideration

Success Through Science®

- Sample Extraction/Clean-up
  - Direct injection/solid phase extraction (SPE)
- Analysis:
  - "Isotope Dilution" LC/MS/MS
  - Calibration: solvent based standards/matrix matched standards
- Data Reduction:
  - Linear vs branched chain PFOS

What to expect when requesting analyses

- Laboratory Accreditation (ISO Guide 17025):
  - SCC (Canada)
  - DoD (USA)
- Calibration Range:
  - Water: 0.02 – 50 ug/L
  - Soil: 0.1 – 10 ug/kg
- Standard Turnaround Time – 10 working days
- Rush Analyses – Minimum 3 days

Questions?

eurofins

Lancaster Laboratories Environmental

The Use of Advanced Instrumental Techniques to Address Emerging and Unique Circumstance Contaminants.

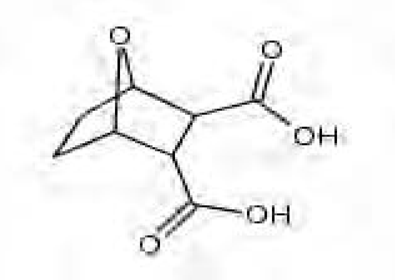
Chuck Neslund, Technical Director

2014 DoD Environmental Monitoring & Data Quality Workshop
April 8-10, 2014

Providing comprehensive scientific resources to environmental clients worldwide.

Case Study #1

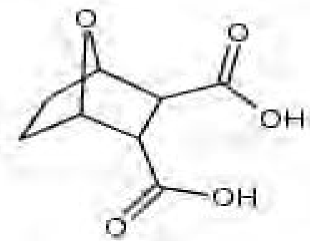
Endothall



- Widely used herbicide for control of aquatic weeds and algae
- Also used with sugar beets, hops, cotton and alfalfa.
- EPA has MCL of 100 ug/l in drinking water
- EPA Method 548.1 used for analysis in water

Case Study #1

Endothall

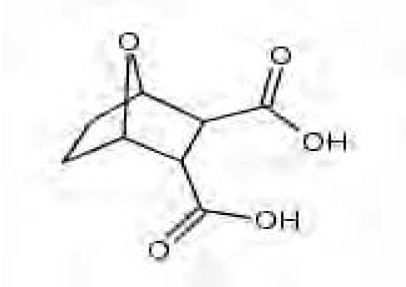


EPA Method 548.1

- Extract 100 mls of water with ion exchange SPE
- Derivatize with acidic methanol
- Analyze by GC/MS
- Method MDLs listed around 2 ug/l

Case Study #1

Endothall

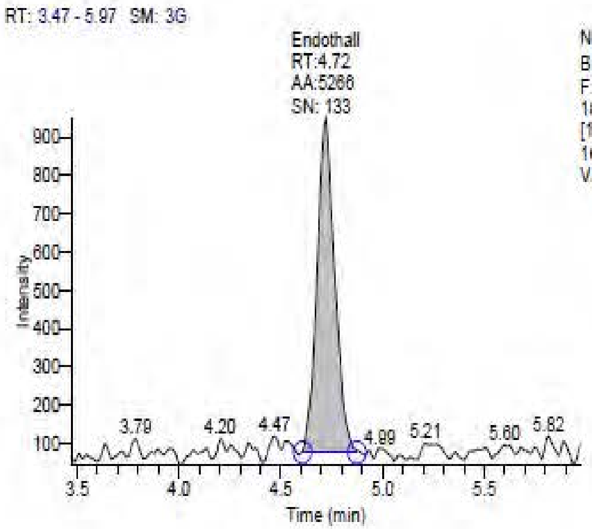


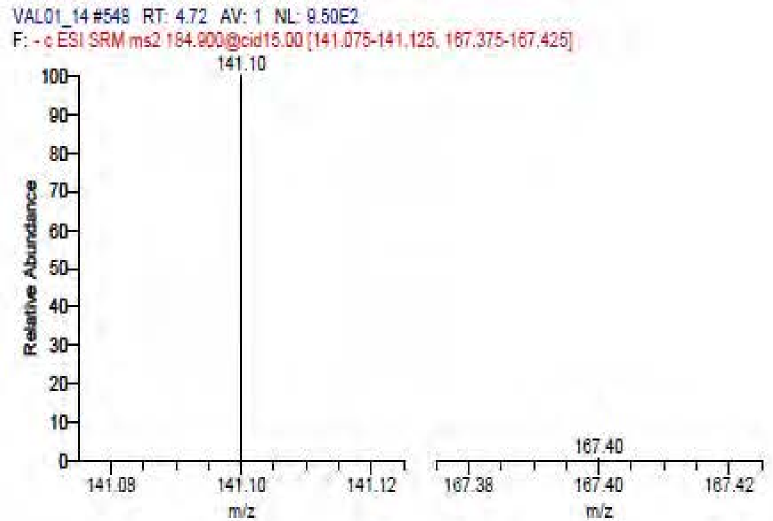
- Client needed soil samples analyzed in addition to waters
- No proven methodology for soil
- Dicarboxylic acid functionality looked suitable for LC/MS/MS approach
- Extract from soil? Optimally use water...maybe ion pairing reagent

Case Study #1

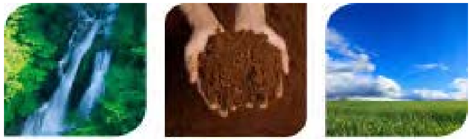
LOQ Level Standard for Endothall – 50 ng/g in soil

10 ng/ml in solution

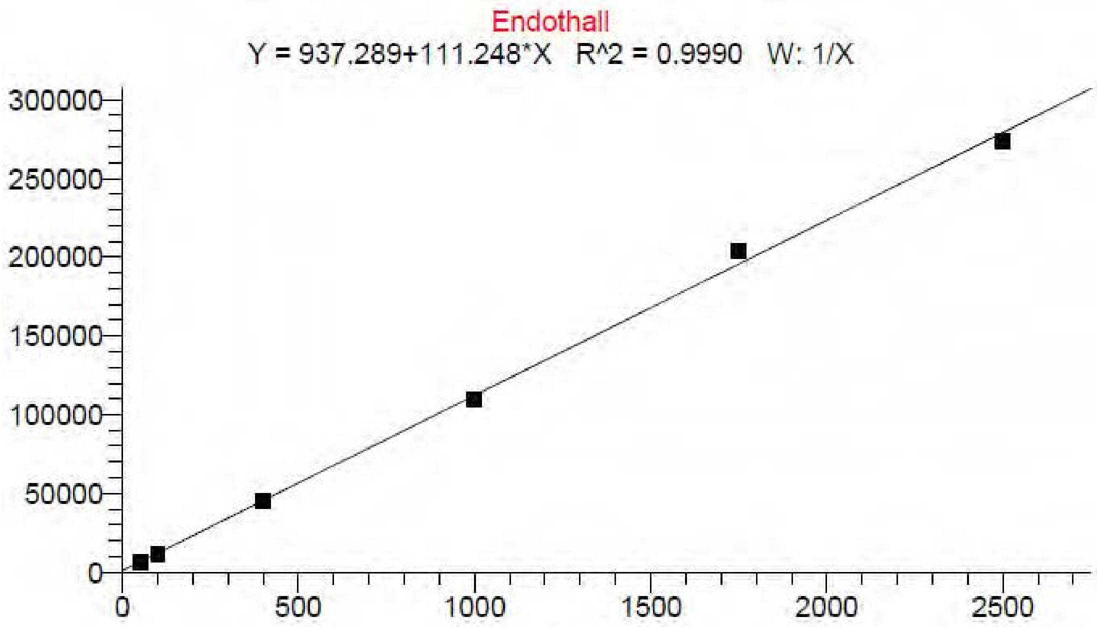




Case Study #1



Calibration Curve for Endothall – 50 ng/g to 2500 ng/g in soil

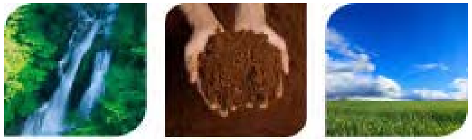


Recoveries of 70-130

LOQ = 50 ng/g

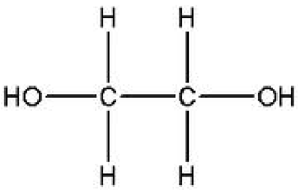
MDL = 25 ng/g

Case Study #2

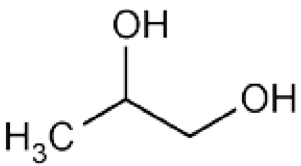


Glycols

Ethylene Glycol



Propylene Glycol

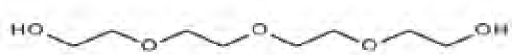


2-methoxy ethanol

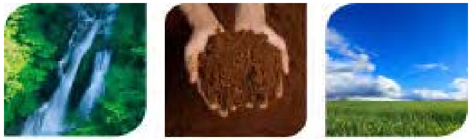
Diethylene Glycol

Triethylene Glycol

Tetraethylene Glycol



Case Study #2



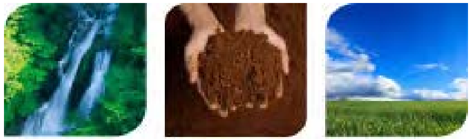
Glycols

- Group of compounds is very water soluble which makes extraction and concentration difficult
- Typical approach has been to use a GC/FID method like SW-846 8015, with direct aqueous injection (DAI)
- Sample matrix can have significant impact on what is detected (false positives)
- Sensitivity not spectacular, 5-10 mg/l common, optimized systems may do a little better

Glycols

- What about application of LC/MS/MS?
- Well suited for DAI, better selectivity and sensitivity?
- Concern about small size of molecules, particularly ethylene and propylene glycol
- How effectively would they ionize?

Case Study #2

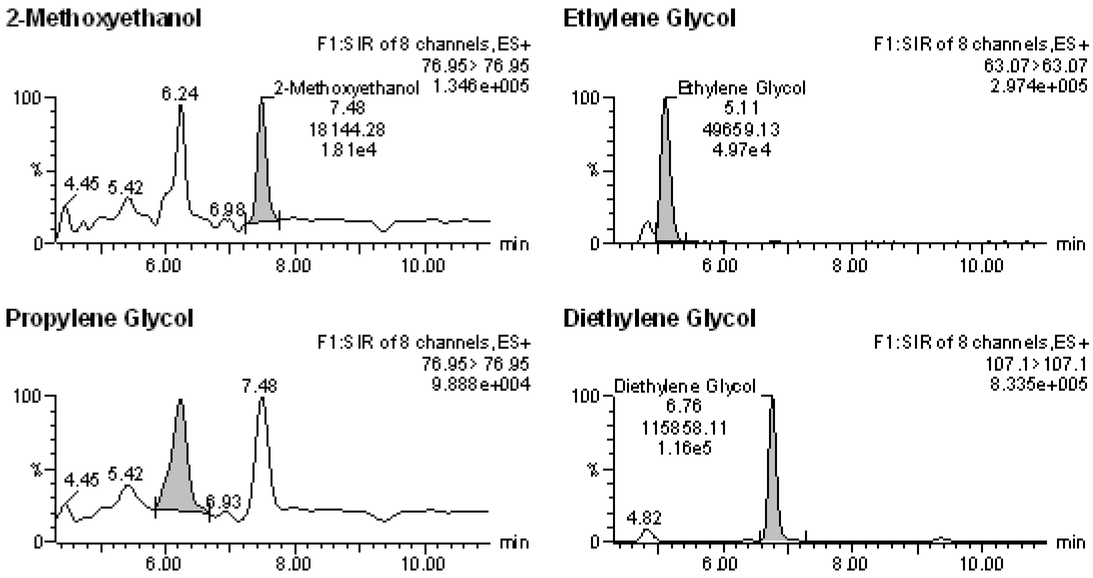


Glycols

Resolution – split the analysis into two analytical runs

a. Selected Ion Reaction (SIR)

b. Multiple Reaction Monitoring (MRM)



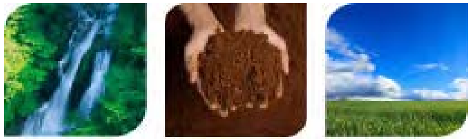
Ethylene Glycol – 500 ug/l

Propylene Glycol – 100 ug/l

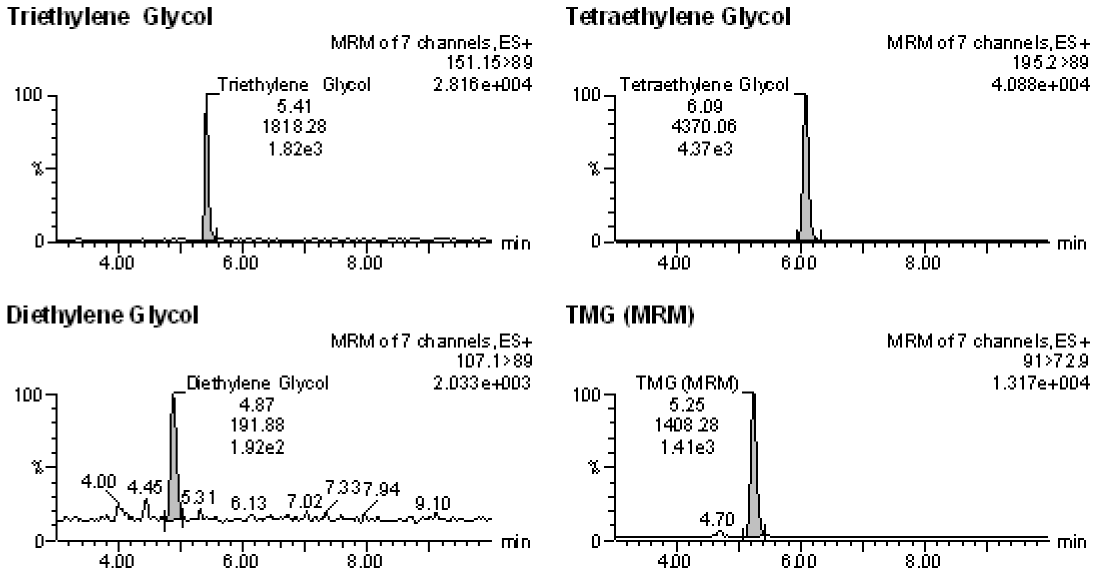
2-methoxyethanol – 100 ug/l

Diethylene glycol – 25 ug/l

Case Study #2



Glycols



Diethylene glycol – 25 ug/l

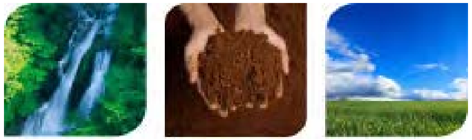
Triethylene glycol – 25 ug/l

Tetraethylene glycol – 25 ug/l

Diethylene glycol can be reported from either mode

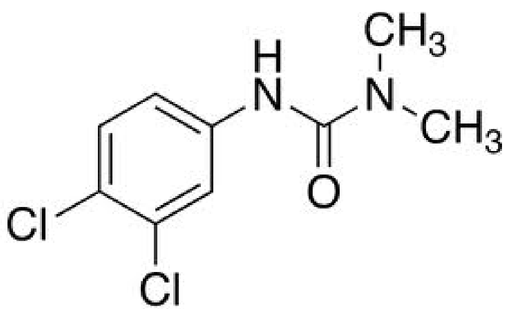
Note use of a surrogate, tetramethylene glycol

Limit for tetraethylene glycol improves

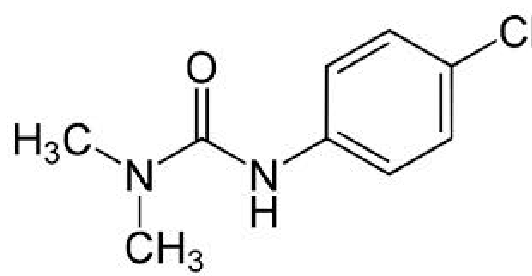


Phenyl Urea Herbicides

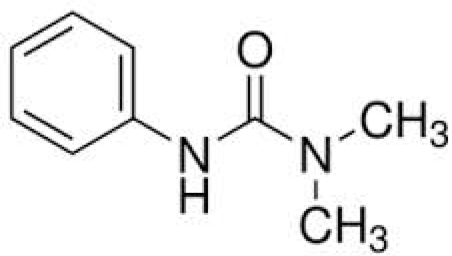
Diuron



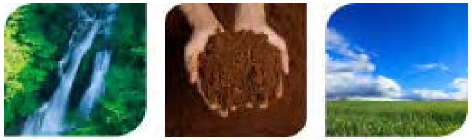
Monuron



Fenuron



Case Study #3

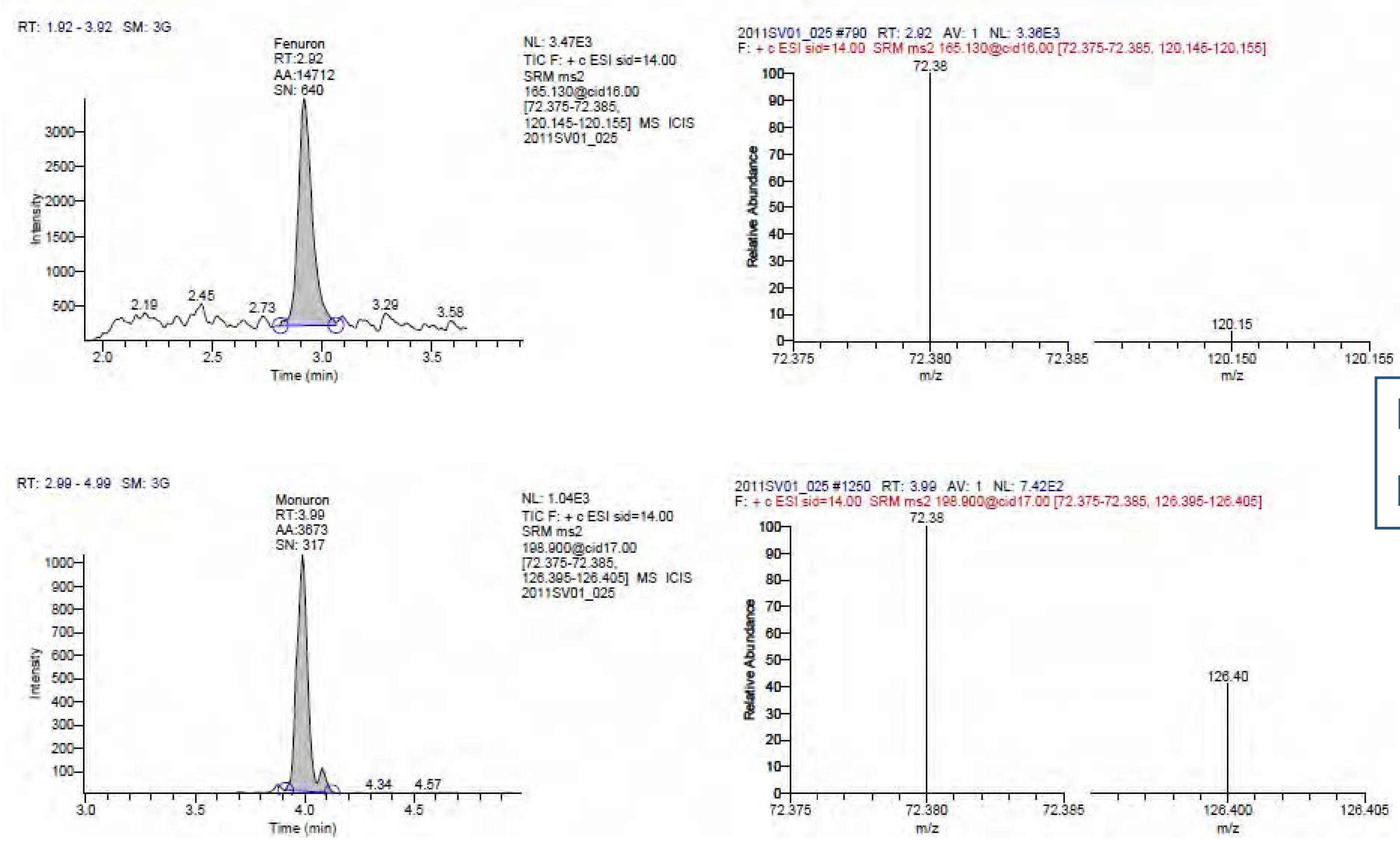


Phenyl Urea Herbicides

- Used for pre- and postemergent control of broadleaf and grassy weeds
- Also used on fruit and nut crops, grains, cotton, corn, etc.
- Analysis of compounds referenced in SW-846, Method 8321B
- Use of a generalized extraction resulted in sub-ppm limits
- Desire to optimize for low level detection

Case Study #3

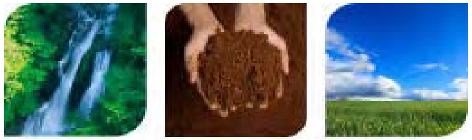
Phenyl Urea Herbicides



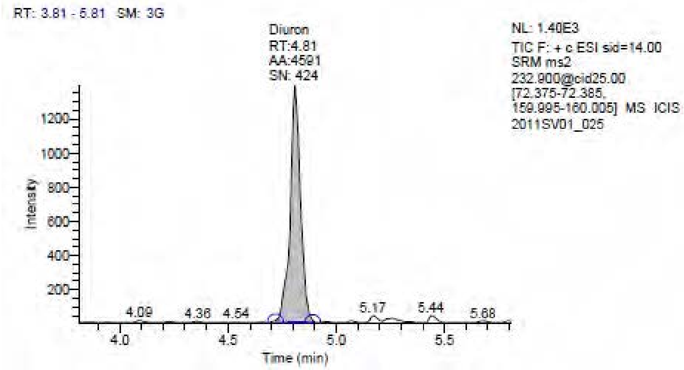
Fenuron – 1 ng/ml

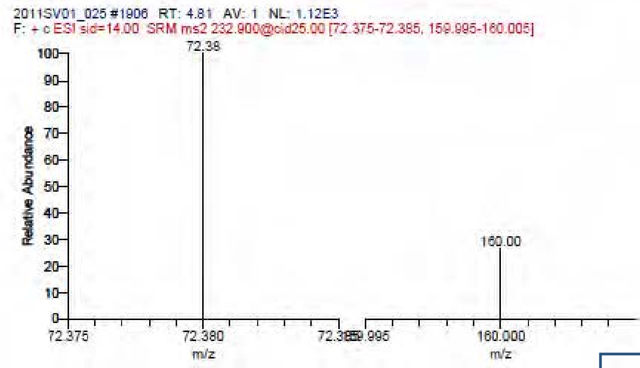
Monuron – 1 ng/ml

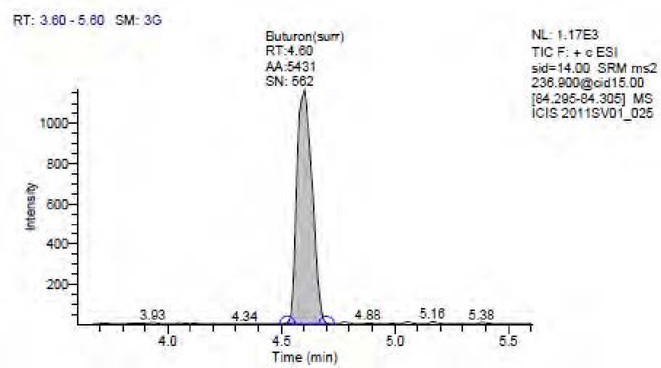
Case Study #3

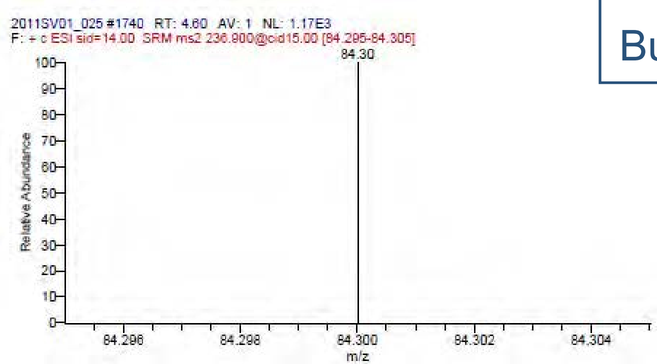


Phenyl Urea Herbicides









Diuron – 1.5 ng/ml

Buturon – 1.5 ng/ml

Case Study #3



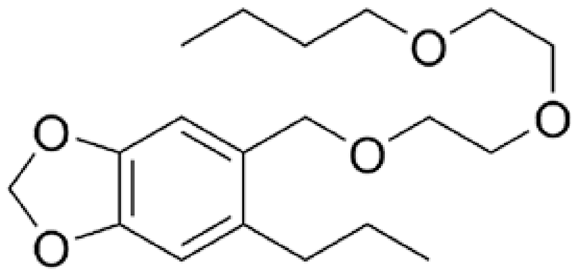
Phenyl Urea Herbicides

- 5 grams of soil blended with water and acetonitrile
- Extract cleaned up on SPE column
- Able to report low limits for analysis

	<u>LOQ</u>
Monuron -	0.2 ng/g
Fenuron -	0.2 ng/g
Diuron -	0.3 ng/g

Case Study #4

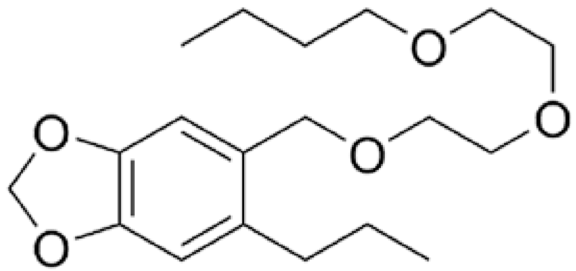
Piperonyl Butoxide



- Used as a synergist in pesticide formulations
- Enhances the potency of pesticide compounds like pyrethrins, pyrethroids and certain carbamates
- Used in over 1500 EPA registered products
- Home use and restaurants a significant consumer of products employing piperonyl butoxide

Case Study #4

Piperonyl Butoxide

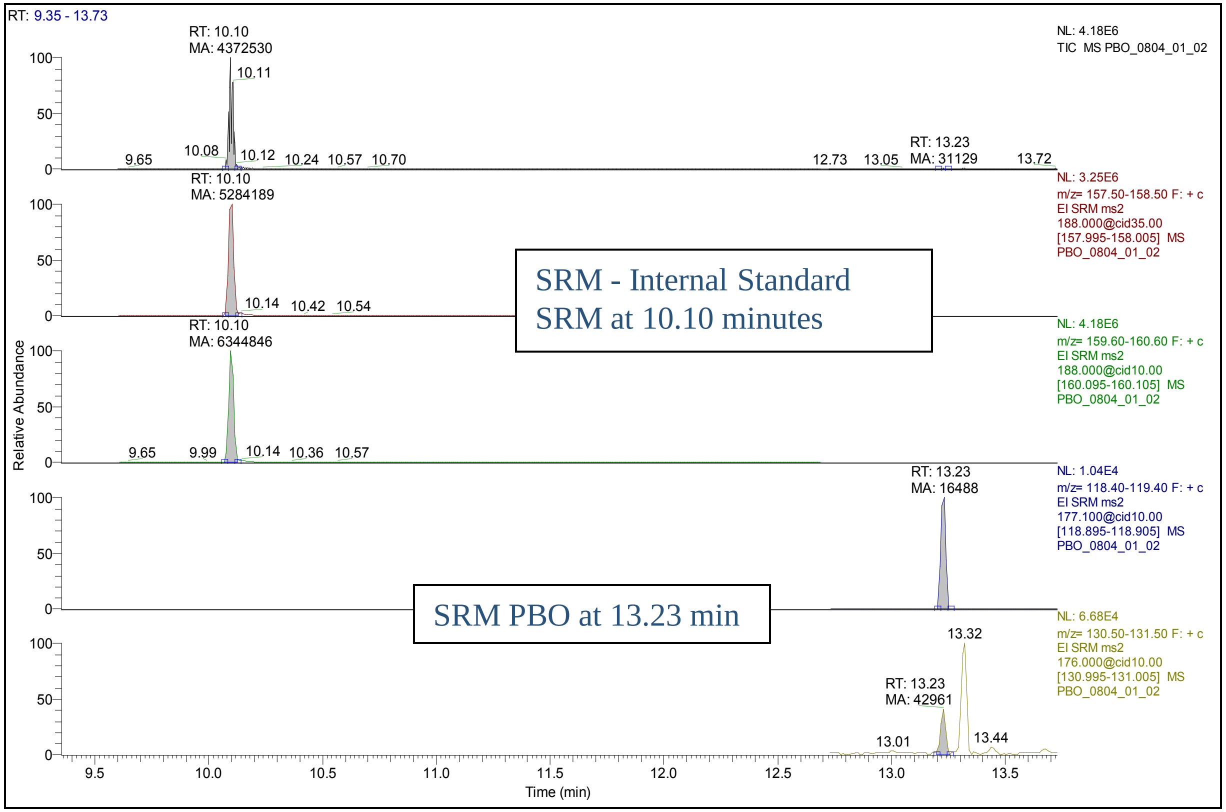


- Client with project to assess residual levels in WWTP sludges and biosolids
- Treatment of processes to degrade PBO, so need for low level analysis
- Extraction and clean-up of sludge extracts a challenge to meet low limits
- Take advantage of selectivity and sensitivity of GC/MS/MS to reach goals

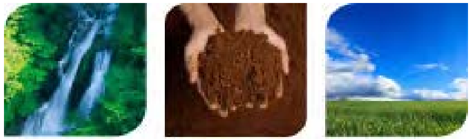
Case Study #4

Piperonyl Butoxide

LOQ Level Standard at 0.5 ng/ml

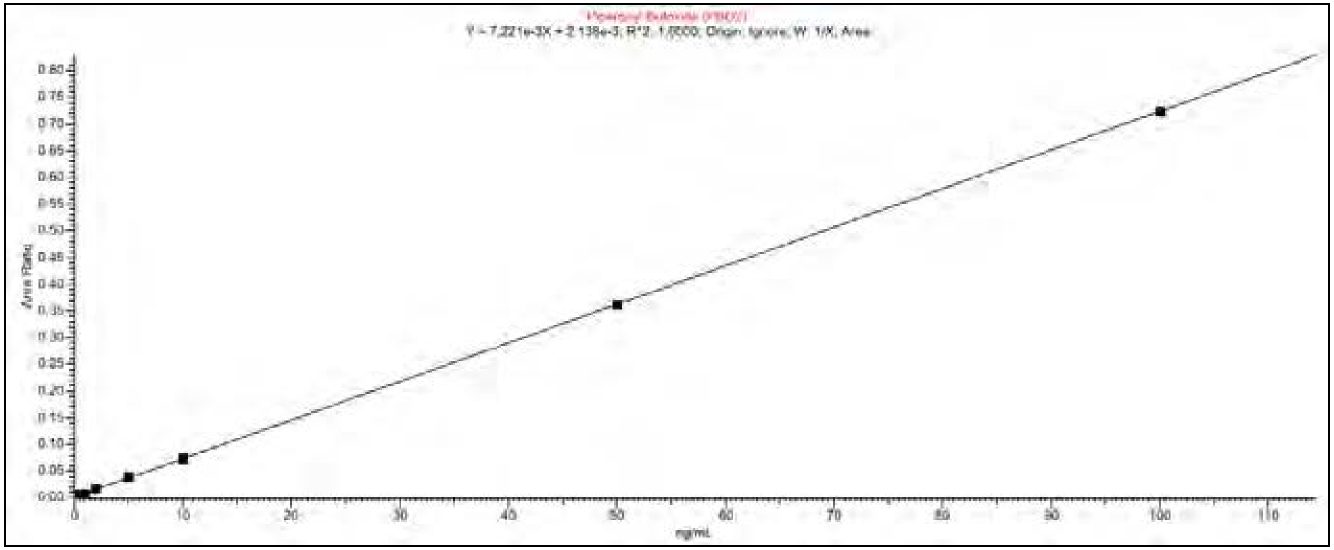


Case Study #4



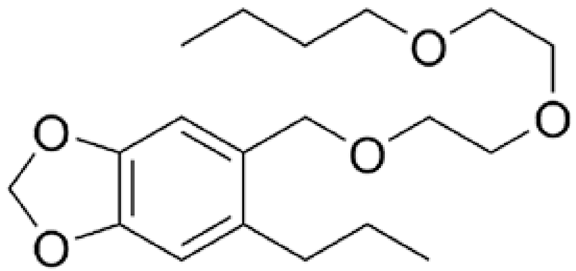
Piperonyl Butoxide

Calibration Curve - 0.5 ng/ml to 100 ng/ml



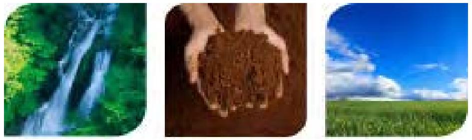
Case Study #4

Piperonyl Butoxide



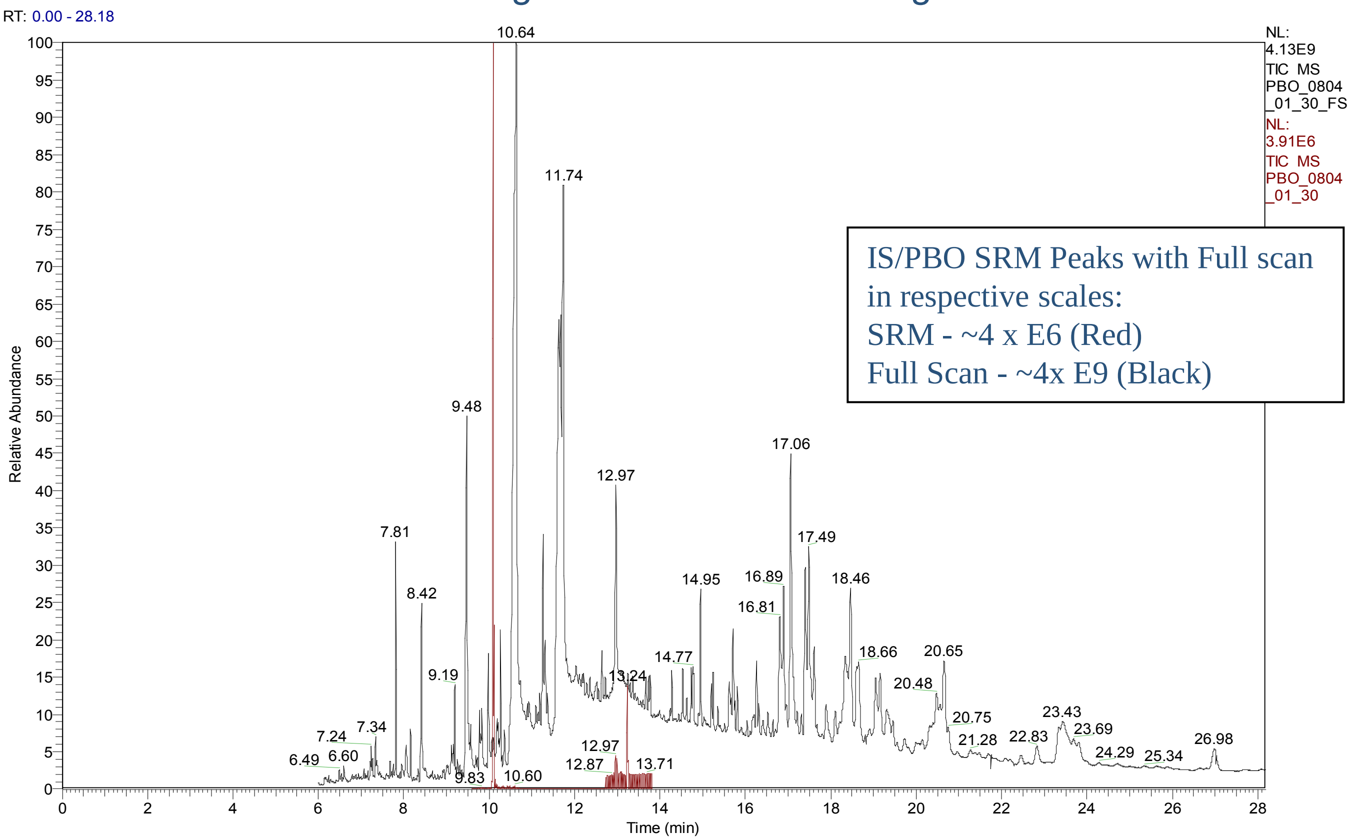
- Matrix present with the higher solids content presents problem
- Greater sensitivity and selectivity of GC/MS/MS allowed us to
    a. Use less sample for extraction (liq/liq extraction)
    b. Avoid use of column clean-ups that ultimately may have reduced recoveries.
- Under this scenario, still able to detect to 25 ng/l

Case Study #4

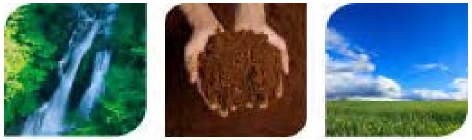


Piperonyl Butoxide

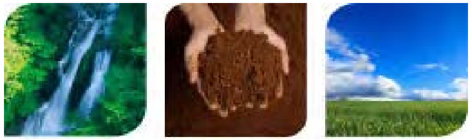
Full Scan Chromatogram vs SRM Chromatogram of Matrix



Conclusions

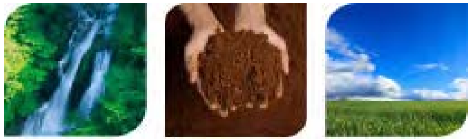


- While not exactly mainstream yet, the analytical techniques of LC/MS/MS and GC/MS/MS hold great promise
- Superior selectivity and sensitivity enable reporting in difficult matrices at lower levels
- Better sensitivity allows reduction in sample amounts and reduction in sample processing techniques
- Useful tools to consider for application to compounds not already well defined by the more standard analytical methods



Acknowledgement

- Meng Yu (Eurofins Lancaster Laboratories Environmental)
- Tim Trees (Eurofins Lancaster Laboratories Environmental).



Thank you

Nature Access and Environmental Justice:

Addressing Nature Access Disparities in the United States

MARCH 2021 | WHITEPAPER | NATUREQUANT.COM

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Introduction

Given the clear connections between nature and health, and the existing inequities in nature access in the U.S., Environmental Justice frameworks must include a measurement for nearby-nature.

A clean and healthy environment is a basic necessity of human life, as are balanced ecosystems, biodiversity, and other elements of nature on which people depend. Indeed, more than 100 constitutions across the world now include a human right to a healthy environment, with those documents serving as a powerful tool to protect the natural world.

The EJ movement promotes environmental, economic, and social justice by recognizing the direct links between economic, environmental, and health issues and demands a safe, clean community and workplace environment for all. A healthy and safe environment is a public good to which every person in the United States has an equal right, both in principle and in law. However, the reality is that American society has distributed nature's benefits—as well as the harms of an industrial society—unequally by race, income, and education status.

Certain environmental injustices related to harmful pollutants, such as the disproportionate concentration of toxic air pollution near communities of color, have been thoroughly demonstrated; but automatically measuring proximity to nature has been more elusive. As discussed below under Nature Access and Environmental Justice, nearby natural elements, like outdoor green spaces, blue spaces, and tree canopy, are unevenly and inequitably distributed. Particularly in communities of color and low-income communities, nature is often out of reach for many families. As the links between exposure to nature and mental and physical health become clear, access to nature must become another central component of the EJ movement.

"...access to nature must become another central component of the EJ movement."

One potential reason that access to nature has not been rigorously examined is that technical challenges exist in nature quantification and nature exposure assessment. Additionally, the scientific, medical, and political communities lack consensus around a standard definition or measurement of nature or nature exposure. Finally, at least in the United States, there is limited public promotion or understanding of the health benefits nature exposure can provide.

To address this dilemma, NatureQuant has developed innovative technology tools to quantify the natural elements for any static location or area (yielding a "NatureScore™"). As demonstrated below under Nature Score and Health, NatureScore™ technology shows that beneficial natural elements are not equally distributed across socioeconomic and racial groups. As a result, communities of color and low-income communities are far more likely than other communities to live in a place that is deprived of the benefits of nature, including nearby places that allow them to get outside safely and access clean water, clean air, and diverse wildlife.

The “Urban Century”

Humanity is undergoing a monumental shift, rapidly moving from a largely natural, outdoor existence to life in a more built, urban surrounding. Our current “built environment” is dramatically different from the one we occupied for 99% of human history, as it physically separates us from the natural world. Most people—over half globally, and approximately four in five in the United States—live in urban areas, where nature contact tends to be limited. This means that humans are increasingly disconnected from nature. In short, there is a growing nature deficit. As we have begun systematically studying the impact of this fundamental change, links between declining nature exposure and increasing depression, anxiety, heart disease, and obesity over the last five decades are becoming clear.

A large and growing body of scientific literature demonstrates that contact with nature (broadly defined as green space, parks, forests, bodies of water, etc.) can lead to measurable psychological and physiological health benefits. Natural areas also have been linked to other positive effects, like improved property values, lower pollution, reduced crime rates, strengthened communities, and slowed viral and bacterial disease transmissions. On par with changes in exercise or diet, nature contact offers promise both as prevention and as treatment of many serious diseases. Additionally, potential advantages of nature exposure include low cost (relative to conventional medical interventions), safety, and practicality (not requiring individualized attention from highly trained professionals). Few, if any, medications or other interventions can boast these attributes.

The Science Behind Nature Exposure and Health

Overwhelming evidence in the scientific literature links nature exposure to improvements in physiological and psychological human health. Over 150 observational studies and 100 interventional studies, tracking over 300 million individuals from 20 countries investigating 100 unique health outcomes, have convincingly demonstrated that greater nature exposure results in improvements in health span and longevity.[1] In short, these studies prove that nature exposure can result in a longer, healthier, and even happier life.

HOW DOES NATURE HAVE SUCH A PROFOUND INFLUENCE ON HUMAN HEALTH?

We don't yet have the whole answer, but a number of associations and direct links are becoming clear. Part of the answer is that humans have become more separated from nature than ever before, with a dramatic shift worldwide to more people living in urban environments.[2,3] This shift coincides with increases in the primary causes of death. According to the National Center for Health Statistics, in the United States the primary causes of death include heart and vascular disease, cancer, chronic respiratory disease, cerebrovascular disease (i.e., stroke), Alzheimer's and related dementias, and diabetes. While many of the underlying causes of these diseases are well known (for instance, sedentary lifestyle, poor eating habits, and chronic psychological stress), there are contributing or mediating factors as well, many of which are associated with a lack of nature exposure. Figure 1, below, displays data from a meta-analysis demonstrating the strong relationship between nature exposure (specifically, green space) and improvements in all-cause mortality. A meta-analysis is an evidence-based study with a greater ability to extrapolate outcomes to the greater population than individual studies. What this meta-analysis demonstrates is that, consistently across a number of independent studies collectively investigating over 8 million people, a greater level of greenness surrounding a person's home was associated with a longer life. Importantly, these epidemiological studies control for many potential alternative explanations (such as socio-economic status), definitively demonstrating the health benefits of nature exposure. As displayed in the graph, all studies and the combined data are shifted towards the level of greenness favoring all-cause mortality prevention.

	Deaths (n/N)	Hazard ratio (95% CI)	Weight (%)
Crouse et al	106, 180/1, 265, 515	0.94(0.94-0.95)	15.5
Villeneuve et al	181, 110/574, 840	0.98(0.97-0.99)	15.6
James et al	8604/108, 630	0.88(0.82-0.94)	3.9
Wilker et al	929/1645	0.96(0.92-1.02)	5.2
Vienneau et al	363, 553/4, 284, 680	0.96(0.95-0.96)	15.5
Ji et al	18, 948/23754	0.95(0.94-0.96)	15.7
Nieuwenhuijsen et al	28, 391/792, 649	0.92(0.88-0.96)	7.1
Zijlema et al	5889/9218	0.98(0.93-1.03)	5.8
Orioli et al	198704/1263721	0.99(0.98-0.99)	15.6
Total	912, 308/8, 324, 652	0.96(0.94-0.97)	100.0

Random-effects model

Heterogeneity: I2 = 95%;p<0·0001

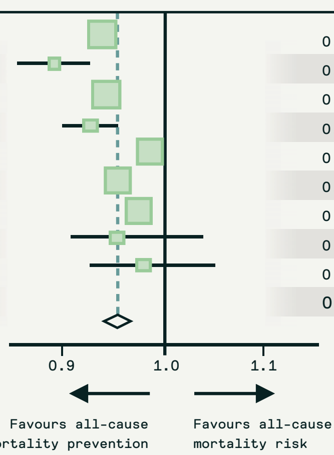


Figure 1. Results from individual studies and a meta-analysis strongly demonstrating the influence of exposure to greenness favors longevity and prevention of all-cause mortality even when many other factors are considered.

The relationships that underpin the health and longevity benefits of nature exposure are complex and multifaceted, and a number of psychophysiological and social pathways have been proposed that generally link the benefits of nature to health, including improved air quality, increased physical activity, more frequent social contacts, and decreased stress.[5]

In an effort to simplify the empirical literature from distinct disciplines, four domains that emphasize different functions of nature have been proposed by Markevych et al (2017)[6]:

1) Environmental Quality, 2) Stress Reduction 3) Physical Activity, and 4) Social Contacts discussed on the following pages, and graphically represented above in Figure 2.

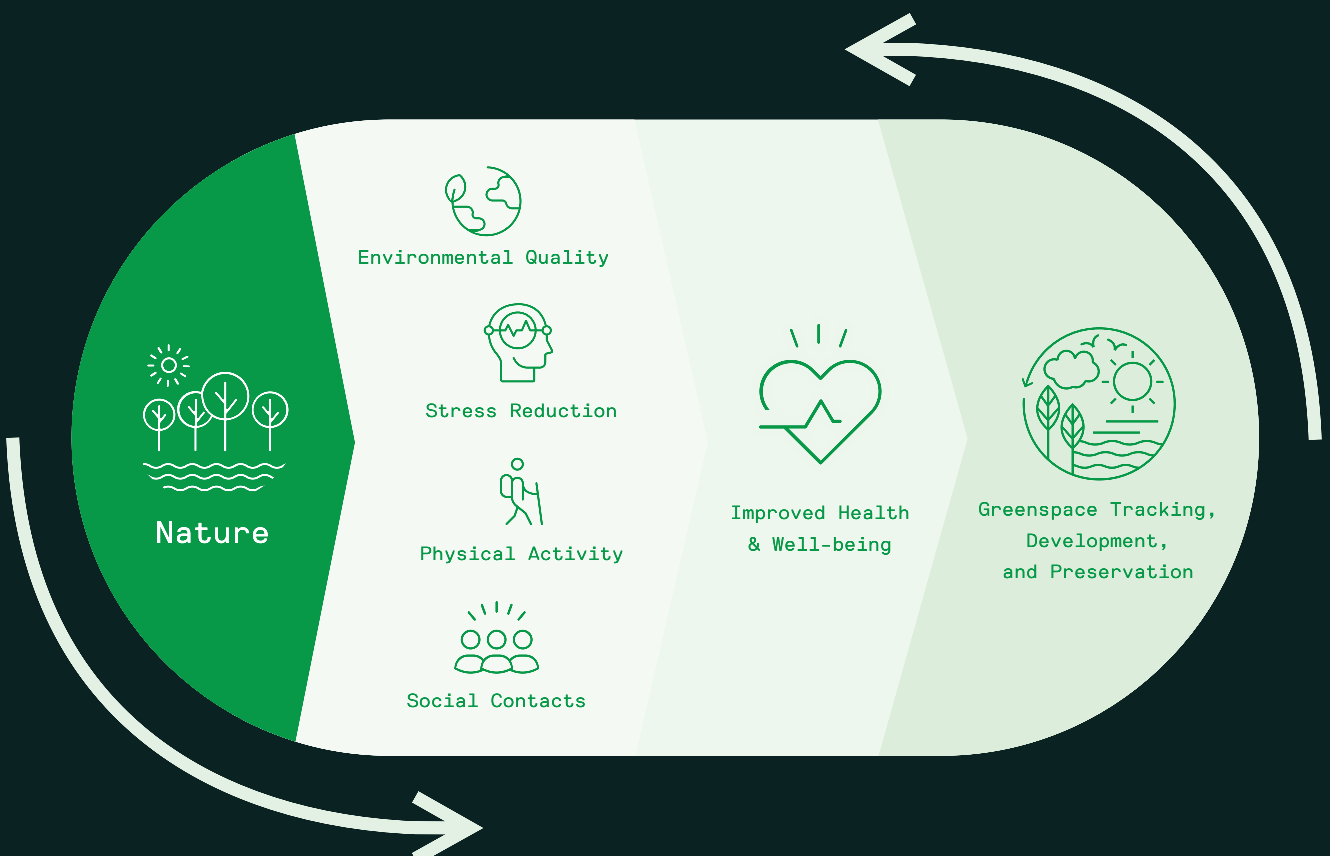


Figure 2

1. ENVIRONMENTAL QUALITY

In this paradigm, Environmental Quality captures the benefits of the urban natural environment through the effects on air quality, noise reduction, reducing urban "heat island effects", lowering building energy costs[7], and improving microbial biodiversity enhancement. In terms of improving microbial biodiversity, use of greenspace in cities increases exposure to a range of micro-organisms, including bacteria, protozoa, and helminths, which are abundant in nature and may be important for the development of the immune system and the regulation of inflammatory responses. This "old friends," hypothesis proposes that lack of exposure to immunoregulatory microorganisms in modern urban societies is resulting in an epidemic of inflammatory disease, as well as psychiatric disorders in which chronic, low-level inflammation is a risk factor. Recent studies indicate that treatment with a specific soil bacterium, Mycobacterium vaccae, may alleviate depression and PTSD[8]. Lastly, nature promotes sustainability through habitats for urban wildlife and reducing flood risk by decreasing impervious surface area[9].

2. STRESS REDUCTION

Stress Reduction encompasses the mental and physiological aspects of human health through reduction in stress. Given the links between mental and physical health, the importance of the natural environment on psychological health cannot be over emphasized.[10,11] A number of studies link exposure to the natural environment with mental health benefits through mechanisms such as visibility of urban green spaces for rest and restitution.[12–15] Factors such as improved mood, elevated self-esteem, reduced cognitive fatigue, enhanced attentional capacity and well-being, promoted emotional recovery, and reduced inflammation have all been reported.[12,16]

3. INCREASING PHYSICAL ACTIVITY

Increasing Physical Activity occurs through enjoying accessible, safe, and pleasing places to exercise and experience the natural environment. The benefits of exercise on health are well known and go beyond improvements in typical biomarkers such as reduced blood pressure, improved blood chemistry, and greater strength and endurance.[17] In this context, nature also plays an important role in creating a "culture of health," a culture that supports health improvement by fostering healthy, equitable communities that enable everyone to make healthy lifestyle choices.[18]

4. SOCIAL CONTACTS

Social Contacts describes the benefits of urban natural environments through improved social interactions among people[19] and greater social cohesion.[20] Nature contributes to improved social contacts by building a sense of community, focused on trust, shared norms and values, positive and friendly relationships, and feelings of being accepted and belonging.[21] Residents in neighborhoods with more, and/or higher quality streetscape greenery experience less stress and more social cohesion; in addition, they spend more time on physical activity.[22]

Access and exposure to natural environments impact health and well-being within each of these domains through numerous physiological pathways, including lowering concentrations of cortisol, lowering heart rate and blood pressure, decreasing sympathetic nerve activity, and increasing parasympathetic activity.[23,24] A growing body of evidence suggests this relationship is especially strong for low-income and nature-deprived urban populations.[25,26] Lower exposure to green space in these populations has been associated with a number of lifestyle diseases, such as obesity, Type II diabetes, and osteoporosis, as well as stress-related illnesses, such as depression, heart diseases, and mental fatigue.[22,25]

Importantly, improved health and well-being support the preservation and restoration of nature, allowing for a cycle of health and nature improvements. This comes from the perceived value of the natural environment in cities, pride in community, and improved property values.

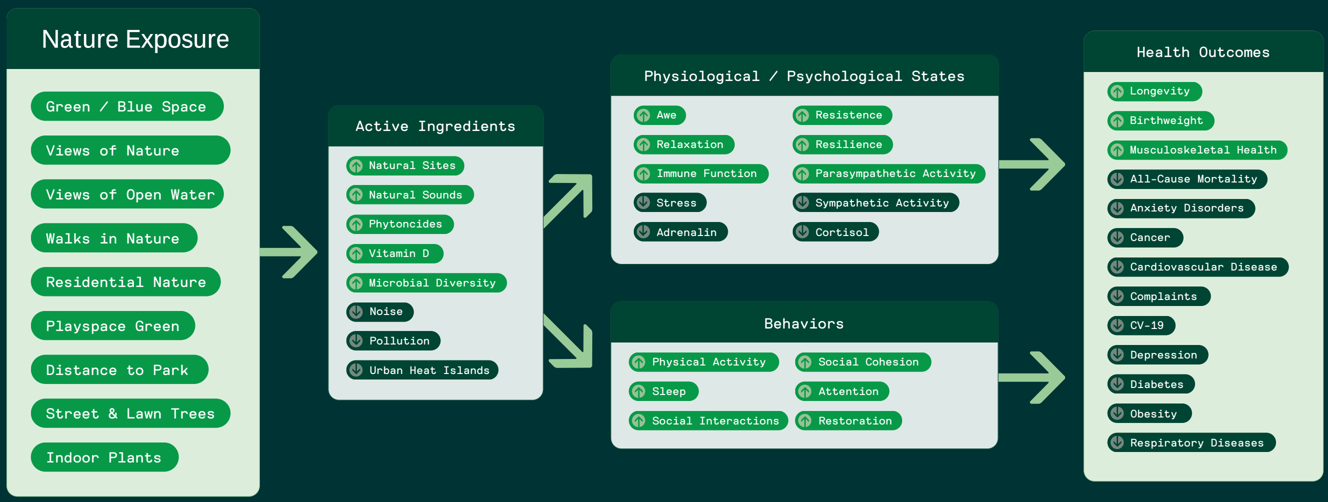


Figure 3

NatureScore™ and Health

While it is difficult to untangle the many components that influence a population's health, the analysis below compares NatureScore™ by census tract against health statistics for the 500 largest cities in the U.S. The analysis demonstrates a strong association between the presence of nature and health outcomes. Every negative health indicator is inversely associated with observable nature elements (more nature, less disease), while life expectancy is positively correlated (more nature, longer life).

These findings become critically important because, as detailed below, access to nature is not equitably available.

All of these correlations are statistically significant, with confidence intervals exceeding 99.9%.

Pearson Correlation to NatureScore

Based on 29,296 Urban Census Tract Scores, Grouped by State

	Diabetes	Frequent Mental Distress	Frequent Physical Distress	Obesity	Physical Inactivty	Life Expectancy
National Average	-0.18	-0.36	-0.27	-0.25	-0.32	+0.34

NatureScore mesasurements derived from 29,296 census tracts and the 500 largest U.S. cities. U.S. Small-area Life Expectancy Estimates Project (USALEEP):Life Expectancy Estimates File, National Center for Health Statistics. 2010-2017.

NatureScore™

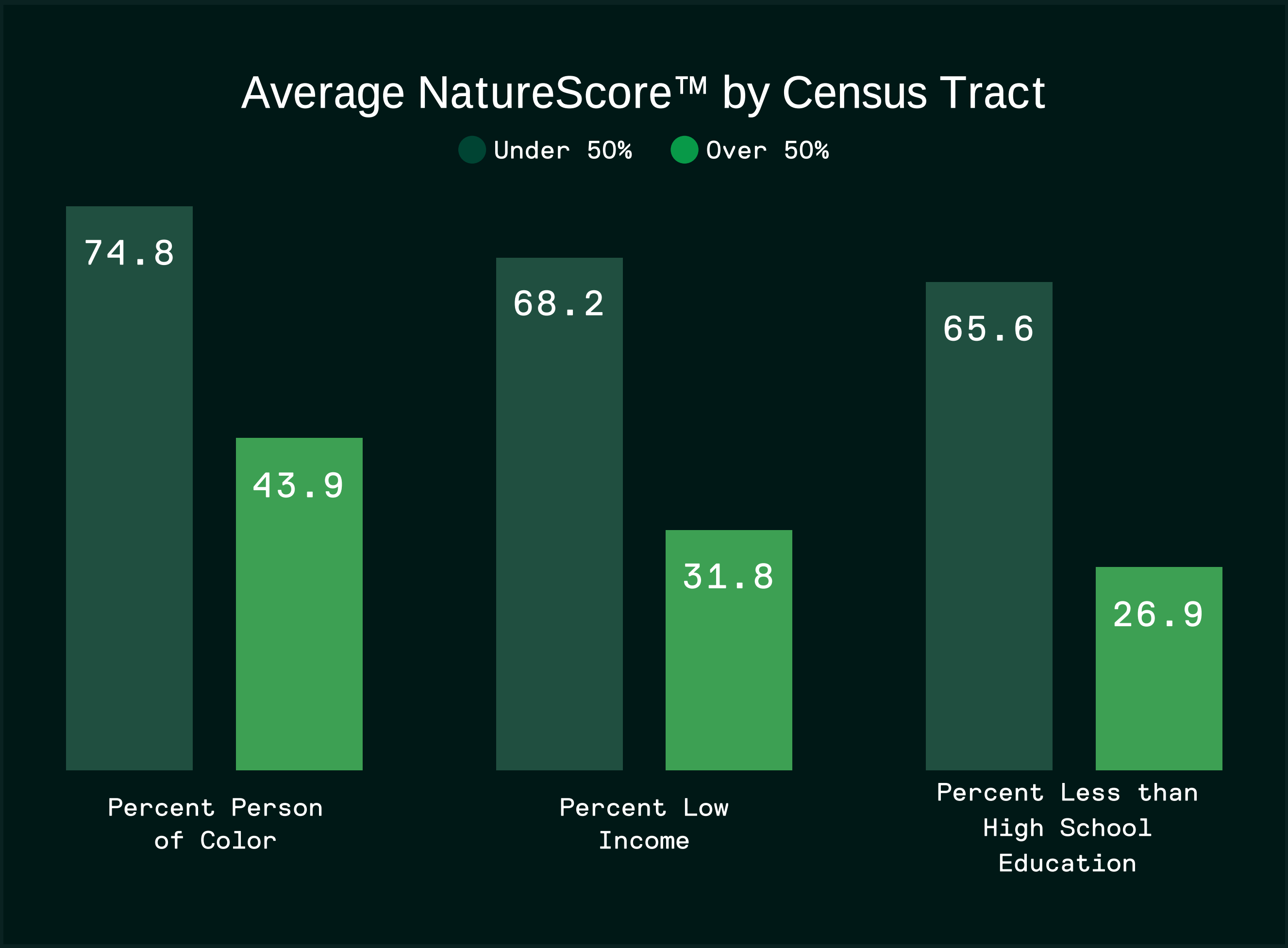
By NatureQuant

Illustrative Data Sources. NatureQuant synthesizes numerous data sources to create a "NatureScore™" for any given location. By applying evolving algorithms to an ever-increasing body of health, location, and natural element databases, NatureQuant teases out the critical elements of exposure to optimize the health impacts of the tracking tools.

Nature Access and Environmental Justice

The following analysis examines the distribution of natural elements by neighborhood (as defined by 217,739 census block groups) to help understand the types and extent of disparities in nature access that exist in the United States. Further, this analysis looks at the correlations between NatureScore™ and various EJ Indexes for pollution and disease risks.

This report is intended to supplement, not supplant, the many individual voices and efforts that have been identifying and working to correct existing inequities and injustices. The data helps confirm the scale of racial and economic disparities in nature access. Notably, families of color and/or low income have materially less access to nature than others; in short, these communities are disproportionally nature-deprived.



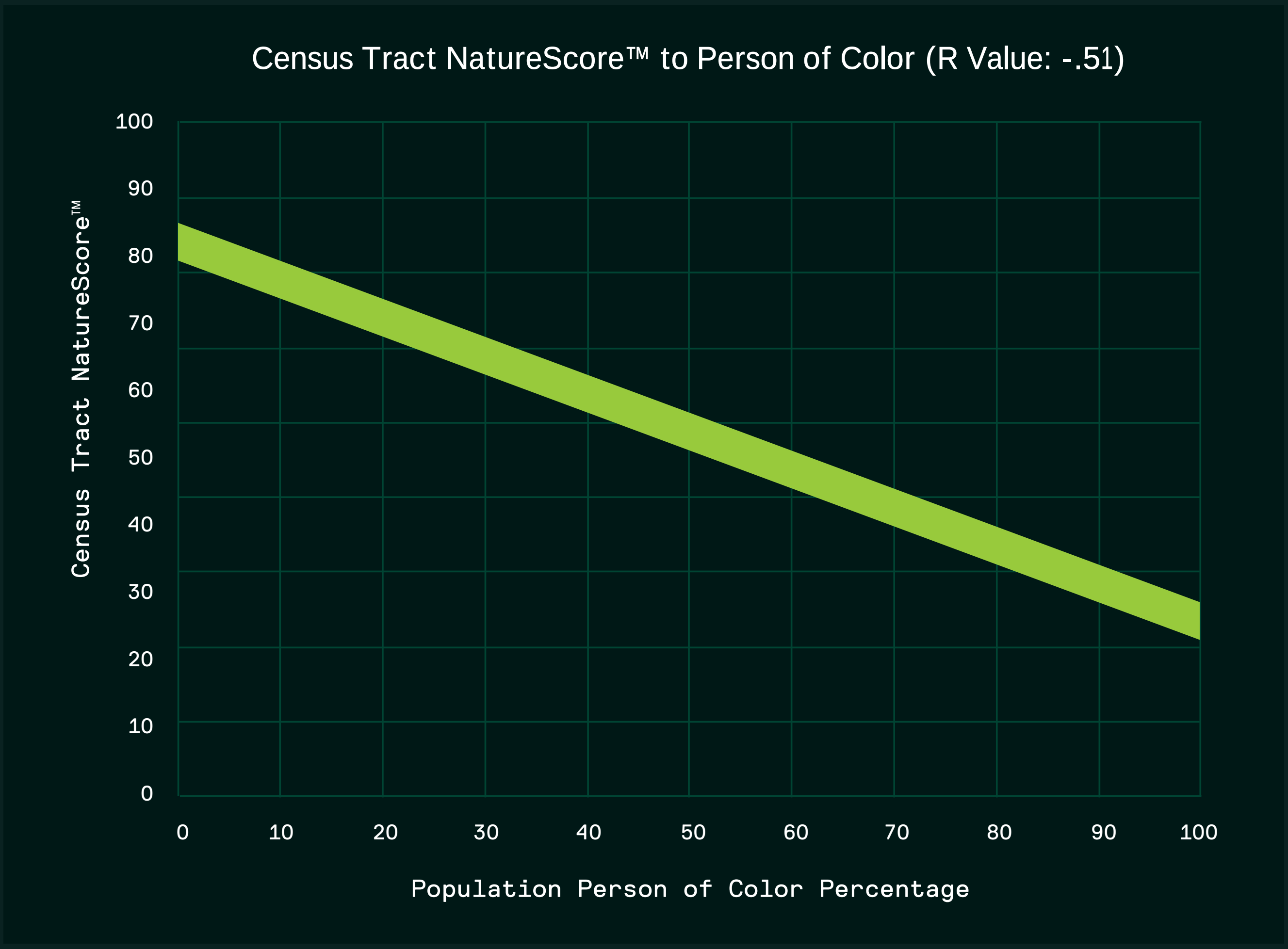
In this analysis we have compared NatureScore™ by census tract block to the following demographic indicators, collected from the EPA's EJScreen.

Percent People of Color: The percent of individuals in a block group who list their racial status as "a race other than white alone" and/or list ethnicity as "Hispanic or Latino"; thus, all people other than non-Hispanic white-alone individuals are considered a Person of Color. The word "alone" in this case indicates that the person identifies as a single race, not multiracial.

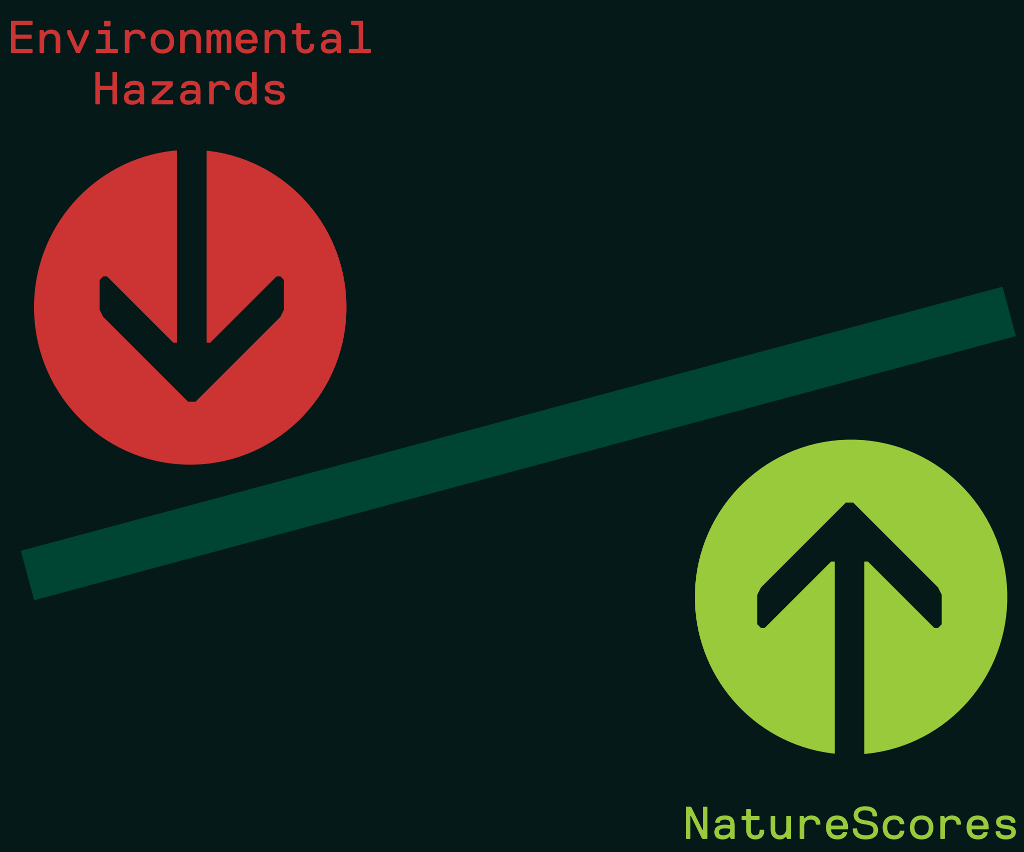
Percent Low-Income: The percent of a block group's population in households where the household income is less than or equal to twice the federal poverty level.

Less than High School Education: Percentage of people age 25 or older in a block group whose education falls short of a high school diploma.

The NatureScore™ correlation analysis reveals significant inequities in nature access for marginalized populations. For example, while the national percentage of people of color is 37%, in 66% of the census block groups with a NatureScore™ of 10 or below (nature deprived) the majority of the population were people of color. Across all three demographic metrics we measured, on average, tracts reporting wealthier, whiter, and more highly educated populations had materially higher NatureScores (indicating better access to nature and its protective benefits).



There is a powerful inverse relationship between the presence of nature and the reduction of environmental risks.



Further, we examined the relationship between NatureScores™ and various environmental hazards. The results yield a strong negative association across all measures. Put simply, in areas with more nature people generally find better air quality and less environmental risk. Note that all of these correlations are statistically significant with confidence intervals exceeding 99.9%.

Pearson Correlation to NatureScore

R-Score, Based on 217,739 Census Block Groups

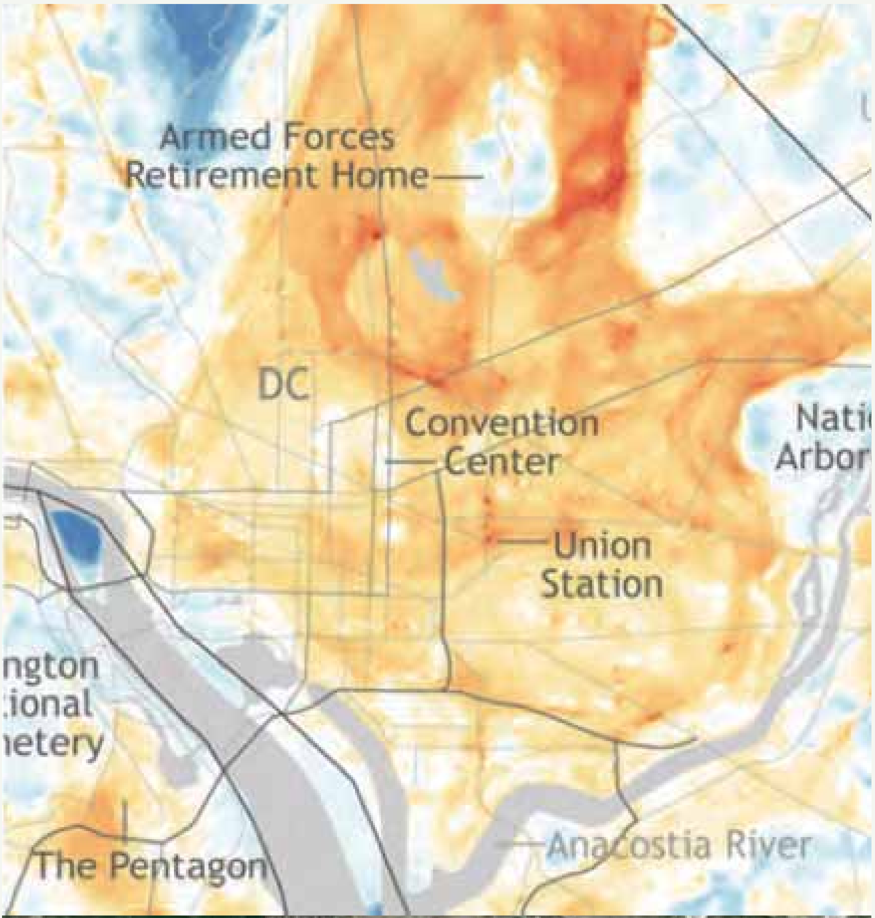
NatureScore™ ↑	↓
Demographic Indicators	
% People of Color	-0.51
% Low-Income	-0.19
% less than high school	-0.25
Air Quality Indicators	
Air Quality Indicators	-0.57
Air Toxics Cancer Risk	-0.26
Air toxics Respiratory Hazard Index	-0.42
Ozone level in Air	-0.23
PM2.5 Level in Air	-0.32
Environmental Hazard Indicators	
Traffic proximity and volume	-0.41
Proximity to National Priorities List (NPL)	-0.16
Proximity to Risk Management Plan	-0.29
Proximity to Treatment Storage	-0.31
EPA Environmental Justice Indices	
EJ Index for Diesel Particulate Matter	-0.38
EJ Index for Air Toxics Cancer Risk	-0.38
EJ Index for Air Toxics Respiratory Hazard	-0.39
EJ Index for Ozone Level in Air	-0.41
EJ Index for PM2.5 Leel in Air	-0.40

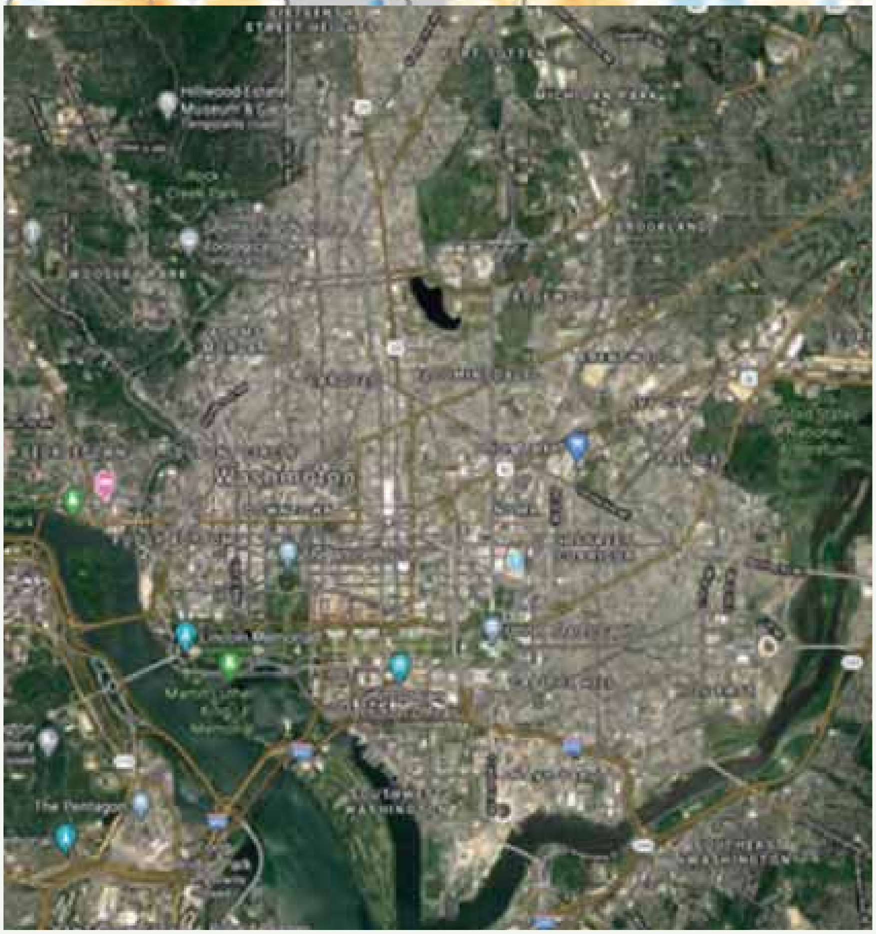
*Data Sourced from EPA EJSCREEN, 3/22/21 (https://www.epa.gov/ejscreen/what-ejscreen)

Nature and Urban Heat Islands

Urban Heat Islands occur when cities replace natural land cover with dense concentrations of pavement, buildings, and other surfaces that absorb and retain heat. This effect increases energy costs (e.g., for air conditioning), air pollution, and heat-related illness and mortality. Relatedly, climate change will likely lead to more frequent, more severe, and longer heatwaves during summer months. Extreme heat events often affect our most vulnerable populations first; indeed, heat-related mortality rates for the elderly have increased markedly in the last decade. Trees, green roofs, and vegetation can help reduce Urban Heat Island effects by shading building surfaces, deflecting radiation from the sun, and releasing moisture into the atmosphere.

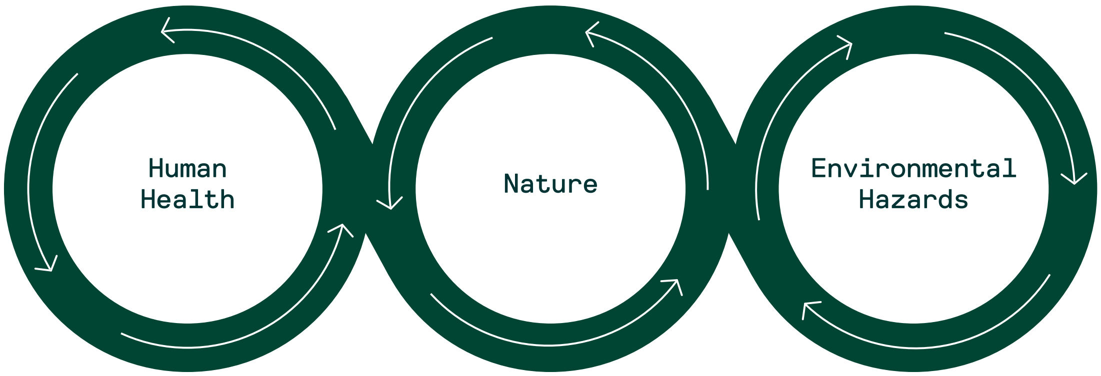
As the image below shows, a lack of natural elements can provide an excellent predictor of where Urban Heat Islands may occur. Note that the areas with the least vegetation cover are generally the hottest; red areas are hotter while blue areas are cooler.





NatureQuant analyzed data from 4,165 census tracts using the CalEPA Urban Heat Island Index and found that the NatureScore™ by census tract predicted the prevalence of Urban Heat Islands. In coastal cities with consistent coastal winds, the correlations were weaker (R: 0.22), but for inland cities, a stronger connection was found (R: 0.38). While many factors influence the existence of an Urban Heat Island, NatureScore™ can provide a helpful proxy.

While it is now understood that nearby nature bestows powerful health benefits, the mitigation of Urban Heat Islands is another reason for individuals, city planners, and businesses to track and monitor nature.



Nature Access is a Health Infrastructure Tool and Human Right

Inequities in nature access are particularly concerning because nature is not an amenity but a necessity for everyone's health and well-being. In the places where human activities in the United States have destroyed the most nature, fewer trees filter the air and provide shade on a hot day; fewer wetlands and marshes clean the water and protect communities from floods; fewer parks offer children a place to play and adults to unwind; and fewer public spaces invite all people to forge a strong community and build solidarity.

Most existing models, like the EPA EJSCREEN or CalEnviroScreen, do an excellent job of matching sensitive populations and environmental hazards (like pollution); but they largely ignore the direct and clear benefits that proximity to nature can provide.

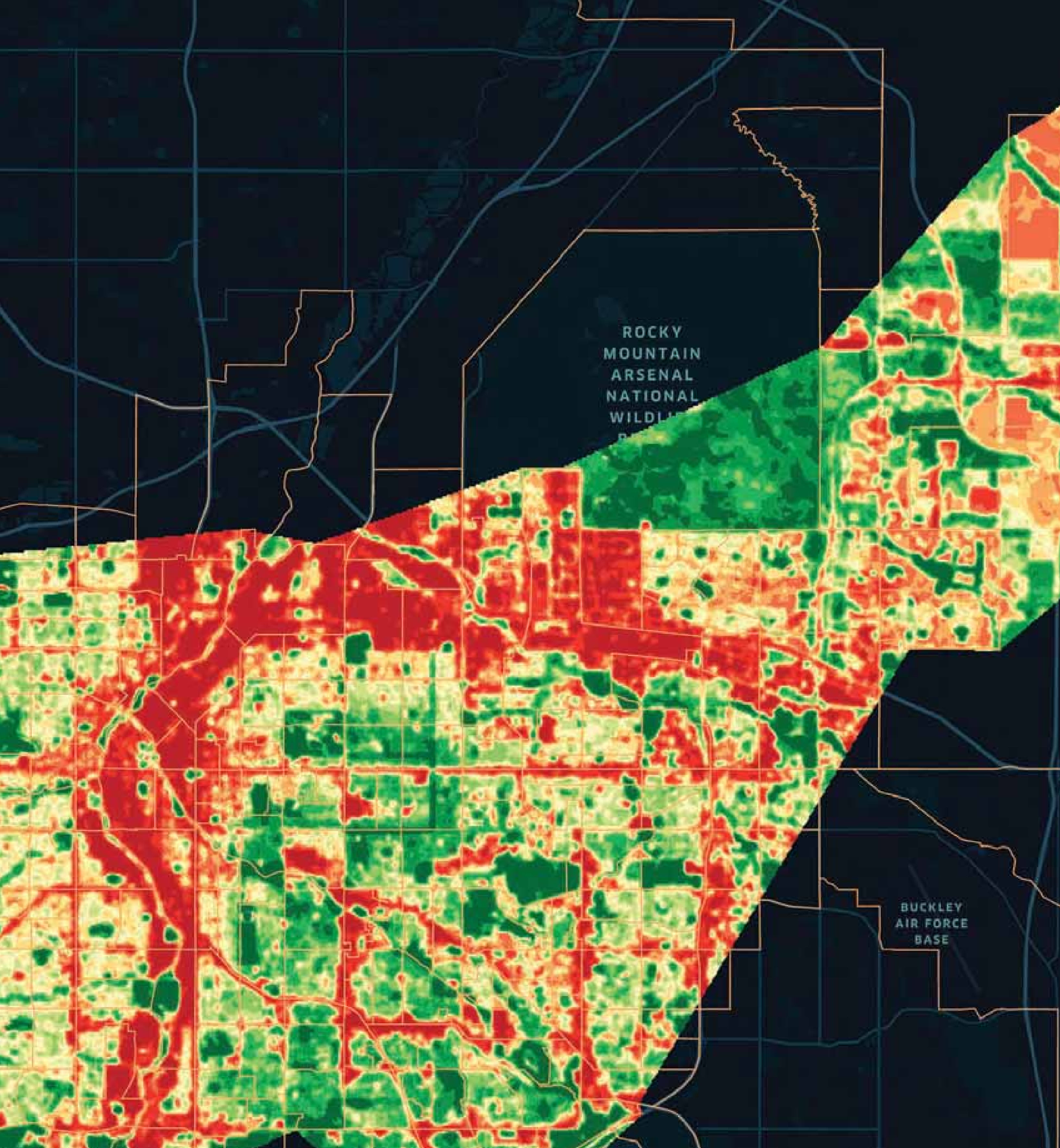
> Given the clear connections between nature and health, most Environmental Justice frameworks must include a measurement for nearby-nature (like the NatureScore™).

The Nature Quantification Solution:

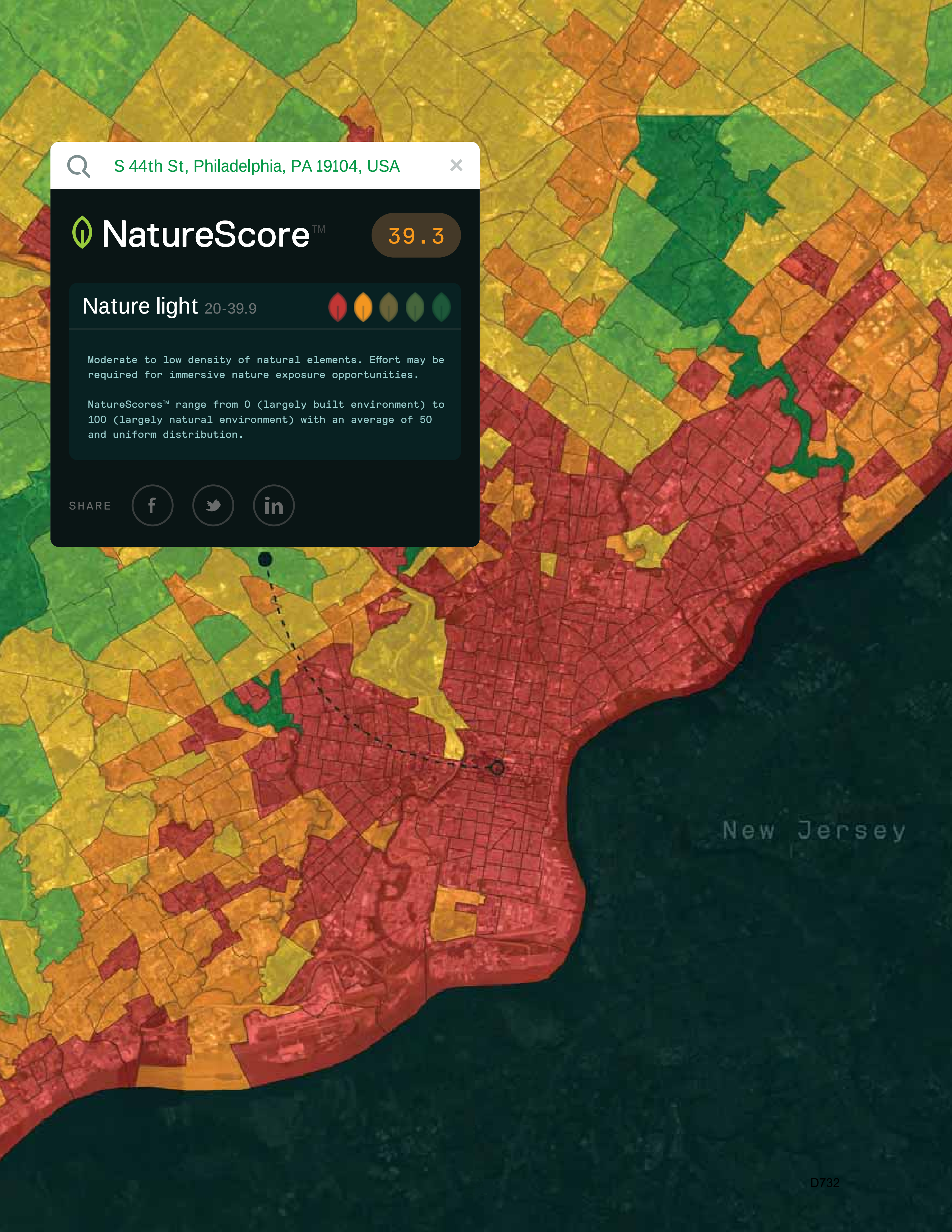
NatureScore™

NatureScore™ measures the amount and quality of natural elements for any location using a patent-pending system. For each physical address, NatureQuant analyzes and blends various data sets and processed information within a given radius, including satellite infrared measurements, GIS and land classifications, park data and features, tree canopies, air, noise, and light pollutions, and computer vision elements (aerial and street images).

The considered elements are weighted to create the highest correlation with the predictive health impacts of given natural elements via a machine learning process. Note that certain "natural" elements that have not demonstrated positive health correlations, like sand or rock, therefore do not contribute to a high NatureScore like live vegetation.



NatureQuant has aggregated precise NatureScore™ measurements by location to provide averages for every census tract in the United States. This data can help identify nature deficient neighborhoods.


S 44th St, Philadelphia, PA 19104, USA
NatureScore™
39.3
Nature light 20-39.9
Moderate to low density of natural elements. Effort may be required for immersive nature exposure opportunities.
NatureScores™ range from 0 (largely built environment) to 100 (largely natural environment) with an average of 50 and uniform distribution.
SHARE
New Jersey
D732

IT IS CLEAR:

Nature exposure is not a luxury. Nature is a necessity.

NatureScore™

Learn more about the NatureScore™ system:

NatureScore™

Whitepaper

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Navajo Birth Cohort Study initiated in 2010 to address the impacts of uranium exposure on child health outcomes

Birth Outcomes, Child Development

- **Home Environmental Assessment**
  - **Locations of nearly 600 homes**
  - **Indoor dust**
  - Radon
  - Gamma survey indoors and outdoors
  - **Drinking water**
- **Enrollment Survey**
  - Occupational history
  - Activity Survey
  - Family history of exposures
- **Biomonitoring (mom, baby)**
  - **Urine metals (36-element panel)**
  - **Whole blood (Pb, Cd, total Hg)**
  - **Serum (Cu, Se, Zn)**

Sample Collection Timepoints

	Blood	**Urine**
Mother	▪ Enrollment ▪ Delivery	▪ Enrollment ▪ Delivery
Father	▪ Enrollment	▪ Enrollment
Baby	▪ Birth (cord blood) ▪ 2-6 months ▪ 12 months	▪ Birth ▪ 2-6 months ▪ 12 months

NBCS → NBCS/ECHO

NBCS
Navajo
Birth Cohort Study

ECHO
Environmental influences
on Child Health Outcomes
A program supported by the NIH

NBCS (2010-2018)

Enrolled 780 women during pregnancy, exposure assessment, assessment of child development through 1 year of age

NBCS/ECHO

Enrolled 481 (179 pregnant mothers and 302 children from NBCS

NBCS-ECHO Plus – 2019-2024

- Continue enrollment to 1200 (316 children and 20 new pregnant mothers)
- Add common elements developed by ECHO consortium (allows us to compare exposures/outcomes with national sample
- Assessment continues through the age of 9

ECHO (Environmental influences on Child Health Outcomes

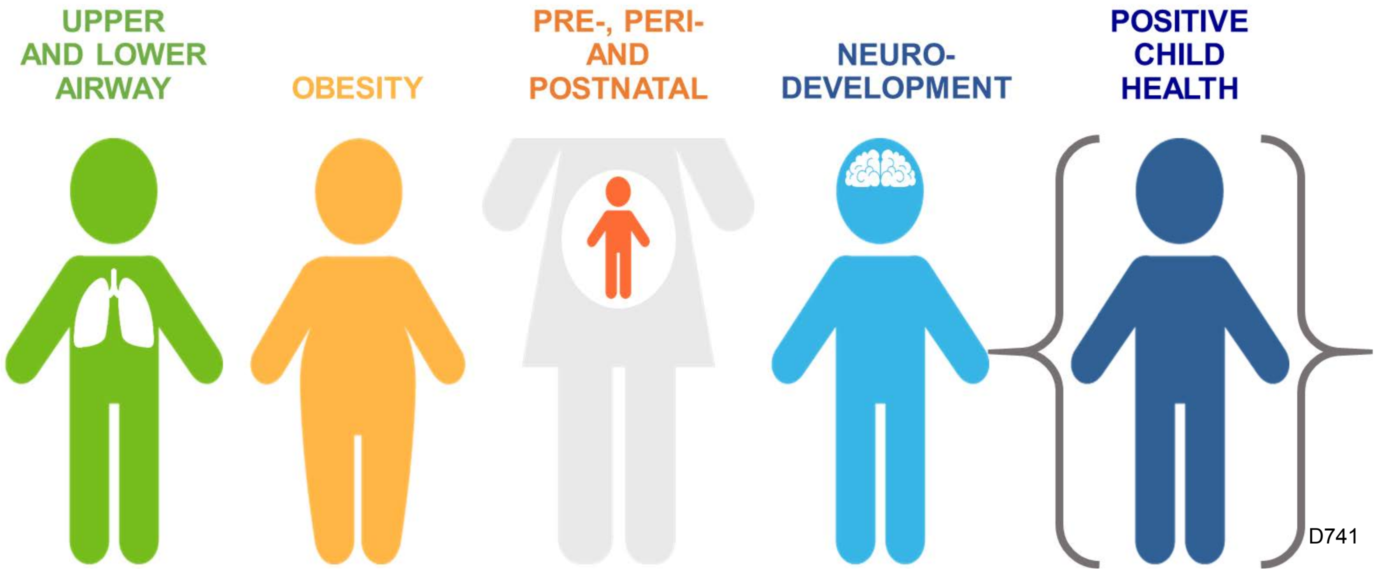
MISSION:
To enhance the health of children for generations to come

VISION:
To become one of our nation's pre-eminent research programs in child health

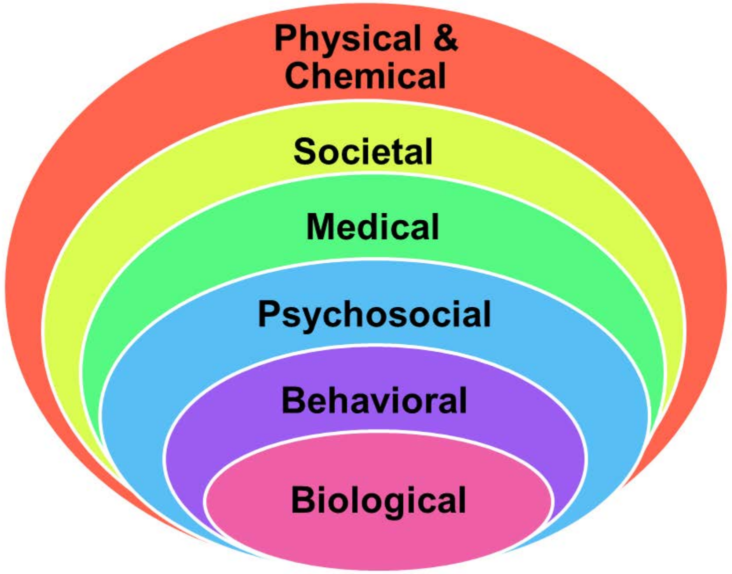
LONG-TERM GOALS:
Scientific: To inform high-impact programs, policies, and practices that improve child health

Strategic: To establish best practices for how to conduct Team Science in the 21st century

Focus on key pediatric outcomes



Data Collection



Pregnancy

- Surveys and questionnaires
- Blood and urine for metals

At delivery:

- Collect blood and urine for metals

From birth through 8 years of age:

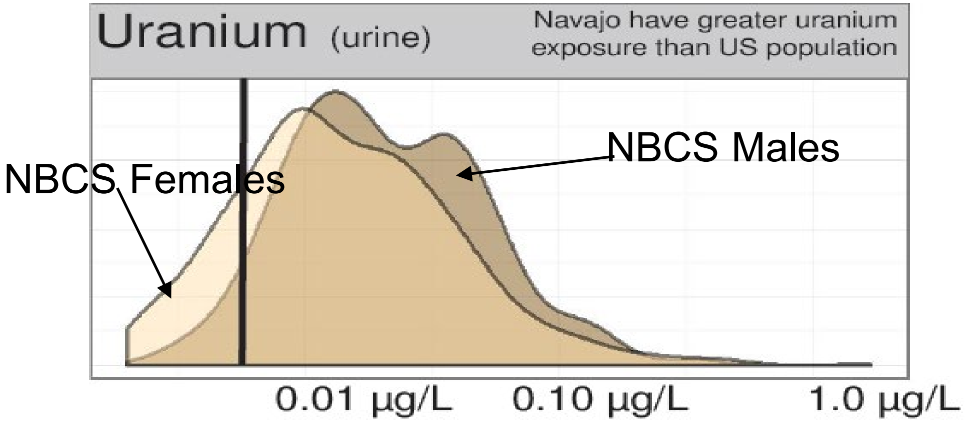
- Collect data from surveys and questionnaires
- Collect blood and urine every year
- Annual ASQ assessment
- Between the ages of 3-5 and again between the ages of 6-8 we conduct physical and neurodevelopmental assessments.

Art by Mallery Quetawki

Exposures seen from biomonitoring of key metals

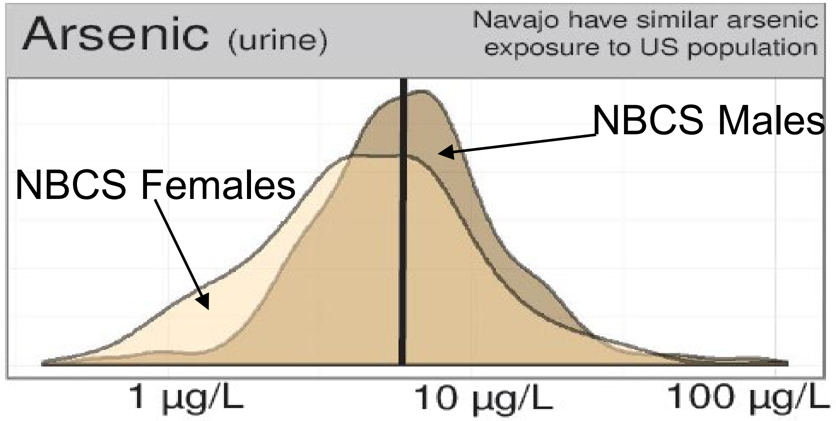
Uranium (kidney toxicity; estrogen mimicker)

- Black vertical line represents the 50th percentile for US population
- NBCS median urinary uranium concentrations exceed the US median (36% of men and 26% of women have urine uranium above national norms



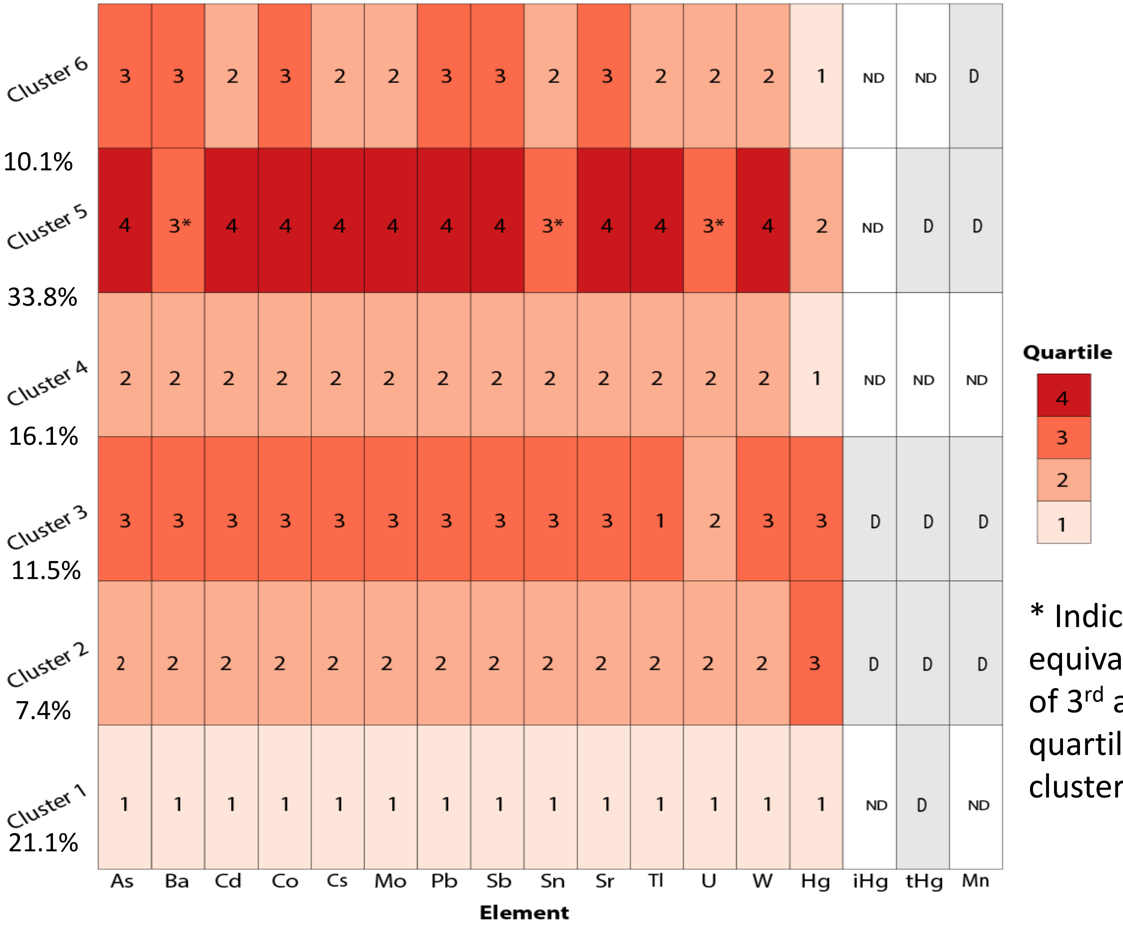
Arsenic (cancer, immunotoxicity)

- Distribution of urine total arsenic in NBCS females and males
- NBCS median urinary total arsenic concentrations are similar to the US median
- Exposure sources very different – in US, population exposures primarily seafood, rice



Exposures reflect patterns of mixtures

- More than 20% of moms have low exposures
- Overall rate of preterm birth in cohort 7%
- ~45% have mixture exposures that create a 3-fold greater risk of preterm birth (clusters 5 & 6)
- Mercury modulates the risk downward



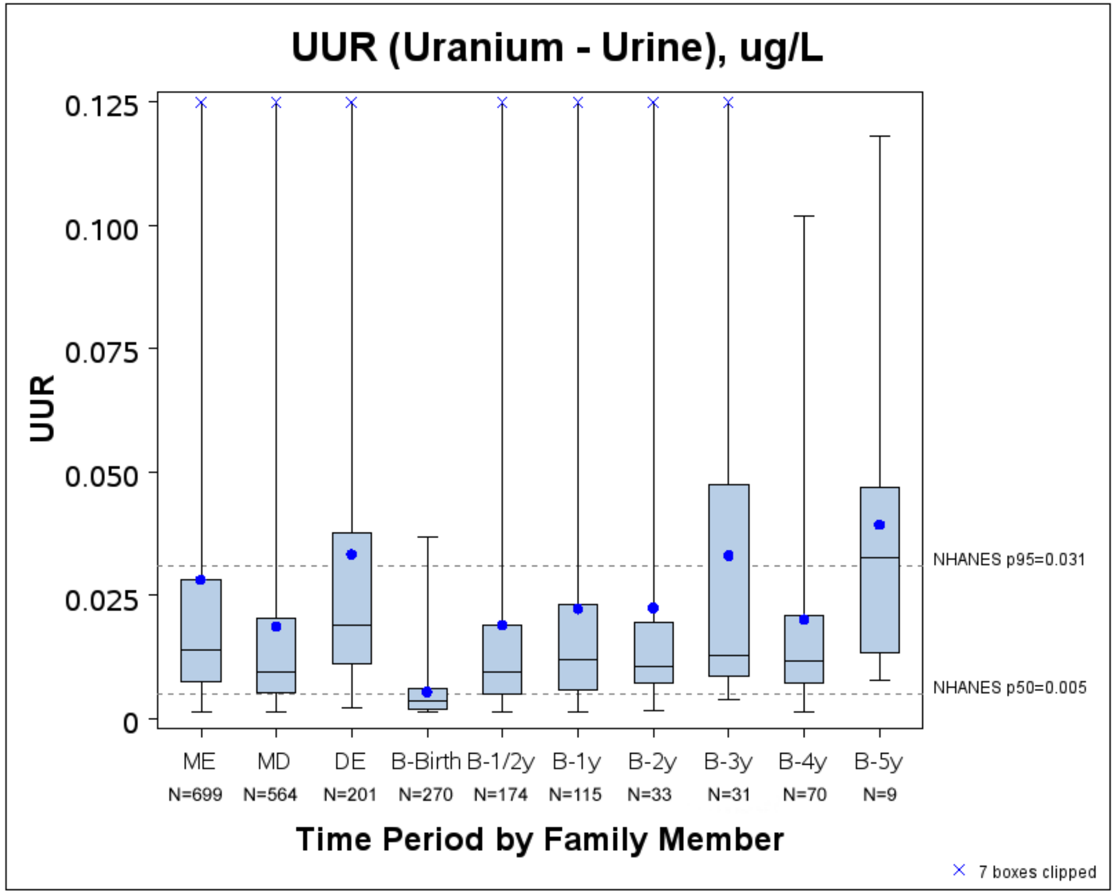
* Indicates equivalence of 3rd and 4th quartiles in cluster

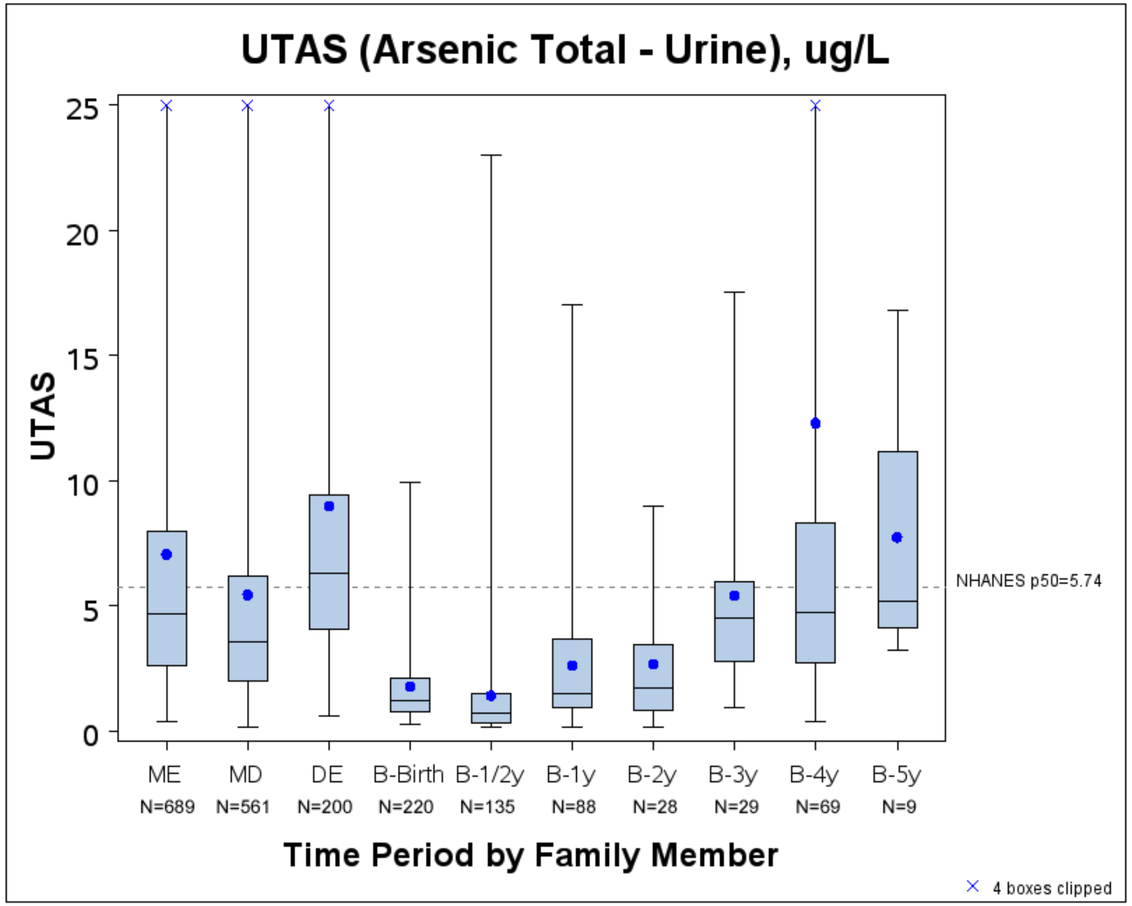
Summary of mean posterior probability from the fully adjusted model and relative risk of preterm birth by exposure cluster

Exposure Cluster	Group Size (N)	Empirical Probability	Mean Posterior Probability (95% CI)	Relative Risk (95% CI)	Probability EC_i>EC1
1	88	0.034	0.045 (0.018-0.081)	*Reference Group*	*Reference Group*
2	31	0.032	0.049 (0.012-0.109)	1.362 (0.25-3.638)	50.46
3	48	0.042	0.059 (0.023-0.108)	1.647 (0.44-3.936)	65.97
4	67	0.090	0.093 (0.049-0.148)	2.587 (0.9-5.678)	92.57
5	141	0.092	0.097 (0.065-0.134)	2.706 (1.059-5.768)	96.26
6	42	0.119	0.117 (0.058-0.19)	3.295 (1.046-7.437)	95.74

*Posterior probability >0.95 that EC is above 1 compared to reference cluster (EC2).

NBCS/ECHO: Exposures begin in childhood
By age 4, children are reaching adult concentrations





- Median concentration for urine uranium in the US ***adult*** population from NHANES (2015-16) = (0.005 μg/L)
- NBCS children birth to age 4 = 0.0035 – 0.013 μg/L

- Median concentration for total arsenic in urine in the US ***adult*** population from NHANES (2015-16) = (5.41 μg/L)
- NBCS children birth to age 4 = 1.2 – 4.5 μg/L

D745

Detailed Neurodevelopmental Assessments (between ages of 3-5 and again at 7-8)

Domain	Measure
Cognitive	DAS-II
Language	OWLS-2
Adaptive skills	Vineland
Social-Emotional	CBCL, SRS-2 (questionnaires)
Behavioral Observation	TOF, CARS-2
Medical	Medical and Developmental History, Physical Exam
Social cognitive functioning	Eye tracking measure

ND Assessment Summary

- Navajo preschoolers performed within the average ranges across multiple direct assessments and parent-report measures, except on the verbal domains across both modalities.

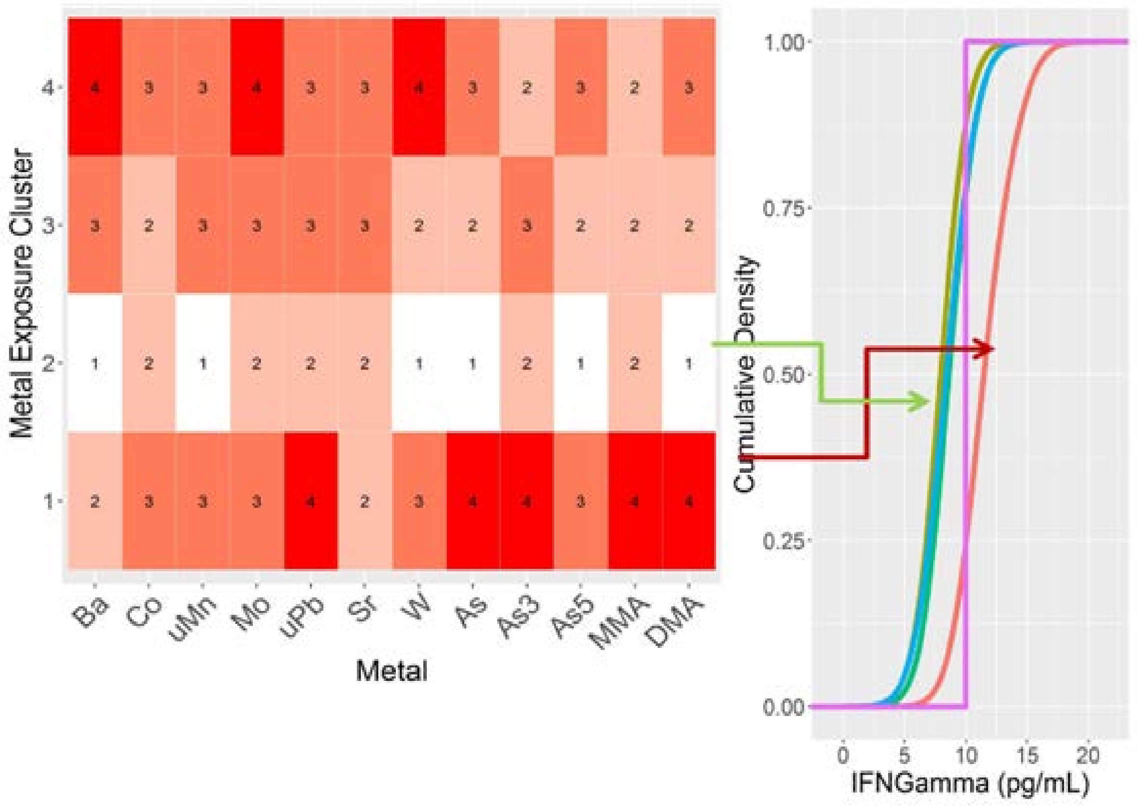
- High prevalence of language disorder independent of intellectual disability, general developmental delay, and autism spectrum disorder (validity of test instrument?, other reasons?)

Associations between metal exposures and inflammatory responses in NBCS Mothers

- Maternal inflammation has been associated with increased risk of having a child with a neurodevelopmental disorder

- Biomarkers of inflammation are associated with chronic health conditions including cardiovascular disease, cancer, diabetes, chronic kidney disease and autoimmune disorders

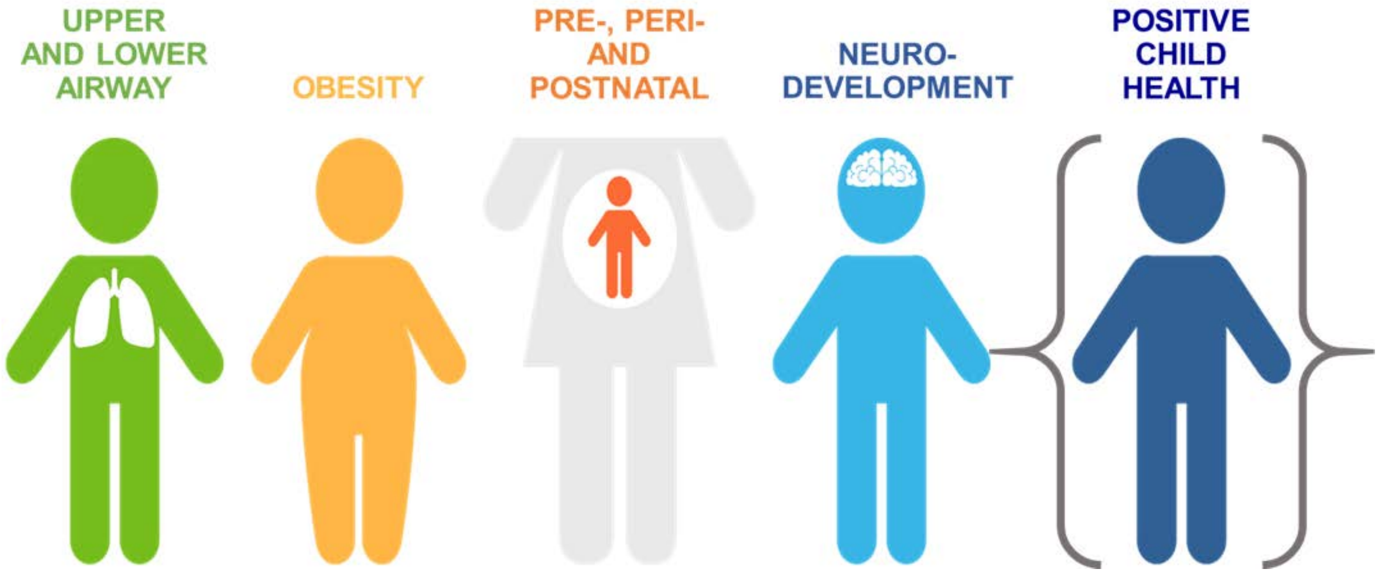
Inflammatory marker levels vary by metal exposure clusters



D748

Continuation of study……

- Assess the relationship between exposures and ND and other health outcomes.
- Compare NBCS to national sample to increase understanding of the influence of early life environmental exposures on health trajectory of Navajo children.



NBCS
Navajo
Birth Cohort Study

ECHO
Environmental influences
on Child Health Outcomes

NBCS-ECHO Flow Chart - basis for discussing most appropriate interactions for NBCS-ECHO

Recruitment

- In hospital Ob/Gyn waiting room
- Literature in Peds clinics
- Currently done by UNM or SRIC staff &/or NNDOH CHER

Enrollment

- Verification of pregnancy and notification of enrollment- NBCS-ECHO staff
- Consent & HiPAA – requires a private space (partitioned, office or conference room); done by UNM,NNDOH & SRIC staffs

Biological Sample Collection (Enrollment)

- Blood and urine collected in lab (or in home by NBCS staff) and processed by hospital lab staff trained by UNM

Biological Sample Collection (36 wk/ Delivery)

- Mom urine during 36 week visit to avoid blood contamination
- Mom blood at admission to L&D
- Cord blood and meconium during delivery by L&D staff

Infant Biospecimen Collection

- 2-6 months well-baby visit- NBCS blood and urine in peds or lab trained by UNM staff
- 12 mo urine and blood – peds or lab trained by UNM staff
- If collected during home visit, NBCS-ECHO staff coordinates with lab for processing

Medical Records Abstraction

- Performed by NBCS staff for Mother during pregnancy
- Mother at delivery
- Baby through 8th year

Annual child collections in ECHO (age 2-8)

- Surveys done in clinic or at home by UNM, SRIC and/or NNDOH staff
- Lab samples collected in peds or in lab
- If specimens collected during home visit, NBCS-ECHO staff coordinates with lab for processing

Note:

- All sample collection and processing done by lab staff occurred with intensive UNM training/certification.
- Currently there are 4 developmental assessments, often done by UNM or SRIC staff will conduct all in NBCS-ECHO, either in our space, pediatrics, or home
- UNM/SRIC/NNDOH staff will include phlebotomists and be trained to collect urine samples in home as well.
- NBCS-ECHO staff will also be trained in sample processing
- NBCS-ECHO staff will also be trained in med record abstraction
- **Neurodevelopmental Assessments (age 3-5 and 7-8), completed by UNM and UCSF clinicians. These assessments are completed for several participants over a 2-3 day visit where the clinicians can utilize our office space, an available conference room or exam room and the NNDOH CHR office.**

My name is Rick North. I'm the former executive vice president (CEO) of the Oregon American Cancer Society and former project director for the Oregon Physicians for Social Responsibility. Now retired, I have over 30 years' experience in nonprofit health and environmental management.

Most of my life I believed the CDC's and American Dental Association's assertions that water fluoridation was "safe and effective." When I actually examined the science, I was taken aback. Fluoridation's effectiveness was minimal, at best, and there were numerous associated health risks, as identified by the National Academy of Science's (NAS) authoritative 2006 review, Fluoride in Drinking Water (https://www.nap.edu/catalog/11571/fluoride-in-drinking-water-a-scientific-review-of-epas-standards). Since its publication, hundreds of other peer-reviewed, published studies have added even more evidence of these risks.

My main purpose in writing is to document the evidence that fluoridation *harms, not helps,* low-income families.

First, please consider fluoridation's lack of effectiveness and clear evidence that fluoride's preventive actions are mainly topical, *not* ingested.

The Cochrane Collaboration is considered the gold standard for evaluating effectiveness of medical interventions. Its 2015 report on fluoridation (https://www.cochranelibrary.com/cdsr/doi/10.1002/14651858.CD010856.pub2/full) found 4,677 published studies in its exhaustive literature search. Of those, only 277 studies qualified for the first round of review, with 155 meeting Cochrane's highest quality criteria for inclusion in the study.

The report concluded **"There is insufficient evidence to determine whether water fluoridation results in a change in disparities in caries levels across socio-economic status."** (https://fluoridealert.org/articles/fan-brochure-fluoridation-efficacy-one-pager/)

CDC itself has said **". . . fluoride prevents dental caries predominately after eruption of the tooth into the mouth, and its actions primarily are topical for both adults and children."** (https://www.cdc.gov/mmwr/preview/mmwrhtml/mm4841a1.htm) Even CDC's belief that fluoridation results in a 25% reduction of caries, which in itself is highly questionable, only equates to half a cavity per child.

Finally, World Health Organization data clearly show that cavity rates in children have dropped as much in nations that don't fluoridate as in nations that do. (https://fluoridealert.org/issues/caries/who-data/)

Then consider ingested fluoride's health risks, which, as cited in NAS's Fluoride in Drinking Water, include brain damage, hypothyroidism, kidney damage, diabetes and fluorosis. To take just one example, neurotoxicity, please note the National Toxicology Program's 2020 systematic review (https://fluoridealert.org/wp-content/uploads/ntp.revised-monograph.9-16-2020.pdf), which found compelling data linking fluoride to IQ loss in children. Several of the most recent studies were on pregnant women consuming fluoride or babies being fed formula mixed with fluoridated water:

– **25 of 27** of the studies determined to be the highest quality linked higher fluoride levels to substantially lower IQs

– **11 of 11** studies detected this IQ loss at levels found in fluoridated water

Low-income families also use more infant formula. A 2019 high-level Canadian study funded by the U.S. National Institutes of Health determined that babies fed formula mixed with fluoridated water averaged 4 IQ points less than those mixed with non-fluoridated water, 9 points lower in non-verbal IQ. (https://www.sciencedirect.com/science/article/pii/S0160412019326145?via%3Dihub)

The chances for academic achievement for low-income children and future success as adults are already diminished by poor nutrition and other environmental pollutants, such as lead in pipes, as shown in Flint, MI and other cities. It is not right to expose them to another toxic substance further reducing their capabilities.

Cheap water filters don't eliminate fluoride. Low-income families can't afford expensive filters (typically at least $300/$400) or bottled water to avoid the health risks of fluoridated water. *They have no choice*. And since Black and Hispanic families are more likely to be below the poverty level, they are disproportionately harmed. Such notable Black civil rights leaders as former U.N. ambassador Andrew Young, Rev. Gerald Durley and Rev. Bernice King, daughter of Martin Luther King, Jr., have publicly opposed fluoridation. And LULAC, the nation's oldest and largest Hispanic advocacy organization, is also formally opposed. (http://fluoridealert.org/wp-content/uploads/FAN-Environmental-Justice-Brochure-Final.pdf)

If people want fluoride, they can get it very inexpensively in toothpaste or mouthwash and apply it topically, where it's most effective. But no one should have the right to force anyone else to ingest a drug that they don't want in their drinking water. This is clearly a social and environmental justice issue. Fluoridation is unethical for low-income families and should be ended immediately.

Thank you for your consideration.

Sincerely,

Rick North
Wellesley, MA
781-431-6419

Blue Grass Army Depot *(Richmond, KY)*

Source: Kentucky Environmental Foundation

Clean Harbors Colfax *(Colfax, LA)*

Source: Louisiana Department of Environmental Quality

Former Atlantic Fleet Weapons Training Area *(Vieques, PR)*

Source: Cease Fire Campaign

Holston Army Ammunition Plant *(Kingsport, TN)*

Source: Volunteers for Environmental Health and Justice

Radford Army Ammunition Plant *(Radford, VA)*

Source: EPA

Andersen Air Force Base Open Detonation Disposal Training *(Yigo, Guam)*

Source: Department of Defense

December 2010

Ohio Environmental Protection Agency

Division of Air Pollution Control

All Ohio Air Toxics Report

Statewide Air Toxics Study

Ted Strickland, Governor
Lee Fisher, Lt. Governor
Chris Korleski, Director

D757

STATE OF OHIO

ALL OHIO AIR TOXICS REPORT

DECEMBER 2010

PREPARED BY

AIR TOXICS UNIT

DIVISION OF AIR POLLUTION CONTROL

OHIO ENVIRONMENTAL PROTECTION AGENCY

D758

EXECUTIVE SUMMARY

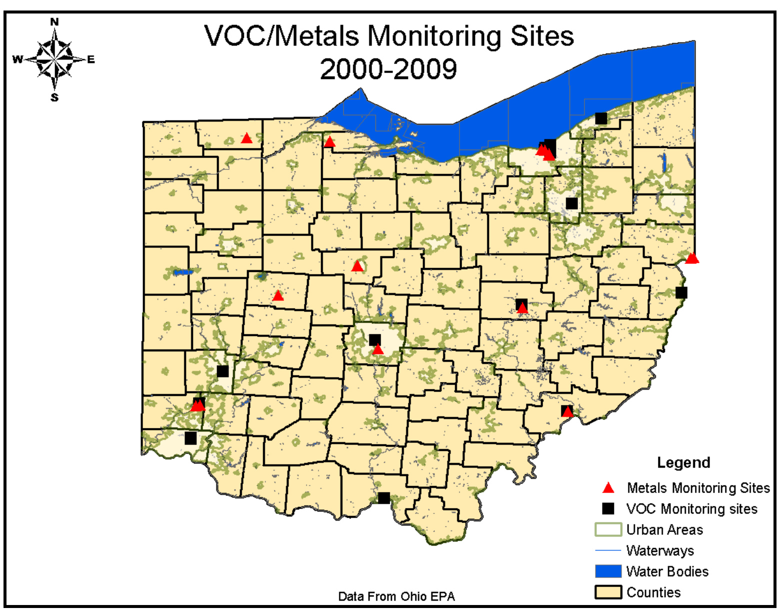
The Ohio EPA, Division of Air Pollution Control (DAPC), has completed the first Ohio statewide air toxics monitoring study. This study presents and summarizes the air monitoring efforts conducted by the division and provides information to the public regarding the levels of volatile organic compounds (VOCs) and heavy metals compounds measured throughout Ohio. For this report, staff evaluated ambient (outdoor) air monitoring data from 34 air toxics monitoring sites located in 16 Ohio counties.

Figure 1 shows the locations of the ambient monitoring sites evaluated in this study. The ambient air measurements were collected over approximately nine years. However, Ohio EPA collected samples for different time frames at various locations within this nine-year period. For example, some sites were sampled for three years (from 2000-2003 or 2004-2007), while other sites, such as the Washington County and Butler County sites were monitored for the entire nine-year period. Air sampling locations for heavy metals are represented by red triangles; locations for VOCs are represented by black squares.

Heavy metal sampling was conducted using a high-volume total suspended particulate (TSP) sampler, with 24-hour samples collected once every six days. Each group of filter samples was analyzed for the following metals: arsenic, beryllium, cadmium, and chromium, lead, nickel, manganese and zinc. Some of the filters were also analyzed for iron.

Most of the VOC samples were collected using a whole-air sampling system that pumps ambient air into an evacuated stainless steel canister. Samples were collected over a 24-hour time period at various intervals, as determined by the goal of each study. The ambient air samples were analyzed by Ohio EPA, Division of Environmental Services. More than 70 VOCs are typically targeted by the analytical system for identification and quantification.

Figure 1. Ohio Air Toxic Monitoring Sites



Air Toxic Risk Summary

In this report, the ambient air monitoring results are evaluated in an inhalation risk assessment. The risk assessment focuses on the direct inhalation of contaminants measured in ambient (outdoor) air for a given sampling period. This process uses U. S. EPA methods to estimate both the cancer and non-cancer health effects that individuals or populations may experience as a result of prolonged (long-term) exposure to toxic compounds. In an air pollution risk assessment, exposure is assumed to be constant inhalation (24 hours per day, 365 days per year, for 30-70 years) at the measured concentrations of air pollutants. This estimate centers on the additional lifetime risk predicted from the exposure being analyzed, beyond that due to any other factors. These estimates are calculated utilizing cancer and non-cancer potency factors that U.S. EPA considers to be plausible upper-bounds (i.e., actual risk may be lower, but sensitive populations and children may be at a higher risk).

Cancer risk is expressed as a probability usually represented in scientific notation as a negative exponent of 10. For example, an additional lifetime risk of contracting cancer of 1 chance in one million (or one additional person in 1,000,000 people) is written as $1x10^{-06}$ or 1.0 E-6. U.S. EPA has established a range of "acceptable" health risk values for carcinogens (cancer) compounds between one in 1,000,000 (1×10^{-06}) and one in 10,000 (1×10^{-04}) based upon feasible risk reduction strategies. While it is not always possible or feasible to remove all traces of a chemical released into the environment, Ohio EPA generally takes action to reduce the health risks associated with exposure to air pollutants that exceed the 1×10^{-04} risk level.

Non-cancer risks are evaluated by estimating the ratio of the individually measured concentrations to the no-effect level, known as the Hazard Quotient (HQ). The Hazard Quotients (HQs) can be added together to yield a total estimate of non-carcinogenic risk known as the Hazard Index (HI). Concentrations below these “no-effect” levels are generally regarded as "safe." Any individual non-cancer chemical that is above 100% (1.0) of the HQ is considered potentially unacceptable and merits further investigation.

The total cancer and non-cancer risks (HI) for each monitoring location are summarized in Tables 1 and 2 below. **Table 1** represents the chronic (long-term) cancer and non-cancer risk for **VOCs** and **Table 2** summarizes the cancer and non-cancer risk for **heavy metals**. Each table contains a list that separates current and historic sites in alphabetical order.

Table 1. Total Cancer and Non-Cancer Risk Estimates for VOCs

	County	Year	Address	Total HI	Total Risk
Current Sites					
	Butler/Verity School	2000–2009	1900 St. John's Rd.	0.52	6.03E-05
	Cuyahoga/Fire Station #11	2000–2009	7629 Broadway Ave.	0.56	8.22E-05
	Cuyahoga/Fire Station #22	2000–2009	7300 Superior Ave.	0.50	6.75E-05
	Cuyahoga/St. Theo.	2006- 2009	2547 St. Tikon	0.43	4.43E-05
	Cuyahoga/Tri-C College	2006-2009	2900 Community College Ave.	0.57	7.15E-05
	Franklin/OSU Fairgrounds	2003-2009	Korbel Ave.	0.49	7.33E-05
	Jefferson/SEDO	2004-2009	618 Logan St.	2.74	1.63E-04
	Washington/Washington County Career Center	2000-2009	Rt. 2	1.13	1.37E-04
Historic Sites					
	County	**Year**	**Address**	**Total HI**	**Total Risk**
	Coshocton/Coshocton Health Department	2000-2002	724 South 7th St.	0.59	4.89E-05
	Cuyahoga/G.T. Craig	2000-2003	E. 14th & Orange	0.81	8.383E-05
	Hamilton/Cincinnati State Community College	2003-2007	10100 Reading Rd.	0.55	9.81E-05
	Hamilton/Reading Pool	2000-2002	1601 West. St.	0.79	1.12E-04
	Lake/Lake County Hospital	2000-2004	71 E. High St.	0.49	6.76E-05
	Montgomery/Downtown Dayton Library	2003-2004	215 E. Third St.	0.87	1.13E-04
	Scioto/Portsmouth Water Treatment Plant	2000-2003	4862 Gallia	1.21	1.01E-04
	Summit/Akron Region Air Quality Management District	2000-2003	80 Brittain Rd.	0.59	5.67E-05

Table 2. Total Cancer and Non-Cancer Risk Estimates for Heavy Metals

Current Sites					
	County	**Year**	**Address**	**Total HI**	**Total Risk**
	Butler/Ohio Bell	2004-2009	3901 Jefferson Rd	1.16	5.03 E-05
	Columbiana/Maryland Ave	2001-2009	500 Maryland Ave	4.17	6.04 E-05
	Columbiana/Port Authority	2000-2009*	1250 St. George St	7.53	6.96 E-05
	Columbiana /Water Plant	2000-2009*	2220 Michigan Ave	32.28	2.10 E-04
	Cuyahoga/Ferro "A", Ferro “B”	2000-2009	4150 EAST 56th STR.	3.05	9.17 E-05
	Cuyahoga/Asphalt Plant A	2000-2009	West 3rd STR.	1.26	4.53 E-05
	Cuyahoga /Fortran Printing Inc.	2000-2009	5777 Grant Ave.	1.48	5.61 E-05
	Cuyahoga /St. Theodosius Church	2000-2009	2547 St. Tikhon Ave.	0.92	4.70 E-05
	Cuyahoga/Fire "4A", "4B"	2000-2009	3136 Lorain Ave.	0.44	4.20 E-05
	Franklin/ Woodrow	2000-2009	580 Woodrow	0.35	3.45 E-05
	Fulton	2000-2009	200 Van Buren St.	0.36	3.54 E-05
	Logan/Bellefontaine	2000-2009	1222 Superior Ave.	0.18	3.10 E-05
	Marion/Marion Steel	2000-2009	635 Bellefontaine / Gill Ave.	1.11	4.59 E-05
	Marion/Whitmore	2002-2009	441 Whitmore St.	3.22	1.39 E-04
	Ottawa/Brush Wellman 32	2002-2009	Brush Wellman 32	0.10	6.68 E-06
	Washington/Career Center	2000-2009	Rt. 676 Marietta	2.94	2.94 E-05

Historic Sites					
	County	**Year**	**Address**	**Total HI**	**Total Risk**
	Butler/ Oneida School I	2000-2002	Yankee Rd.	1.65	9.22 E-05
	Coshocton County/ Johnson Plumbing	2000-2002	840 Otsego Ave	1.08	5.76 E-05
	Cuyahoga/IMT	2000-2003	511 West 164th STR.	1.03	6.02 E-05
	Cuyahoga/Gate A	2001-2003	2850 3rd STR.	1.31	6.23 E-05
	Cuyahoga/Fire Station #11	2004-2006	7629 Broadway Ave.	0.76	5.91 E-05
	Cuyahoga/Fire Station #22	2004-2006	7300 Superior Ave.	0.44	8.90 E-06

The cancer risk estimates for most counties are within the 10^{-05} risk range. However, the total risk estimates for the following counties were slightly above the 10^{-04} cancer risk range: Columbiana, Hamilton, Jefferson, Marion, Montgomery, Scioto, and Washington. A detailed review of the cancer risk estimates revealed that the compounds acrylonitrile and benzene contribute to the majority of the risk from VOC compounds, while the chromium concentrations comprise the majority of the risk for the heavy metal compounds at each site.

For 13 out of 16 monitoring sites for VOCs, the total non-cancer risk estimates were below 100 percent. However, the non-cancer risk estimates for over half of the heavy metal monitoring sites were above the 100 percent non-cancer risk level. For each of the counties sampled, manganese comprises the majority of the risk for heavy metal compounds, while naphthalene comprises the majority of the risk for VOCs. Details regarding the air monitoring and risk assessment results are the subject of this Air Toxics Report. These estimates are being used by Ohio EPA to provide additional information to the public regarding Ohio's air quality and to assist the Agency in prioritizing areas in Ohio for further investigation and risk reduction.

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INTRODUCTION

The Ohio EPA, Division of Air Pollution Control (DAPC), has created this first statewide air toxics monitoring summary and analysis report for the State of Ohio. This summary report contains data from air toxics monitoring sites located throughout the state. The data documented in this report was collected by Ohio EPA field offices (five Ohio EPA district offices and nine local air agencies), as well as DAPC Central Office staff. This report is designed to provide information to citizens regarding the measured levels of toxic pollutants in their area, as well as a basic assessment of risk from potential airborne exposure to those pollutants. This report includes ambient air toxics monitoring data results for volatile organic compounds (VOCs) and heavy metal compounds.

This study was compiled and written by the Air Toxics Unit (ATU) of DAPC, with assistance from the Air Monitoring Section (AMS). All laboratory analysis performed on the collected ambient air samples was conducted at Ohio EPA's Division of Environmental Services (DES) laboratory, unless stated otherwise. The creation of this report complements the Ohio EPA's Urban Air Toxic Monitoring Program, initiated by U.S. EPA, to address the potential concerns for high cancer risks from multi-source, multi-pollutant interactions in urban areas. Many of the site results listed here are located in or near Ohio's urban areas that contain numerous industrial sources. These sources, when combined with mobile source emissions, contribute to the total amount of toxic emissions released into Ohio's outdoor air. Heavy metals, VOCs and Polycyclic Aromatic Hydrocarbons (PAHs) are routinely monitored throughout Ohio. Among other things, these measurements provide the opportunity to quantify the concentrations of toxic pollutants in many areas of the state; to assess the general risk to Ohioans from air pollution; to focus study on potential areas of high concentrations; and to document successful reductions of these compounds in Ohio.

In general, the summary of the ambient air measurements presented here span for a period of approximately nine years. Ohio EPA collected samples for different time frames within this nine-year period. For example, some sites were sampled for three years (from 2000-2003 or 2004-2007), while other sites were monitored for the entire time. Most of these sites were chosen either to be representative of a large area being studied as a whole, so as not to be dominated by a single industrial source or sources. Alternatively, some monitoring sites were selected as a result of either citizen requests at or around specific sources of air pollution, and others were selected by DAPC as part of specific air toxic investigations for air pollution research and/or health-study investigations. In some cases, Ohio EPA has provided ambient air monitoring at the request of other state, local and federal health agencies as part of data collection research for specific investigations. Detailed site descriptions will not be addressed in this report, but the information is available by contacting DAPC.

METHODS

VOLATILE ORGANIC COMPOUND SAMPLING AND ANALYSIS

A major component of this Report consists of ambient air sampling for volatile organic compounds (VOCs). These are defined as compounds that are generally found in the vapor (gas) state at normal temperatures. These compounds can be chlorinated or simple hydrocarbons. Most of the VOC samples collected in this study were collected using a whole-air sampling system that pumps ambient air into an evacuated stainless steel canister. The canister acts as a storage container which allows the air sample to be maintained virtually unchanged until it is analyzed. Samples were collected over a 24-hour time period at various intervals between days, as determined by the goal of each study. The specific procedures for VOC sampling and laboratory analysis can be found in the U.S. EPA document *Compendium of Methods for the Determination of Toxic Organic Compounds in Ambient Air* (section TO-14).

During analysis, the volatile tendency of VOC compounds allows them to be vaporized when heated (if not already in that form), and then injected into an analytical device called a gas chromatograph (GC). The typical analytical system used for this study utilized a GC with a special detector called a mass spectrometer (MS). As a sample passes through a GC column, the various compounds separate out of the sample mixture. The compounds exit the column individually, and the concentration of each is detected by the MS. These concentrations are illustrated as peaks on a chromatogram. The area of each peak indicates the concentration of the compound in the sample. Compound identification is accomplished by comparing the retention time of the peaks on chromatogram with those from a chromatogram of a known mixture of compounds. Retention time is the time it takes for a particular compound to reach the detector. As long as the analytical conditions remain the same, a compound from one analysis to the next will have the same retention time.

The combination of a GC/MS can be used to analyze a sample by separating it into its individual components which are then broken down into mass fragments, forming a fingerprint by which a compound can be identified as described above. Over 70 VOCs are targeted by the analytical system for identification and quantification. As the technology and method improves, additional compounds are added to the standard target list. However, if an unidentified compound of significant quantity exists in a sample, it can be tentatively identified during the MS analysis.

HEAVY METALS SAMPLING AND ANALYSIS

Sampling for heavy metals is conducted using a high volume total suspended particulate (TSP) sampler. With this sampler, particulate matter in the air is collected on a pre-weighed glass fiber filter. Sampling is done intermittently with 24-hour samples collected once every six days, although at times different schedules are used. The operating procedures can be found in the *Code of Federal Regulations, 40 CFR, Part 50, Appendix B - Reference Method for Determination of Suspended Particulate Matter in the Atmosphere.* For lead, *40 CFR Part 50, Appendix G* is used.

Filters collected at each site are generally analyzed as a monthly composite. The acid-extracted samples are analyzed by inductively coupled plasma (ICP), which has evolved to include inductively coupled plasma mass spectrometry (ICP-MS) to determine the concentration of the metals parameters in extracted filter sample. The acid extracted droplet of nebulized sample enters the central channel of the ICP, it evaporates and any solids that were dissolved in the liquid vaporize and then break down into atoms. At the temperatures prevailing in the plasma a significant proportion of the atoms of many chemical elements are ionized, each atom losing its most loosely-bound electron to form a singly charged ion. The ions from the plasma are extracted through a series of cones into a mass spectrometer, usually a quadrupole. The ions are separated on the basis of their mass-to-charge ratio and a detector receives an ion signal proportional to the concentration of the parameter to be determined.

METHOD OF ANALYSIS DETECTION LIMITS

The arithmetic mean (average) as listed in these tables contains values for non-detected compounds as half of the detection limit. The detection limit is the lowest measurement the collection and analysis procedure can accurately quantify as a true measurement of the ambient air concentration. Other options for handling values below the detection limit are available, such as using the detection limit itself, or using zero for the measurement result. In this study, the mean (average) concentration used for estimation of long-term exposure contains the value of half the detection limit for results below the lower detection limit of the method (non-detects). Using a value of zero, however, will tend to underestimate the true average, as many values could be well below the detection limit, but greater than zero. Ohio EPA uses the value of half the detection limit as reasonable compromise between overestimating or underestimating the true ambient air concentration of these compounds (which generally follows U.S. EPA guidelines). No values are recorded for compounds that were measured below the detection limit for an entire year (no detected values for 12 consecutive months). Results for non-detected compounds for annual reporting periods are not included in the result data tables.

HUMAN HEALTH RISK ASSESSMENT

Risk assessment is a process which uses current available toxicological information as a basis for estimating the health effects that individuals or populations may experience as a result of prolonged exposure to hazardous substances. Following the risk assessment guidelines provided by the U.S. EPA, Ohio EPA DAPC is providing screening level risk assessment information with selected results for Ohio’s data. The risk assessment process consists of four steps: hazard identification, toxicity assessment, exposure assessment, and risk characterization. Detailed explanations of the above steps have been provided in other Ohio EPA and U.S. EPA documents and will not be discussed in detail here. These steps, when combined, produce a numerical prediction of the probability of an adverse health effect, such as carcinogenicity and / or systematic toxicity, which may occur as a result of constant exposure to a single or multiple toxic compounds for a long period of time.

For the purpose of this study, it is assumed that a hypothetical individual is exposed constantly for 24-hours per day and 365-days per year to the average measured airborne concentrations of the measured pollutants. This assumption creates an exposure scenario for a theoretical person known as the "maximally exposed individual" (MEI). Health risks for the MEI are estimated in this study for the inhalation exposure pathway only. The MEI scenario provides the largest safety buffer in evaluating potential human health risks because the exposure assumptions for the MEI are very conservative, meaning the strictest assumptions are used to estimate the highest risk. The actual (real) health risk will be no greater than the estimated inhalation risk; in fact, the actual risk will most likely be less than those estimated in this study.

Ohio EPA risk assessment procedures follow general guidelines provided by the National Academy of Sciences (NAS) and U.S. EPA (*Risk Assessment Guidance for Superfund - Volume 1- Human Health, December 1989*) and the recently revised U.S. EPA Air Toxics Risk Assessment Reference Library (Volumes 1-3, April 2004). Risk-based screening levels are based upon U.S. EPA's integrated Risk Information System (IRIS) unit risk factors for carcinogens and reference concentrations (RfCs) for non-carcinogenic effects. Some chemicals, such as acrylonitrile, benzene, and 1,2-butadiene, are classified as hazardous because they have been implicated as potential carcinogens. Others, such as naphthalene, toluene, and manganese, are believed to potentially cause non-carcinogenic adverse health effects such as developmental, reproductive, neurobehavioral, or cardio-vascular health implications. The heavy metals of nickel compounds, beryllium, arsenic, cadmium and chromium compounds are classified as human carcinogens. Chromium VI is the most carcinogenic form of the chromium compounds measured.

INHALATION TOXICITY OF COMPOUNDS MEASURED

In order to quantify the toxicity of the chemicals detected and measured in Ohio's ambient air, Ohio EPA uses the toxicity classification method developed and used by U.S. EPA. The method and data for this risk assessment process are located in the U.S. EPA's IRIS website database (Integrated Risk Information System). This method defines a reference concentration (RfC) for non-carcinogenic compounds (compounds that are not known to cause cancer), or unit risk factors (URFs) for carcinogenic compounds (compounds known or suspected of causing cancer in animals or humans).

For carcinogens, the excess lifetime cancer risk resulting from the inhalation of toxic compounds is calculated by multiplying the monitored average ambient air concentrations by the appropriate URF. This initial calculation results is an estimate of the individual additional excess risk caused by breathing the compounds at the measured concentrations for a lifetime. Excess cancer risk is defined as the risk of a health event occurring above and beyond the other risk factors experienced by an individual member of the population, or the population as a whole (such as those caused by genetics, diet, lifestyle, etc.).

To depict total risk from a class of compounds (total VOCs, for example) the individual risk results are added (summed) to estimate the "total" risk for the site. The summation is based upon the principle that the addition of each risk produces a combined total risk estimate. This method is recommended by U.S. EPA in the absence of additional information. It has been suggested in scientific literature that exposure to combinations of pollutants may cause greater or lesser risk than can be explained by merely the summation of the risks from exposure to the substances individually. U.S. EPA currently has limited guidance on quantifying effects from exposure to mixtures of compounds measured in this study. Until further guidance is formulated, Ohio EPA will continue to add the risks associated with the compounds measured in a linear fashion. Note that this is potentially an extremely conservative method of assessing risk.

For non-carcinogenic adverse health effects, RfCs are estimates of daily exposure levels to which the human population, including sensitive subpopulations, may be exposed constantly for long periods (chronic exposure) without an appreciable risk of deleterious health effects. Chronic RfCs are especially developed to be protective for long-term exposures to the compounds.

The non-cancer risks are evaluated by the use of a Hazard Quotient (HQ) which is the ratio of the average daily concentrations compared to the RfC for each compound. If the HQ exceeds 100 percent for a compound, there may be cause for concern about potential health effects as a result of exposure to the compound. Hazard Quotients can be added together to yield a total estimate of non-carcinogenic risk called the Hazard Index (HI) (similar to the adding of carcinogenic risk to obtain a theoretical total).

RISK ESTIMATES IN PERSPECTIVE

For an accurate interpretation of the predicted risks, these risks must be placed in perspective with common daily activities. A range of "acceptable" health risk values for carcinogens has been historically proposed by U.S. EPA. Acceptability ranges from a target goal of one in one million (1×10^{-06}) for regulation of certain source categories of individual toxic air pollutants, to some source categories routinely operating in the one in 10,000 (1×10^{-04}) range. The goal of the federal Residual Risk Program of the Maximum Achievable Control Technology (MACT) standards is a maximum risk of 1×10^{-06} (one in one million) per source category. For some source categories, U.S. EPA is finding this goal difficult to achieve. Details of the MACT program are located in the Clean Air Act Amendments of 1990, Title III.

Similarly, non-carcinogenic estimates of the Hazard Indexes (HI), or an individual calculated HQ below one (100 percent) is generally regarded as a "safe" level of exposure. However, since the HI consists of the summation of many individual HQs, the HI can sometimes approach and/or exceed a value of one and still be considered acceptable.

These health effect evaluation numbers have many safety factors included in the calculations in order to present the most conservative estimate of human health risk. The addition of safety factors is designed to provide the most protective estimations in order to protect public health. The actual potential for human health risk will be no greater than the estimated risk(s); in fact, the actual risk will most likely be less than those estimated in this study.

Most large urban areas in Ohio and the United States exhibit aggregate or total carcinogenic risks in the 10^{-04} to 10^{-05} range. It is common for industrial or light industrial areas to have both cancer and non-cancer risks greater than those in urban areas.

Conversely, some smaller suburban and rural areas have combined carcinogenic risks in the 10^{-06} to 10^{-08} range. Areas of higher risk characteristically may also contain large volumes of congested automobile traffic. When considering long-term human risk, one must consider workers in more congested areas routinely are exposed only during working hours, and return to residential areas of decreased exposure concentrations during non-working hours. While it is certainly desirable (and Ohio EPA's goal) to have urban area aggregate risks minimized to the lowest possible level, a no-risk scenario would be impossible to achieve in a major urban area.

The tables in this report contain the calculations of excess cancer risk for each group of compounds or elements. The URFs as of July 2010 from the IRIS database were used in the calculations. For chemicals for which carcinogenicity is currently under review by U.S. EPA, Ohio EPA obtained values from the ATSDR toxicological information databases. Presently IRIS has many RfCs that are under review. For compounds with more limited toxicological evidence, U.S. EPA guidance suggests a general hierarchy for chronic toxicological data, beginning with 1) IRIS; 2) ATSDR toxicological profiles; (3) California Environmental Protection Agency (CalEPA); (4) International Agency for Research on Cancer (IARC) and the ACGIH (American Conference of Governmental and Industrial Hygienists) threshold limit values (TLVs); and finally, (5) other peer-reviewed literature studies.

RESULTS AND DISCUSSION

Ambient air toxics samples were collected over a 24-hour time period at various intervals (days), as determined by the goal(s) of each study. The site names and details of the locations for the ambient air monitoring sites used in this report are included in Figures 2 and 3 below. **Figure 2** contains the locations for the **VOC monitors** and **Figure 3** shows the locations for the **heavy metal (TSP) monitors**.

Figure 2. Ohio EPA VOC Monitoring Locations

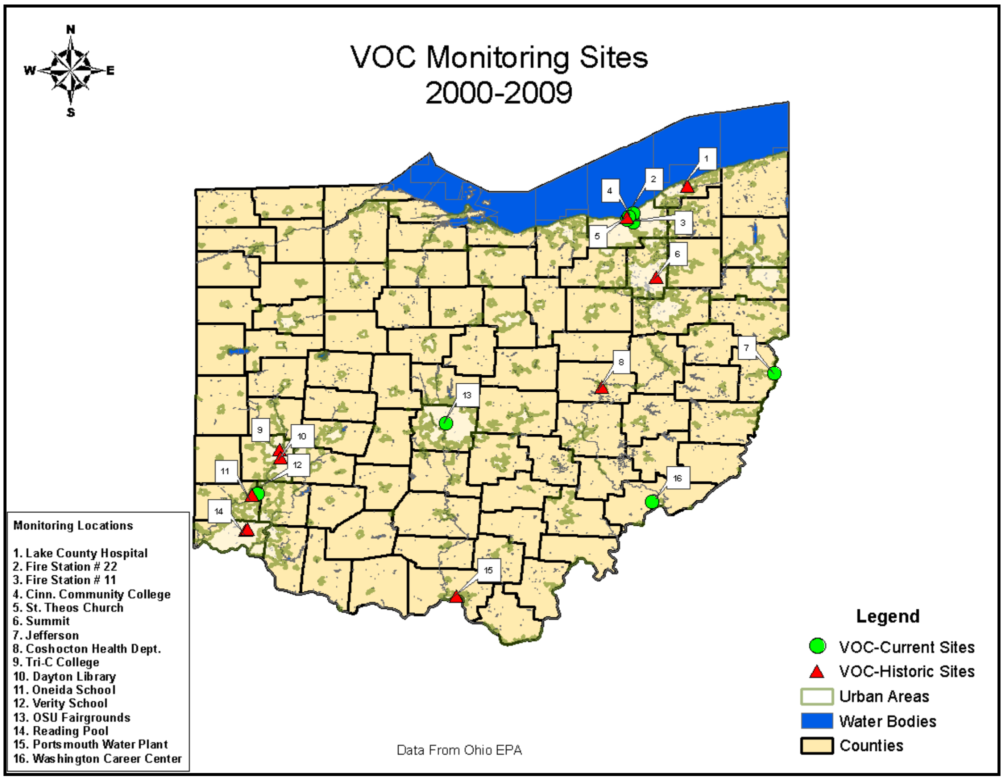
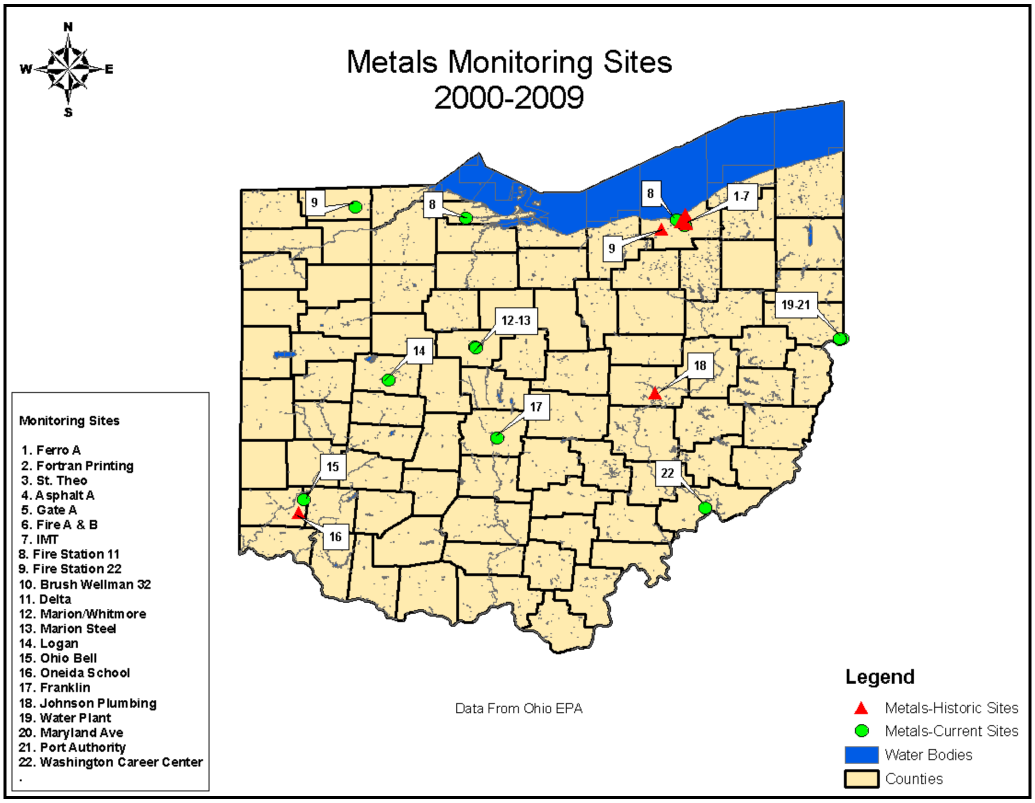


Figure 3. Ohio EPA Heavy Metals (TSP) Monitoring Locations



The general purpose for this report is to characterize the measured concentrations of air toxic pollutants in various areas in Ohio. Some sampling projects included in this report were initiated as a result of special investigations or other compliance-induced activities. Ohio EPA also conducts alternative air toxics sampling studies of shorter duration or sampling times that are not included here. As part of the analysis of the long-term projects included in this report, Ohio EPA created a summary of the resultant risk(s) associated with the average of the measured concentrations (risk assessment was briefly described above). The results are depicted as a breakdown of the risk for each county or sampling location (for counties containing more than one sampling location) for the years sampled. Tables or Figures also include an AIRs number (Aerometric Information Retrieval System - classification method of U.S. EPA) which serves as a specific and distinct identification number for the site location and monitoring data used in the U.S. EPA database. Readers interested in more information may access the database from U.S. EPA's website.

RISK ASSESSMENT RESULTS

The following Tables and Figures summarize and compare the total calculated excess cancer and non-cancer risk(s) from potential exposure to measured concentrations of airborne metals and VOCs for 14 Ohio counties. Figures 4 through Figure 7 (below) compare the cancer and non-cancer risk estimates measured for each county. The figures separate historic monitoring sites from those currently operating in Ohio.

For currently active sites, Jefferson County shows both the highest cancer and non-cancer risk estimates for VOC compounds. Risk estimates for heavy metals are presented in Figures 8 through11. The figures also separate currently active sites from historic measurements. Both figures show that Columbiana County currently represents the highest risk in Ohio for both cancer and non-cancer estimates.

Figure 4. Cancer Risk Estimates for Volatile Organic Compounds

Historic Sites

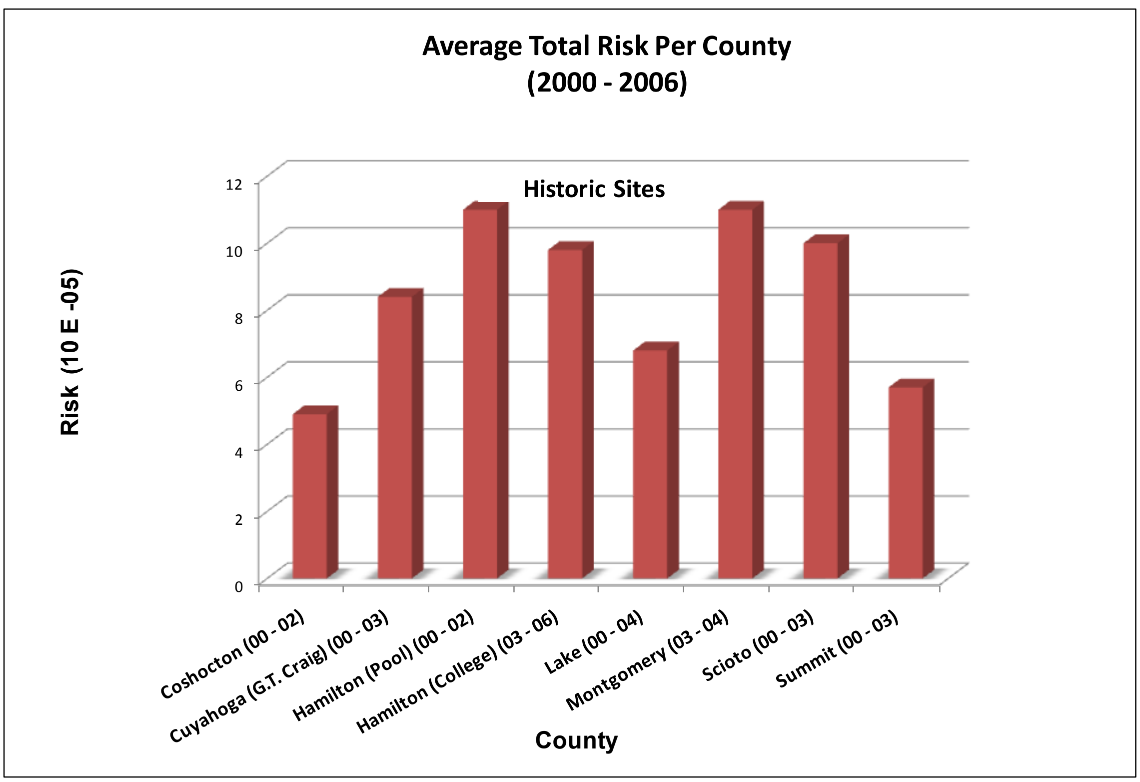


Figure 5. Cancer Risk Estimates for Volatile Organic Compounds

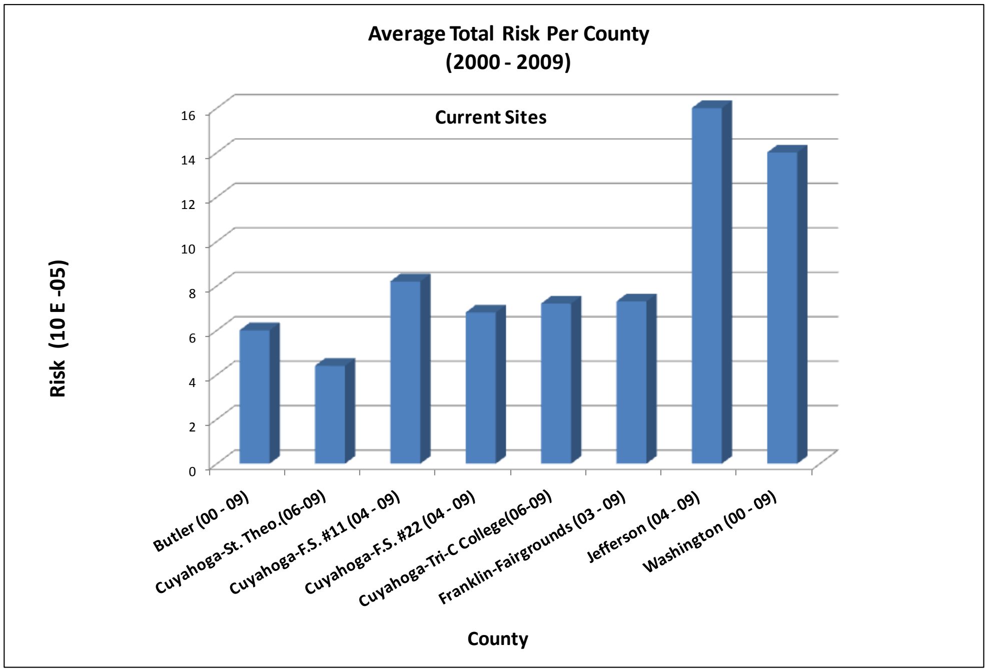


Figure 6. Non-Cancer Risk Estimates for Volatile Organic Compounds

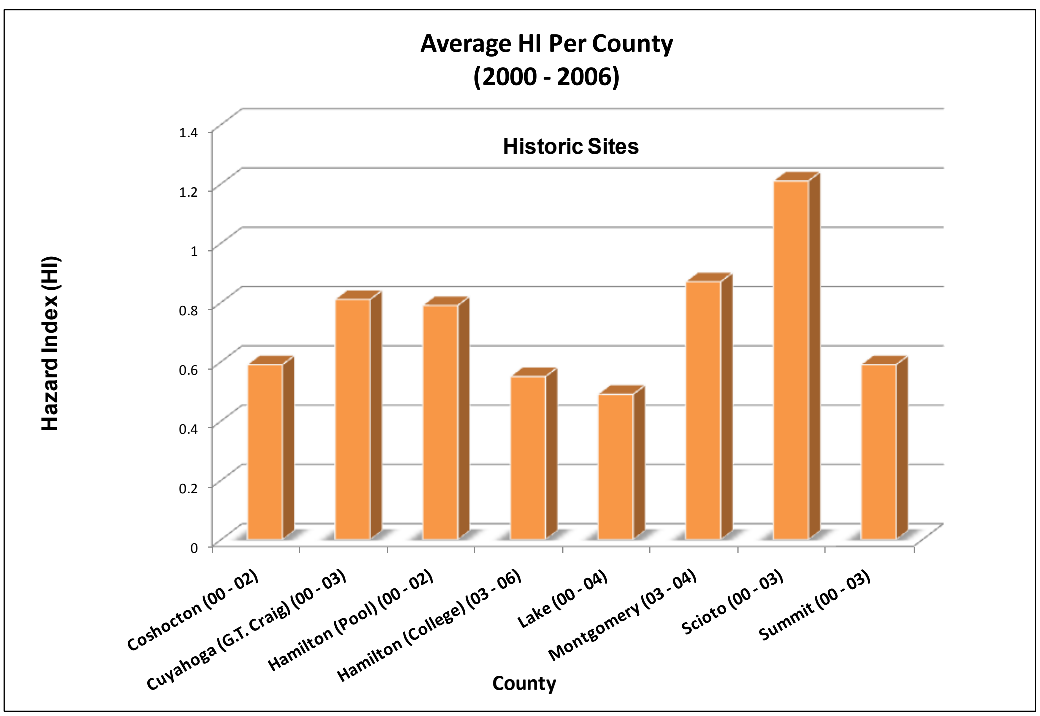


Figure 7. Non-Cancer Risk Estimates for Volatile Organic Compounds

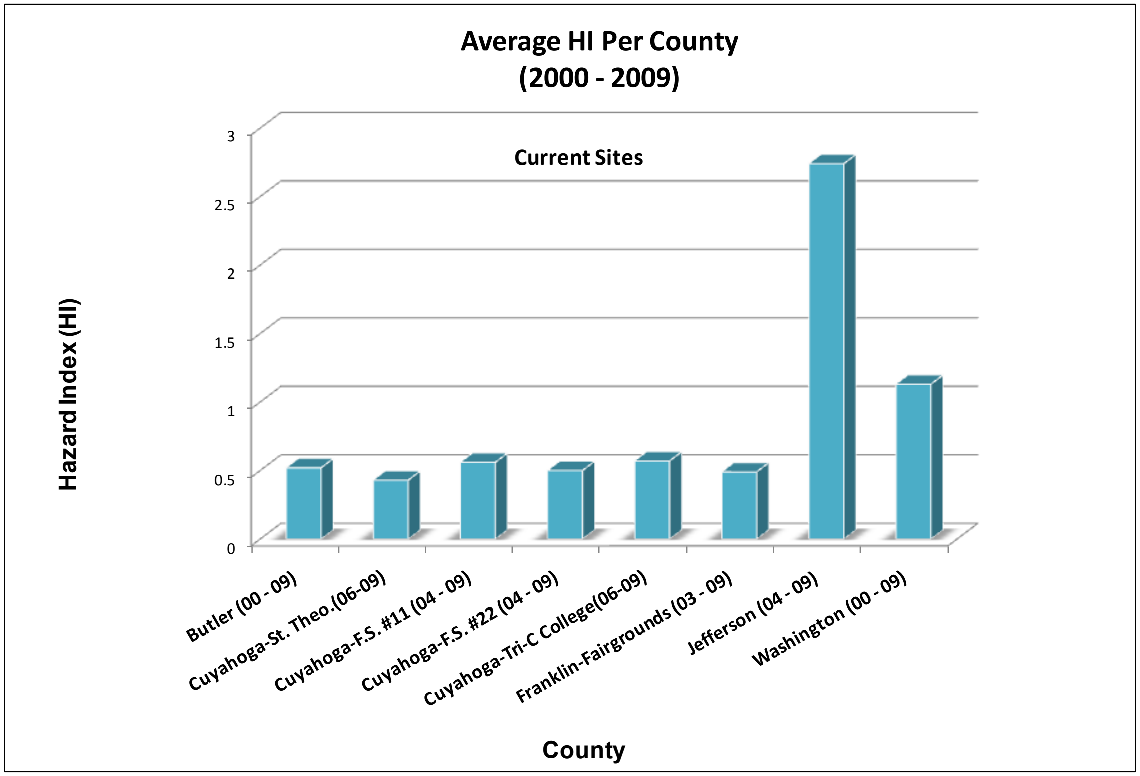


Figure 8. Cancer Risk Estimates for Heavy Metal Compounds

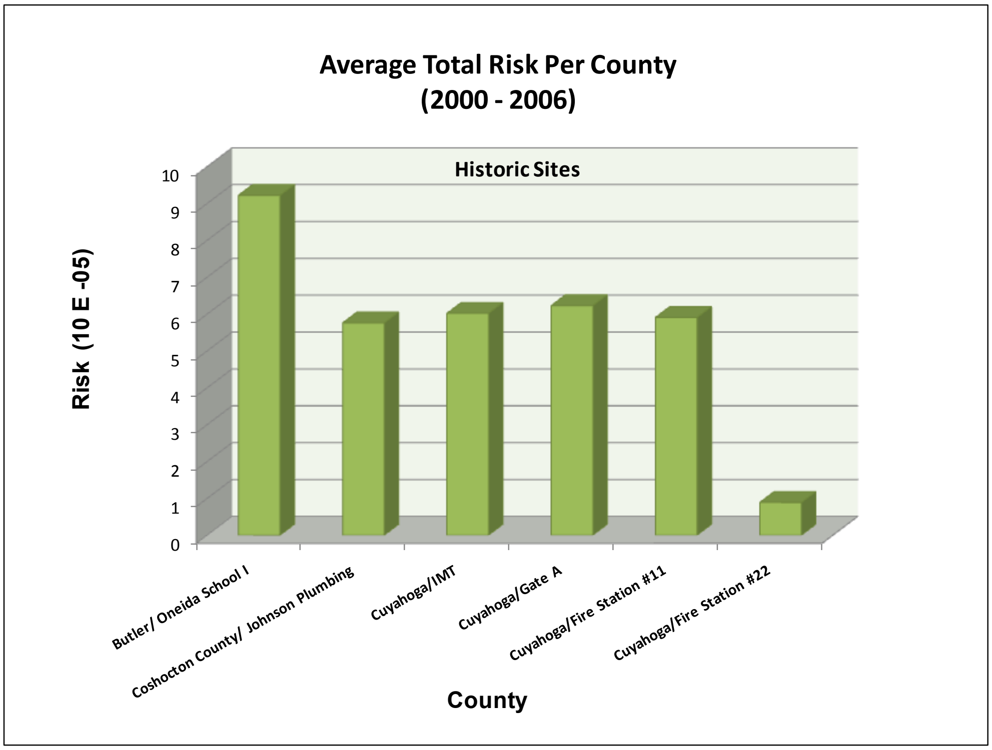


Figure 9. Cancer Risk Estimates for Heavy Metal Compounds

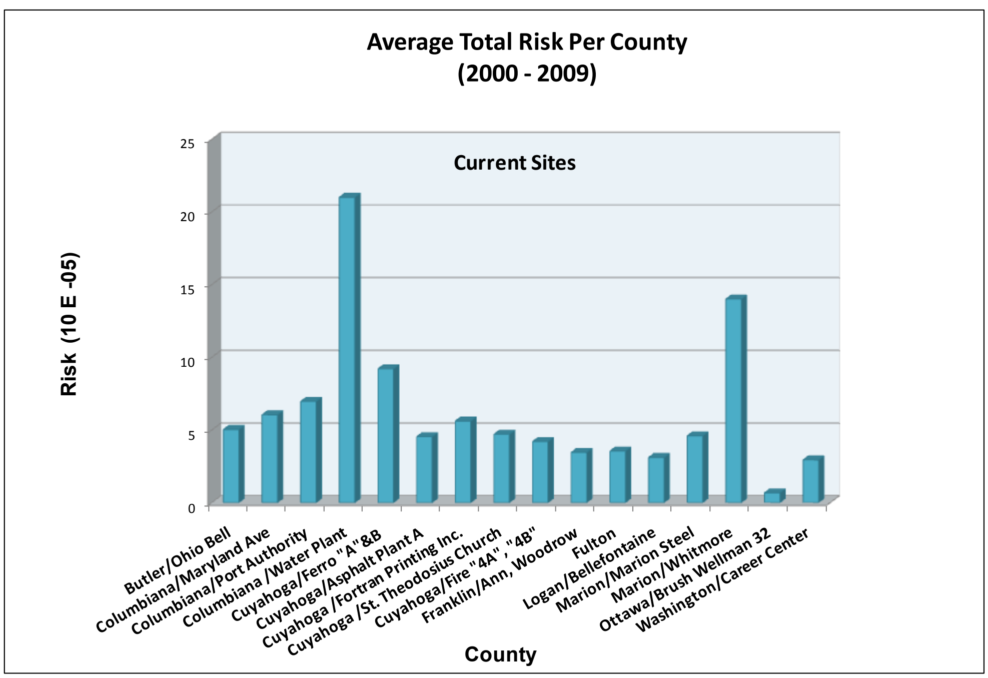


Figure 10. Non-Cancer Risk Estimates for Heavy Metal Compounds

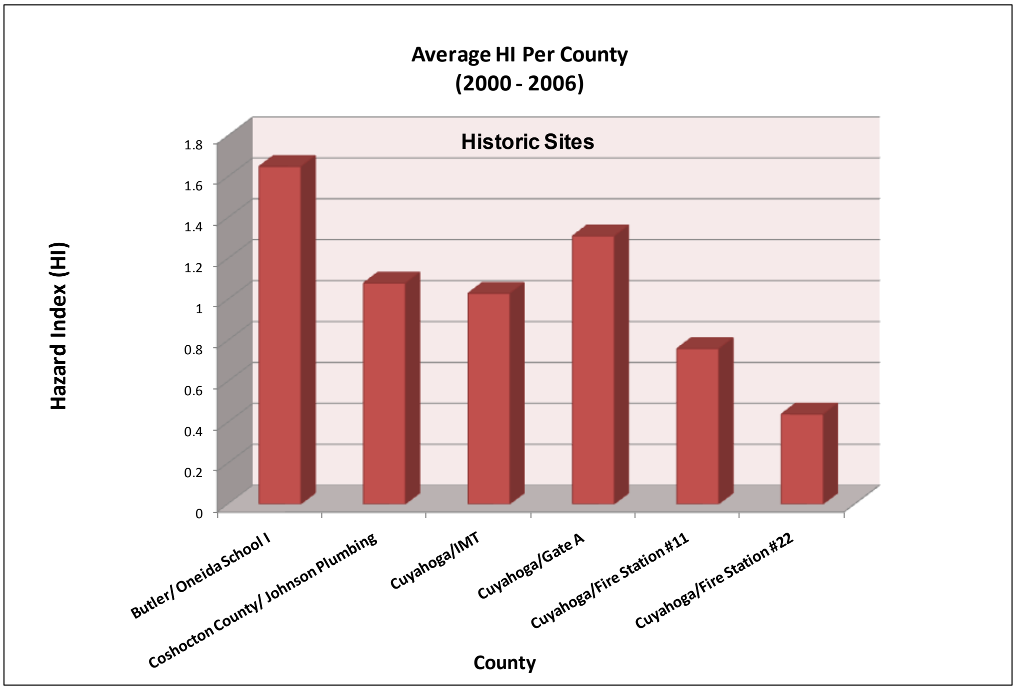
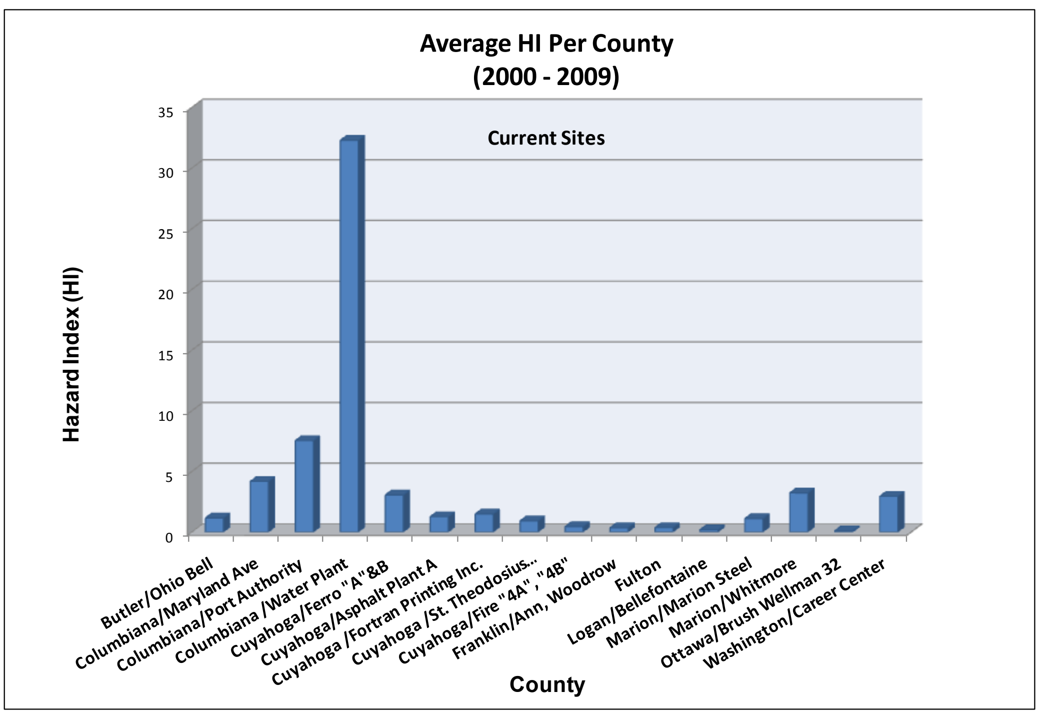


Figure 11. Non-Cancer Risk Estimates for Heavy Metal Compounds



RISK DRIVER SUMMARY

When observing a specific sampling site or geographical area, it frequently becomes apparent that a certain individual compound (or particular group of compounds) dominates risk by contributing the highest concentrations for a specific site or area. Since these compounds dominate or “drive” the risk in this case, they are termed “risk drivers.” When the average concentration of these chemicals is detected above risk-based screening levels at a particular site, the chemical is deemed a potential chemical of concern (COC) and warrants additional investigation. For almost every site, the data show that two compounds acrylonitrile and benzene drive the majority of the cancer risk for potential exposure to ambient air concentrations of VOCs. Chromium drives the cancer risk estimates for heavy metals. The non-cancer risk for VOCs is dominated by naphthalene; manganese drives the non-cancer estimates for heavy metals.

A specific breakdown of how the cancer and non-cancer risk drivers impact the results for each monitoring location is summarized below. The information is summarized by county. The counties vary slightly based upon of the years that the specific monitor(s) were operating.

Figure 12a and 12b compares the VOC risk drivers (acrylonitrile and benzene) concentrations for each county. Likewise, **Figure 13a and 13b** compares the non-cancer risk drivers (naphthalene and manganese) for each county. A brief summary of the potential health effects associated with chronic (long-term) exposure to the chemicals of concern is also described below.

Figure 12a. Ohio County Comparison
Cancer Risk Driver, Benzene

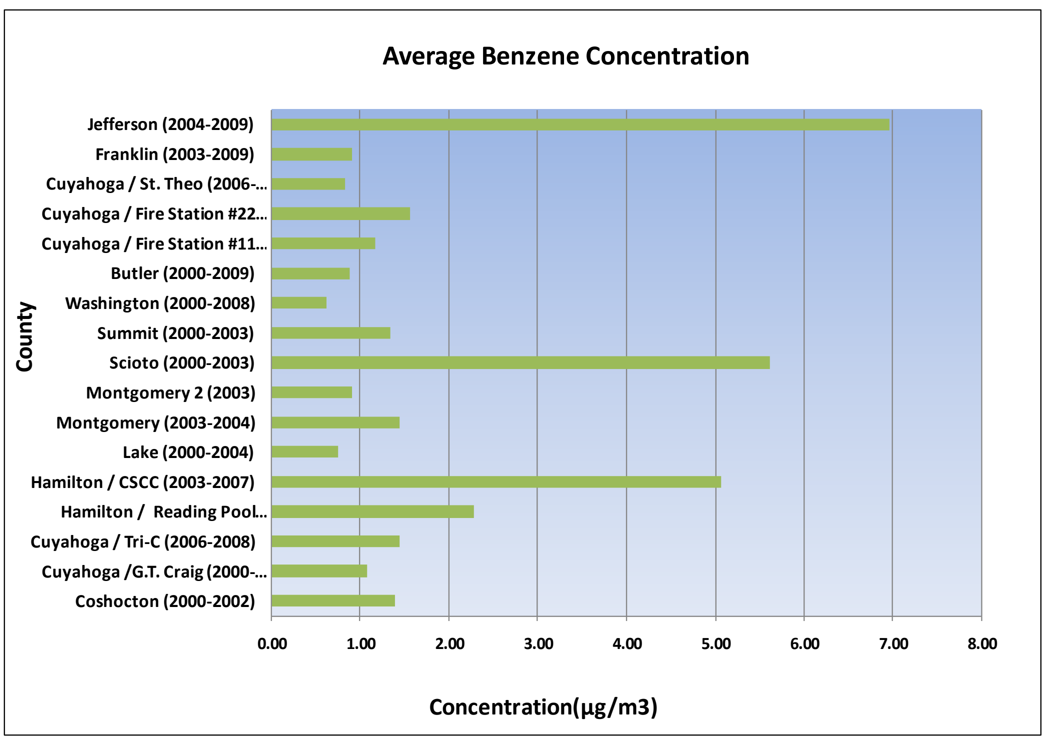


Figure 12b. Ohio County Comparison Cancer Risk Driver, Acrylonitrile

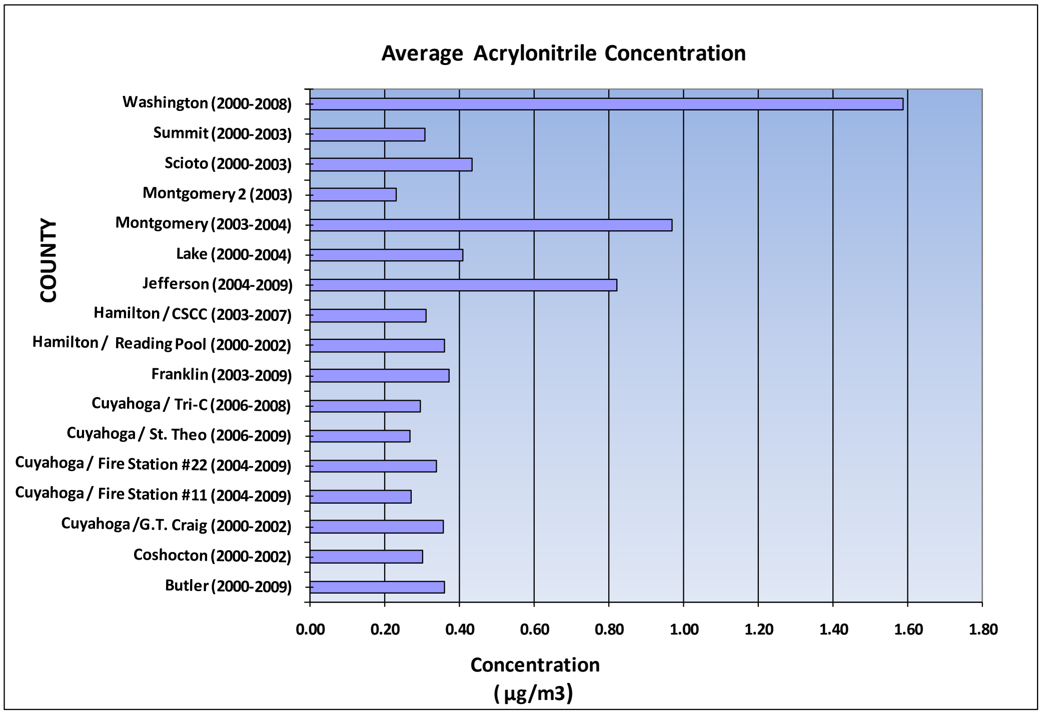


Figure 13a. Ohio County Comparison Non-Cancer Risk Driver, Naphthalene

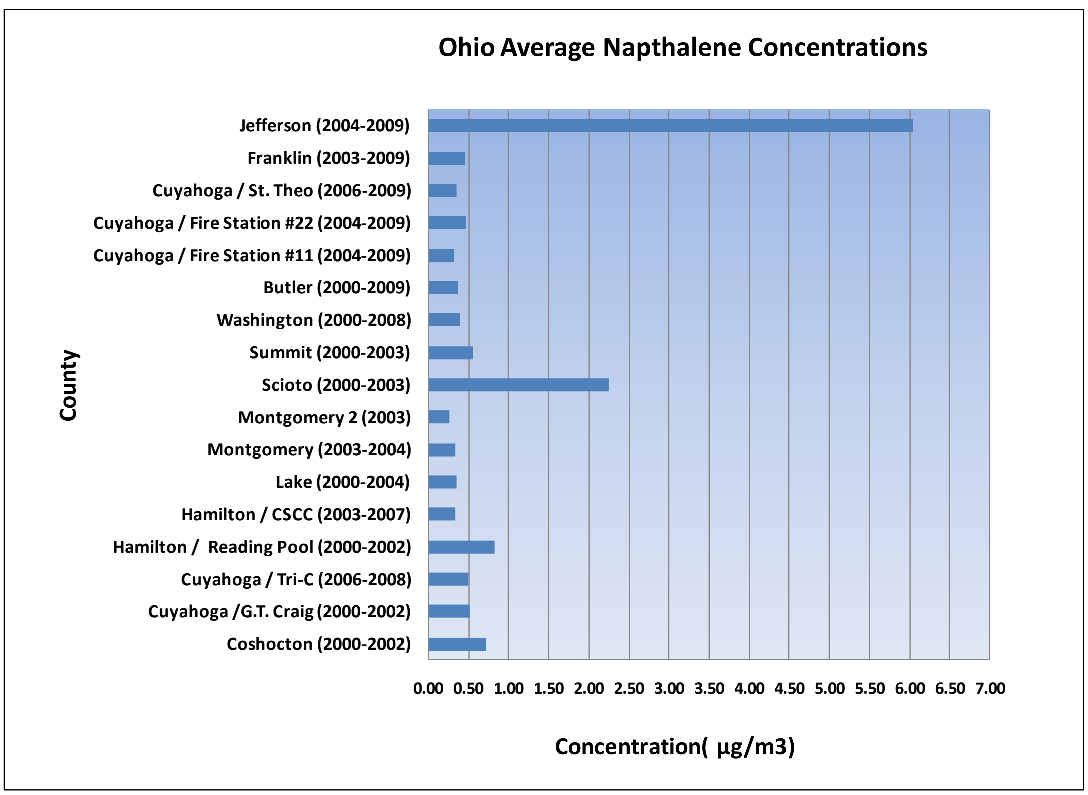
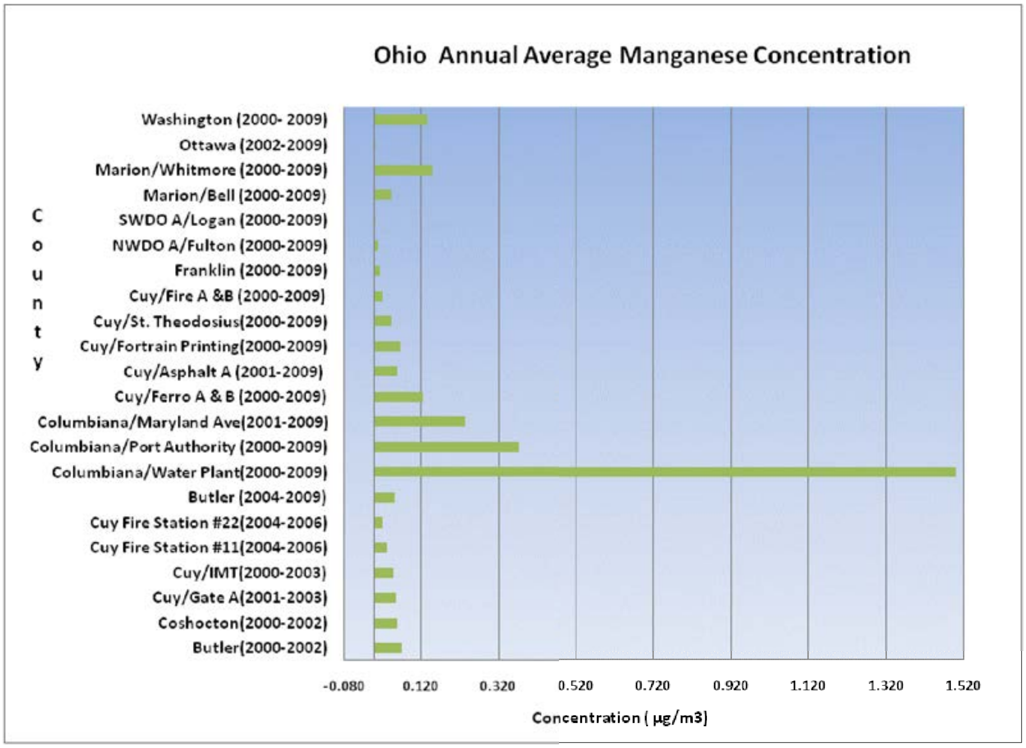


**Figure 13b. Ohio County Comparison/
Non-Cancer Risk Driver, Manganese**



TOXICITY FOR CHEMICALS OF CONCERN

The following health effects information is a brief summary of data collected from U.S. EPA's IRIS and Technology Transfer Network Air Toxics hazard summaries. The Agency for Toxics Substances and Disease Registry (ATSDR - part of the National Center for Disease Control [CDC]) also supplies information concerning the potential health effects caused by toxic air pollutants. Ohio EPA risk assessment procedures follow guidelines provided by the National Academy of Sciences (NAS) and U.S. EPA (*Risk Assessment Guidance for Superfund - Volume 1 - Human Health, December 1989*) and the recently revised U.S. EPA Air Toxics Risk Assessment Reference Library (Volumes 1-3, April 2004).

ACRYLONITRILE

Acrylonitrile is classified as a hazardous air pollutant (HAP) under Title III of the Clean Air Act Amendments. Acute (short-term) exposure to high concentrations of acrylonitrile through inhalation can lead to nose and throat irritation, breathing difficulties, nausea, dizziness, weakness, headaches, and convulsions. Symptoms typically disappear when acute exposure is stopped. Chronic (long-term) non-cancer health effects also include headaches, fatigue and nausea. The primary long-term carcinogenic health effect associated with human exposure to acrylonitrile is lung cancer.

BENZENE

Breathing very high levels of benzene can result in death, while high levels can cause drowsiness, dizziness, rapid heart rate, headaches, tremors, confusion, and unconsciousness. Eating or drinking foods containing high levels of benzene can cause vomiting, irritation of the stomach, dizziness, sleepiness, convulsions, rapid heart rate, and death.

The major effect of benzene from long-term exposure is on the blood. Benzene causes harmful effects on the bone marrow and can cause a decrease in red blood cells leading to anemia. It can also cause excessive bleeding and can affect the immune system, increasing the chance for infection.

Long-term exposure to high levels of benzene in the air can cause leukemia, particularly acute myelogenous leukemia, often referred to as AML. This is a cancer of the blood-forming organs. The Department of Health and Human Services (DHHS) has determined that benzene is a known carcinogen. The International Agency for Research on Cancer (IARC) and U.S. EPA have determined that benzene is carcinogenic to humans.

NAPHTHALENE

Exposure to large amounts of naphthalene may damage or destroy some red blood cells. This could cause a person to have too few red blood cells until the body replaces the destroyed cells. This condition is called hemolytic anemia. Some symptoms of hemolytic anemia are fatigue, lack of appetite, restlessness, and pale skin. Exposure to large amounts of naphthalene may also cause nausea, vomiting, diarrhea, blood in the urine, and a yellow color to the skin. Animals sometimes develop cloudiness in their eyes after swallowing high amounts of naphthalene. It is not clear whether this also develops in people. Rats and mice that breathed naphthalene vapors daily for a lifetime developed irritation and inflammation of their nose and lungs. It is unclear if naphthalene causes reproductive effects in animals; most evidence says it does not.

MANGANESE

Chronic (long-term) exposure to high levels of manganese by inhalation in humans may result in central nervous system (CNS) effects. Visual reaction time, hand steadiness, and eye-hand coordination were affected in chronically exposed workers. A syndrome named “manganism” may result from chronic exposure to higher levels. Manganism is characterized by feelings of weakness and lethargy, tremors, a mask-like face, and psychological disturbances. Respiratory effects have also been noted in workers chronically exposed by inhalation.

CHROMIUM

Chromium exists in multiple valence states. Of all possible valences, trivalent (+3) (chromium III), and hexavalent (+6) (chromium VI) are the most relevant in the environmental setting. Natural ores of chromium +3 are the most common natural form, while most sources of chromium VI are man-made and often associated with electroplating and metal finishing, leather tanning, and textile production. It was also used as a corrosion inhibitor and can be a component of fly ash.

It is customary to treat all chromium data as chromium VI (due to its greater toxicity and carcinogenicity) when specific data is not available on the form of the chromium compound collected. This assumption, while protective, may greatly overestimate the potential risk.

Chronic inhalation of chromium VI results in irritation of the respiratory tract and can result in asthma or other signs of respiratory distress with measurable decrease of respiratory function. In addition to the non-cancer effects of chromium VI inhalation, a number of studies have linked inhalation of chromium VI to lung cancer in humans. Studies of the toxic effects of chronic ingestion of chromium VI are not conclusive, but acute effects following ingestion of moderate doses (0.56 milligram per kilogram per day [mg/kg-d]) are associated with oral ulceration, diarrhea, abdominal pain, indigestion, vomiting and blood system damage.

CONCLUSION

Ohio EPA has completed the data compilation and risk assessment summary included in this report to a) provide a summary of the air toxics monitoring activities conducted by the Division of Air Pollution Control; b) make this information available to the public and other governmental agencies; c) potentially prioritize air toxic monitoring sites for risk reduction; and d) document the reductions in airborne concentrations of these compounds in Ohio.

Many air toxic investigations have already resulted in substantial reduction or elimination of potentially high concentrations of chemicals of concern. For example, Ohio EPA recently completed investigations in Butler County and Summit County. Air toxics enforcement actions have been completed in Scioto County and Hamilton County. Ohio EPA is also collaborating with federal and local agencies regarding a variety of air toxics monitoring and reduction projects in Ohio.

Ohio EPA has generated several individual reports that document activities at several sites. The most recent of these individual air toxic reports are available on the Ohio EPA website (www.epa.ohio.gov/dapc/atu/atu.aspx). As additional information becomes available, Ohio EPA will continue to provide updated information to the public regarding air toxics activities being conducted in the State of Ohio.

For more information:

Risk Assessment Questions
Paul Koval or Diane McClure
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Ambient Monitoring & Laboratory Analysis Questions
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TOXICITY (HEALTH EFFECTS) RESOURCES

Ohio EPA relied on the resources identified below to obtain health effects information used for assessing both the cancer and non-cancer risk. The use of each source was prioritized to be consistent with the recommended U.S. EPA hierarchy.

U.S. EPA'S INTEGRATED RISK INFORMATION SYSTEM [IRIS]

U.S. EPA maintains the Integrated Risk Information System (IRIS) database. Health assessment information on a chemical substance is included in IRIS only after a comprehensive review process of chronic toxicity data by both U.S. EPA and external health scientists (www.epa.gov/IRIS/process.htm). Detailed review of the information for each compound is contained in a Toxicological Review section of the database.

The IRIS database contains information on human health effects that may result from exposure to various chemicals in the environment. IRIS was developed to provide consistent information on chemical substances for use in risk assessments, decision making and regulatory activities. The IRIS system is a collection of individual chemical computer files containing descriptive and quantitative information in the following categories: Oral reference doses and inhalation reference concentrations (RfDs and RfCs, respectively) for chronic non-carcinogenic health effects, and hazard identification, oral slope factors, and oral and inhalation unit risks for carcinogenic effects.

This information represents a consensus opinion of U.S. EPA health scientists interpreting the scientific literature applicable to health effects of a chemical, and using established methodologies to develop values for oral reference dose, inhalation reference concentration, carcinogenic slope factor and unit risk. The products of this work, summarized in IRIS and elaborated in chemical-specific Toxicological Profile documents, have been subject to U.S. EPA's peer review policy. Also included in the IRIS information is a Weight of Evidence determination for the classification of a compound as a carcinogen. As new scientific information becomes available, U.S. EPA will review it, as appropriate, may revise IRIS files accordingly. The IRIS database is available online at http://www.epa.gov/iris.

AGENCY FOR TOXIC SUBSTANCES AND DISEASE REGISTRY [ATSDR]

When IRIS RfC values were not available, ATSDR's inhalation minimum risk levels (MRLs) were used for the non-cancer assessments. Toxicity assessments are performed by ATSDR, which is a branch of the Public Health Service, U.S. Department of Health and Human Services. A Toxicological Profile is prepared by the Division of Toxicology according to ATSDR and U.S. EPA guidelines. The Profile succinctly characterizes the toxicologic and adverse health effects information for the toxic compound. The peer-reviewed profile lists MRLs that are derived when reliable and sufficient data exist to identify the most sensitive health effects from exposure to the compound.

An MRL is an estimate of the daily exposure to a hazardous substance that is likely to be without appreciable risk of adverse non-cancer health effects over a specific duration of exposure. MRLs are derived for acute (1-14 days), intermediate (15-364 days) and chronic (365 days and longer) exposure durations. As such, the MRLs define the depth of toxicological information available for a compound declared toxic by the Profile. MRLs are published in pollutant-specific toxicological profile documents, and also in a table of "comparison values" that ATSDR regularly updates and distributes (available online at http://www.atsdr.cdc.gov/mrls.html).

CALIFORNIA OFFICE OF ENVIRONMENTAL HEALTH HAZARD ASSESSMENT (OEHHA)

The California OEHHA has developed dose-response assessments for many substances, based both on carcinogenicity and health effects other than cancer. The process for developing these assessments is similar to that used by U.S. EPA to develop IRIS values and incorporates significant external scientific peer review. The non-cancer information includes available inhalation health risk guidance values expressed as chronic inhalation reference exposure levels (RELs). OEHHA defines the REL as a concentration level at (or below) which no health effects are anticipated, a concept that is substantially similar to U.S. EPA's non-cancer dose-response assessment perspective. This assessment uses chronic RELs in the same way as RfCs when no IRIS or ATSDR values exist.

OEHHA's quantitative dose-response information on carcinogenicity by inhalation exposure is expressed in terms of the URE, defined similarly to U.S. EPA's URE. This assessment uses specific OEHHA UREs in the same way as U.S. EPA's when no IRIS or values exist. OEHHA's dose response information for carcinogens and non-carcinogens is available online at http://www.oehha.ca.gov/air/hot_spots/index.html.

U.S. EPA HEALTH EFFECTS ASSESSMENT TABLES (HEAST)

HEAST is a comprehensive listing consisting almost entirely of provisional UREs, RfCs, and other risk assessment information for chemicals of interest. Although the assessments summarized in HEAST have undergone review and have the concurrence of individual U.S. EPA program offices, and each is supported by an agency reference, they have not had enough review to be recognized as high-quality, EPA-wide consensus information. Because of these limitations, and the fact that HEAST has not been updated since 1997 and exists only in hard copy (PB97-921199), this assessment uses HEAST information only when no values from the other sources described above are available.

APPENDIX

For a detailed summary of how individual compounds contribute to the overall cancer and non-cancer risk estimates, refer to **Appendices A1** and **A2**. **Appendices A1** and **A2** contains individual summaries of the risk results for the VOC and heavy metal air monitoring sites listed in **Figures 2-5**. **Tables A1-1** to **A1-16** contains the results for the individual VOC monitoring sites. Results for the heavy metal monitoring sites are located in **Tables A 2-1** to **A 2-22**. Listed in each table is the annual average for the compound identified, with the exception of the designation of “ND” which indicates an entire year of non-detected air measurements for the parameter listed. Also listed is the risk estimation information for both the non- cancer (Hazard Index) and excess cancer risk for the site. Risk calculations are only included for measurements exceeding the lower detection limit of the analysis method. As stated previously, some counties contain more than one ambient air monitoring station.

Appendices B1 and B2 contains the yearly average concentration for air toxic compounds at each monitoring site. Yearly averages for individual VOC monitoring sites are listed in **Tables B1-1** to **B1-22.** The yearly average results for the heavy metal monitoring sites are included in **Tables B2-1** to **B2-22**. Each table lists the annual average for the compound identified, with the exception of the designation of “ND” which indicates an entire year of non-detects.

Appendix A1, Average Total VOC Site Risk Per County

A1-1. Butler County (2000-2009)

Source Related VOC Sampling Middletown					**Total Risk**	**2000-2009**		
Location:	HAMILTON							
	Butler County							
	Verity School							
	1900 St. John's Road							
	Middletown							
AIRS#:	39-017-0003							
Compounds	**Average (µg/m3) with half detections**	**RBC**	**Hazard Index**	**UR**	**RISK**	**Frequency**	**Minimum value detected (µg/m3)**	**Maximum value detected (µg/m3)**
acetone	9.28	31000	0.00			260	1.64	42.76
acetonitrile	0.38	60	0.01			96	0.34	1.49
acrylonitrile	0.39	2	0.19	6.80E-05	2.53E-05	36	0.43	4.12
benzene	0.88	30	0.03	7.80E-06	6.88E-06	244	0.32	3.51
1,3-butadiene	0.19	2	0.09	3.00E-05	5.62E-06	24	0.22	0.75
n-butane	2.85					276	0.24	13.55
2-butanone	1.50	5000	0.00			155	0.15	7.96
carbon disulfide	0.98	700	0.00			23	1.56	8.10
carbon tetrachloride	0.51	40	0.01	1.50E-05	7.68E-06	93	0.63	3.40
chlorodifluoromethane	1.16	50000	0.00			249	0.18	6.01
chloroethane	0.17	10000	0.00			1	0.58	0.58
chloroform	0.30	98	0.00	2.30E-05	7.05E-06	3	0.54	1.17
chloromethane	1.07	90	0.01	1.80E-06	1.92E-06	273	0.52	1.84
3-chloropropene	0.18					1	3.07	3.07
cyclohexane	0.20	6000	0.00			5	0.45	1.17
decane	0.41					9	0.58	11.06
dibromochloromethane	0.53					1	6.13	6.13
1,4-dichlorobenzene (para)	0.45	800	0.00	1.00E-05	4.49E-06	9	0.66	1.08
dichlorodifluoromethane	2.68					278	0.00	8.32
trans-1,2-dichloroethene	0.30					1	1.03	7.66
n-dodecane	0.41					1	7.66	1.03
ethanol	1.09					13	0.90	130.01
ethylbenzene	0.32	1000	0.00			22	0.43	1.30
4-ethyltoluene	0.32					1	0.54	0.54
n-heptane	0.34					16	0.41	4.10
hexane	0.60	700	0.00			63	0.42	4.58
methylene chloride	0.33	1000	0.00	4.70E-07	1.57E-07	64	0.35	1.81
4-methyl-2-pentanone	0.29					2	0.48	0.63
a-methylstyrene	0.23					5	0.41	1.19
naphthalene	0.43	3	0.14			9	0.52	2.83
n-nonane	0.46					4	0.58	9.44
n-octane	0.28					1	0.56	0.56
n-pentane	1.38					269	0.35	12.39
propylene	0.78					110	0.09	5.34
toluene	1.23	5000	0.00			225	0.00	18.84
1,1,1-trichloroethane	0.80					130	0.55	3.22
trichloroethene	0.57	60	0.01	2.00E-06	1.13E-06	1	1.88	1.88
trichlorofluoromethane	12.49					277	0.96	230.37
1,1,2-trichloro-1,2,2-trifluoroethar	0.54					30	0.77	1.23
1,2,4-trimethylbenzene	0.43					39	0.49	2.75
1,3,5-trimethylbenzene	0.28					4	0.49	0.74
n-undecane	0.51					6	0.64	11.51
vinyl acetate	1.04	200	0.01			104	0.35	9.86
o-xylene	0.35	100	0.00			28	0.43	1.17
total m+p-xylene	0.72	100	0.01			47	0.43	3.95

A1-2. Coshocton County (2000-2003)

Source Related VOC Sampling Coshocton					Total Risk 2000-2003			
Location:	SEDO							
	Coshocton Co.							
	Coshocton Co. Health Dept.							
	724 South 7th Street							
	Coshocton							
AIRS # :	NA							
Compounds	**Average (µg/m3) with half detections**	**RBC**	**Hazard Index**	**UR**	**RISK**	**Frequency**	**Minimum value detected (µg/m3**	**Maximum value detected (µg/m3)**
acetone	6.55	31000	0.00			56	2.38	19.95
acetonitrile	0.19	60	0.00			5	0.00	0.81
acrylonitrile	0.27	2	0.14	6.80E-05	1.86E-05	8	0.00	1.48
benzene	1.39	30	0.05	7.80E-06	1.08E-05	67	0.32	10.54
1,3-butadiene	0.23	2	0.12	3.00E-05	7.00E-06	20	0.22	1.44
n-butane	8.35					70	1.69	33.28
2-butanone	0.69	5000	0.00			38	0.32	3.83
carbon disulfide	3.29	700	0.00			34	0.31	29.27
carbon tetrachloride	0.63	40	0.02	1.50E-05	9.45E-06	20	0.63	10.07
chlorodifluoromethane	1.03	50000	0.00			57	0.46	10.96
chloroethane	0.15	10000	0.00			4	0.00	1.11
chloromethane	1.18	90	0.01	1.80E-06	2.12E-06	70	0.50	2.48
cyclohexane	0.26	6000	0.00			7	0.38	1.65
decane	0.37					6	0.76	1.34
1,3-dichlorobenzene (meta)	0.32					1	0.00	1.68
1,4-dichlorobenzene (para)	0.33	800	0.00	1.00E-05	3.27E-06	4	0.00	1.38
dichlorodifluoromethane	2.94					70	1.29	4.90
1,1-dichloroethene	0.22	200	0.00	1.60E-06	3.52E-07	2	0.00	1.35
ethanol	0.59					6	0.00	5.65
ethylbenzene	0.43	1000	0.00			17	0.48	3.08
4-ethyltoluene	0.27					1	0.00	1.03
n-heptane	0.57					25	0.41	4.92
hexane	1.10	700	0.00			43	0.78	5.29
methylene chloride	0.57	1000	0.00	4.70E-07	2.66E-07	24	0.35	3.30
4-methyl-2-pentanone	0.22					1	0.00	0.86
a-methylstyrene	0.31					5	0.48	1.98
naphthalene	0.57	3	0.19			8	0.00	29.35
n-nonane	0.40					12	0.58	2.68
n-octane	0.51					12	0.47	4.02
n-pentane	3.23					70	0.80	12.98
propylene	1.09					7	0.43	15.49
styrene	0.23	1000	0.00	5.70E-07	1.31E-07	1	0.00	0.77
tetrachloroethylene	0.83			9.50E-07	7.89E-07	19	0.00	13.57
toluene	4.07	5000	0.00			69	0.49	35.04
1,2,4-trichlorobenzene	0.42					4	0.00	1.63
1,1,1-trichloroethane	3.84					63	0.55	27.83
trichlorofluoromethane	10.35					70	2.02	101.14
1,1,2-trichloro-1,2,2-trifluoroethane	0.43					3	0.00	1.00
1,2,4-trimethylbenzene	0.57					25	0.49	4.62
1,3,5-trimethylbenzene	0.29					2	0.00	1.33
n-undecane	0.34					2	0.00	0.96
vinyl acetate	0.36	200	0.00			3	0.00	0.81
o-xylene	0.46	100	0.00			24	0.43	2.47
total m+p-xylene	1.50	100	0.01			48	0.48	13.89

A1-3. Cuyahoga County, Fire Station 11 (2004-2009)

Source Related VOC Sampling Cleveland						Total Risk	2004-2009	
Location:	CLEVELAND							
	Cuyahoga Co.							
	Fire Station #11							
	7629 Broadway Ave.							
	Cleveland							
AIRS#:	39-035-0068							
Compounds	**Average (µg/m3) with half detections**	**RBC**	**Hazard Index**	**UR**	**RISK**	**Frequency**	**Minimum value detected (µg/m3)**	**Maximum value detected (µg/m3)**
acetone	11.15	31000	0.00			141	2.00	47.51
acetonitrile	0.57	60	0.01			80	0.34	3.19
acrylonitrile	0.23	2	0.11	6.80E-05	1.54E-05	1	0.00	1.24
benzene	1.17	30	0.04	7.80E-06	9.10E-06	123	0.38	7.03
bromodichloromethane	0.42					2	0.00	0.80
bromomethane	0.24	5	0.05			1	0.00	0.54
1,3-butadiene	0.17	2	0.09	3.00E-05	5.14E-06	17	0.00	1.00
n-butane	4.32					142	0.55	16.64
2-butanone	1.94	5000	0.00			90	1.47	12.98
carbon disulfide	0.79	700	0.00			2	0.00	1.78
carbon tetrachloride	0.56	40	0.01	1.50E-05	8.40E-06	60	0.00	1.38
chlorodifluoromethane	1.63	50000	0.00			129	0.53	34.66
chloroethane	0.17	10000	0.00			2	0.00	0.42
chloroform	0.32	98	0.00	2.30E-05	7.26E-06	3	0.00	1.17
chloromethane	1.11	90	0.01	1.80E-06	1.99E-06	142	0.54	2.00
3-chloropropene	0.21					5	0.00	0.81
cyclohexane	0.25	6000	0.00			10	0.00	1.79
decane	0.42					12	0.00	1.22
dibromomethane	0.45					2	0.00	0.78
1,4-dichlorobenzene (para)	0.41	800	0.00	1.00E-05	4.07E-06	5	0.00	2.47
dichlorodifluoromethane	2.90					143	1.63	6.92
1,1-dichloroethane	0.26					2	0.00	0.61
1,2-dichloroethane	0.26			2.60E-05	6.66E-06	2	0.00	0.57
1,1-dichloroethene	0.25	200	0.00	1.60E-06	4.03E-07	2	0.00	0.59
cis-1,2-dichloroethene	0.25					2	0.00	0.59
trans-1,2-dichloroethene	0.25					2	0.00	0.56
1,2-dichloropropane	0.29	4	0.07			1	0.00	0.51
1,2-dichloro-1,1,2,2-tetrafluoroethane	0.44					1	0.00	0.77
ethylbenzene	0.41	1000	0.00			32	0.00	1.69
4-ethyltoluene	0.32					6	0.00	0.93
n-heptane	0.48					44	0.00	3.28
hexachlorobutadiene	0.67			2.20E-05	1.47E-05	1	0.00	1.17
hexane	1.03	700	0.00			82	0.70	4.23
methylene chloride	1.62	1000	0.00	4.70E-07	7.62E-07	118	0.17	17.02
4-methyl-2-pentanone	0.32					19	0.00	2.13
naphthalene	0.37	3	0.12			12	0.00	1.57
n-nonane	0.36					9	0.00	1.10
n-octane	0.33					9	0.00	1.26
n-pentane	2.69					143	0.47	11.80
propylene	1.60					83	0.00	8.95
n-propyl benzene	0.31					1	0.00	0.84
styrene	0.27	1000	0.00	5.70E-07	1.55E-07	2	0.00	0.81
tetrachloroethylene	0.52			9.50E-07	4.91E-07	17	0.00	2.92
toluene	2.75	5000	0.00			134	0.38	15.83
1,1,1-trichloroethane	0.35					2	0.00	0.82
1,1,2-trichloroethane	0.34			1.60E-05	5.49E-06	1	0.00	0.55
trichloroethene	0.40	60	0.01	2.00E-06	7.93E-07	14	0.00	1.77
trichlorofluoromethane	2.01					143	0.67	8.99
1,1,2-trichloro-1,2,2-trifluoroethane	0.55					21	0.00	1.61
1,2,4-trimethylbenzene	0.55					41	0.00	2.95
1,3,5-trimethylbenzene	0.33					6	0.00	0.98
n-undecane	0.41					4	0.00	1.21
vinyl acetate	0.73	200	0.00			35	0.00	7.39
vinyl chloride	0.16	100	0.00	8.80E-06	1.41E-06	1	0.00	0.31
o-xylene	0.42	100	0.00			36	0.00	1.74
total m+p-xylene	1.17	100	0.01			52	0.43	6.51

A1-4. Cuyahoga County, Fire Station 22 (2004-2009)

Source Related VOC Sampling Cuyahoga					**Total Risk**	**2004-2009**		
Location:	CLEVELAND							
	Cuyahoga Co.							
	Fire Station #22							
	7629 Broadway Ave.							
	Cleveland							
AIRS#:	39-035-0068							

Compounds	Average (µg/m3) with half detections	RBC	Hazard Index	UR	RISK	Frequency	Minimum value detected (µg/m3)	Maximum value detected (µg/m3)
acetone	13.15	31000	0.00			138	2.61	54.64
acetonitrile	0.50	60	0.01			76	0.34	2.35
acrylonitrile	0.28	2	0.14	6.80E-05	1.89E-05	21	0.46	1.13
benzene	1.52	30	0.05	7.80E-06	1.18E-05	136	0.16	11.82
1,3-butadiene	0.19	2	0.10	3.00E-05	5.75E-06	29	0.11	1.35
n-butane	6.14					141	0.33	35.66
2-butanone	1.79	5000	0.00			77	0.15	6.49
carbon disulfide	0.81	700	0.00			2	1.87	2.02
carbon tetrachloride	0.55	40	0.01	1.50E-05	8.24E-06	53	0.63	1.20
chlorobenzene	0.30	1000	0.00			1	1.10	1.10
chlorodifluoromethane	1.51	50000	0.00			128	0.50	33.24
chloroethane	0.17	10000	0.00			1	0.29	0.29
chloroform	0.35	98	0.00	2.30E-05	7.98E-06	2	0.63	2.25
chloromethane	1.12	90	0.01	1.80E-06	2.01E-06	143	0.52	2.00
cyclohexane	0.26	6000	0.00			13	0.34	1.62
decane	0.44					10	0.58	2.56
1,4-dichlorobenzene (para)	0.41	800	0.00	1.00E-05	4.10E-06	7	0.60	1.32
dichlorodifluoromethane	2.74					143	1.29	4.25
1,1-dichloroethane	0.26					1	0.49	0.49
1,2-dichloroethane	0.26			2.60E-05	6.67E-06	1	0.40	0.40
1,1-dichloroethene	0.26	200	0.00	1.60E-06	4.09E-07	1	0.56	0.56
cis-1,2-dichloroethene	0.26					1	0.63	0.63
trans-1,2-dichloroethene	0.26					1	0.56	0.56
1,2-dichloro-1,1,2,2-tetrafluoroethane	0.45					1	0.70	0.70
ethylbenzene	0.43	1000	0.00			35	0.43	2.13
4-ethyltoluene	0.34					5	0.79	1.03
n-heptane	0.50					48	0.41	2.79
hexane	1.48	700	0.00			92	0.70	16.21
methylene chloride	0.68	1000	0.00	4.70E-07	3.19E-07	68	0.35	25.36
4-methyl-2-pentanone	0.64					23	0.41	9.83
naphthalene	0.41	3	0.14			11	0.52	3.09
n-nonane	0.36					6	0.58	0.94
n-octane	0.32					6	0.56	0.79
n-pentane	3.59					141	0.68	29.51
propylene	1.50					82	0.09	8.26
n-propyl benzene	0.32					3	0.49	0.84
tetrachloroethylene	0.52			9.50E-07	4.92E-07	16	0.68	2.71
toluene	3.38	5000	0.00			136	0.38	32.03
1,1,1-trichloroethane	0.35					2	0.55	0.65
trichloroethene	0.39	60	0.01	2.00E-06	7.88E-07	12	0.54	2.26
trichlorofluoromethane	1.68					139	0.96	5.39
1,1,2-trichloro-1,2,2-trifluoroethane	0.54					15	0.77	1.38
1,2,4-trimethylbenzene	0.58					33	0.49	3.74
1,3,5-trimethylbenzene	0.35					8	0.64	1.13
n-undecane	0.45					4	0.70	4.41
vinyl acetate	0.73	200	0.00			23	0.74	3.87
o-xylene	0.47	100	0.00			36	0.00	2.61
total m+p-xylene	1.36	100	0.01			52	0.87	10.42

A1-5. Cuyahoga County, G.T. Craig (2000-2003)

Risk Calculations
Air Toxics VOC Sampling Cleveland Area **2000-2003**

	CLEVELAND
Location:	Cuyahoga Co.
	G.T. Craig
	E. 14th & Orange
	Cleveland
AIRS#:	39-035-0060
Sampler:	Xon. 911-J23460

Compounds	Average (µg/m3)	RBC	HI	UR	RISK	Frequency	Minimum value detected (µg/m3)	Maximum value detected (µg/m3)
acetone	9.19	31000	0.00			42	2.23	23.75
acetonitrile	0.73	60	0.01			11	0.34	4.20
acrylonitrile	0.59	2	0.30	6.80E-05	4.02128E-05	10	0.46	1.04
benzene	1.13	30	0.04	7.80E-06	8.81373E-06	73	0.32	3.51
bromomethane	0.39					1	0.78	0.78
1,3-butadiene	0.36	2	0.18	3.00E-05	1.07139E-05	21	0.22	0.75
n-butane	10.77					73	0.29	59.43
2-butanone	2.09	5000	0.00			44	0.44	5.90
carbon disulfide	2.76	700	0.00			19	1.59	8.08
carbon tetrachloride	0.70	40	0.02	1.50E-05	1.04856E-05	23	0.63	1.13
chlorodifluoromethane	0.92	50000	0.00			56	0.35	3.89
chloroethane / ethyl chloride	0.32	10000	0.00			2	0.37	0.40
chloromethane / methyl chloride	1.12	90	0.01	1.80E-06	2.02067E-06	73	0.45	1.86
cyclohexane	0.40	6000	0.00			4	0.34	0.96
decane	0.62					3	0.76	1.11
1,4-dichlorobenzene (para)	0.90	800	0.00	1.00E-05	9.01892E-06	2	0.78	1.92
dichlorodifluoromethane	2.78					73	1.72	3.91
ethanol	1.79					2	3.39	6.41
ethylbenzene	0.88	1000	0.00			19	0.43	2.21
n-heptane	0.85					18	0.41	1.60
hexane	1.52	700	0.00			45	0.74	3.48
methyl-butyl ether	0.73					1	1.84	1.84
methylene chloride / dichloromethane	1.00	1000	0.00	4.70E-07	4.69215E-07	42	0.35	4.17
4-methyl-2-pentanone	1.03					6	0.45	2.62
napthalene	0.61	3	0.20			5	0.58	0.94
n-nonane	0.62					4	0.58	0.94
n-octane	0.44					3	0.51	0.84
n-pentane	3.99					72	0.59	12.39
propylene	1.90					12	0.77	5.51
styrene	0.29	1000	0.00	5.70E-07	1.63971E-07	1	0.51	0.51
tetrachloroethylene	0.82			9.50E-07	7.75903E-07	6	0.75	1.15
toluene	2.56	5000	0.00			72	0.45	8.67
1,1,1-trichloroethane	0.73					30	0.55	1.53
trichloroethene	0.58	60	0.01	2.00E-06	1.15E-06	4	0.59	1.13
trichlorofluoromethane	1.66					68	1.12	2.98
1,1,2-trichloro-1,2,2-trifluoroethane	0.63					9	0.84	1.46
1,2,4-trimethylbenzene	0.86					21	0.49	1.62
1,3,5-trimethylbenzene	0.32					1	0.54	0.54
n-undecane	0.42					1	0.70	0.70
vinyl acetate	1.02	200	0.01			8	0.70	2.64
o-xylene	0.86	100	0.01			24	0.43	2.30
total m+p-xylene	2.35	100	0.02			42	0.56	10.42

A1-6. Cuyahoga County, St. Theodosious (2006-2009)

Source Related VOC Sampling Cleveland					**Total Risk**	**2006-2009**		
Location:	Cuyahoga Co.							
	St. Theodosious							
	2547 St. Tikon							
	Cleveland							
AIRS#:	39-035-0038							
Compounds	**Average (µg/m3) with half detections**	**RBC**	**Hazard Index**	**UR**	**RISK**	**Frequency**	**Minimum value detected (µg/m3)**	**Maximum value detected (µg/m3)**
acetone	14.50	31000	0.00			69	1.95	114.02
acetonitrile	0.42	60	0.01			42	0.35	2.01
acrylonitrile	0.26	2	0.13	6.80E-05	0.00001734	12	0.43	0.61
benzene	0.82	30	0.03	7.80E-06	6.43061E-06	62	0.35	3.83
1,3-butadiene	0.16	2	0.08	3.00E-05	0.000004875	4	0.31	0.62
n-butane	4.96					85	0.43	23.53
2-butanone	1.52	5000	0.00			46	1.47	7.67
carbon tetrachloride	0.52	40	0.01	1.50E-05	7.73347E-06	18	0.63	1.01
chlorodifluoromethane	1.06	50000	0.00			63	0.60	3.50
chloromethane	1.23	90	0.01	1.80E-06	2.20823E-06	82	0.62	2.68
cumene	0.34	400	0.00			1	1.03	1.03
decane	6.74					38	0.58	51.21
1,4-dichlorobenzene (para)	0.46	800	0.00	1.00E-05	0.0000046	2	3.07	3.07
dichlorodifluoromethane	2.71					74	1.29	4.90
n-dodecane	0.38					13	1.81	1.81
ethylbenzene	0.42	1000	0.00			12	0.52	3.78
4-ethyltoluene	0.50					12	1.08	2.85
n-heptane	0.98					28	0.41	16.80
hexane	1.04	700	0.00			47	0.70	3.88
methylene chloride	0.80	1000	0.00	4.70E-07	3.77346E-07	46	0.35	7.99
4-methyl-2-pentanone	0.47					15	0.53	2.95
naphthalene	0.37	3	0.12			3	0.52	1.10
n-nonane	5.89					26	0.58	44.59
n-octane	0.65					13	0.98	4.53
n-pentane	2.71					70	0.47	10.03
n-propylbenzene	0.52					13	1.03	3.39
propylene	0.94					42	0.52	5.51
toluene	2.63	5000	0.00			78	0.45	15.07
1,1,1-trichloroethane	0.37					2	0.60	0.65
trichloroethene	0.39	60	0.01	2.00E-06	7.79E-07	4	0.70	1.29
trichlorofluoromethane	3.84					82	0.56	78.66
1,1,2-trichloro-1,2,2-trifluoroethane	0.57					20	0.77	1.00
1,2,4-trimethylbenzene	1.67					29	0.54	13.27
1,3,5-trimethylbenzene	0.64					21	0.69	4.28
n-undecane	2.62					23	1.15	17.26
vinyl acetate	0.93	200	0.00			29	0.81	5.28
o-xylene	0.61	100	0.01			26	0.48	3.73
total m+p-xylene	1.56	100	0.02			32	0.87	14.33

A 1-7. Cuyahoga County, Tri-C College (2006-2008)

Source Related VOC Sampling Cleveland				**Total Risk**	**2006-2008**			
Location:	CLEVELAND							
	Cuyahoga Co.							
	Tri-C College							
	2900 Community CollegeAve.							
	Cleveland							
AIRS#:	39-035-0071							

Compounds	Average (µg/m3) with half detections	RBC	Hazard Index	UR	RISK	Frequency	Minimum value detected (µg/m3)	Maximum value detected (µg/m3)
acetone	87.68	31000	0.00			56	4.04	807.66
acetonitrile	0.58	60	0.01			34	0.35	2.18
acrylonitrile	0.25	2	0.12	6.80E-05	1.67E-05	7	0.46	0.74
benzene	1.44	30	0.05	7.80E-06	1.12E-05	55	0.42	5.75
1,3-butadiene	0.28	2	0.14	3.00E-05	8.38E-06	17	0.22	1.77
n-butane	8.38					54	0.69	64.18
2-butanone	8.63	5000	0.00			50	0.15	82.57
carbon disulfide	0.62	700	0.00			3	1.56	3.11
carbon tetrachloride	0.54	40	0.01	1.50E-05	8.08E-06	15	0.63	1.07
chlorodifluoromethane	1.75	50000	0.00			39	0.78	8.49
chloroethane	0.17	10000	0.00			1	0.34	0.34
chloromethane	1.33	90	0.01	1.80E-06	2.39E-06	56	0.76	2.27
cyclohexane	0.43	6000	0.00			18	0.34	3.34
decane	1.02					22	0.76	12.80
1,4-dichlorobenzene (para)	1.48	800	0.00	1.00E-05	1.48E-05	22	0.66	10.82
dichlorodifluoromethane	3.27					56	1.19	34.62
1,2-dichloroethane	0.30			2.60E-05	7.89E-06	4	1.05	1.94
ethylbenzene	0.63	1000	0.00			15	0.48	14.76
4-ethyltoluene	0.32					2	0.74	1.13
n-heptane	0.79					33	0.45	3.98
hexane	1.39	700	0.00			41	0.70	7.05
methyl-butyl ether	0.31					1	0.94	0.94
methylene chloride	0.66	1000	0.00	4.70E-07	3.10E-07	36	0.35	2.81
4-methyl-2-pentanone	1.54					28	0.49	9.83
a-methylstyrene	0.30					1	0.68	0.68
naphthalene	0.51	3	0.17			9	0.63	3.72
n-nonane	0.40					6	0.63	1.84
n-octane	0.35					6	0.51	1.07
n-pentane	7.95					56	1.30	70.82
propylene	3.97					38	1.03	24.09
n-propyl benzene	0.32					1	1.38	1.38
styrene	0.28	1000	0.00	5.70E-07	1.58E-07	1	1.75	1.75
tetrachloroethylene	0.48			9.50E-07	4.57E-07	5	0.34	2.65
toluene	4.68	5000	0.00			56	0.49	20.72
trichloroethene	0.60	60	0.01	2.00E-06	1.20E-06	12	0.64	11.29
trichlorofluoromethane	1.90					55	0.62	3.82
1,1,2-trichloro-1,2,2-trifluoroethane	0.64					10	0.77	3.68
1,2,4-trimethylbenzene	0.67					16	0.74	2.70
1,3,5-trimethylbenzene	0.32					2	0.69	0.79
n-undecane	1.08					18	0.64	26.85
vinyl acetate	1.60	200	0.01			26	0.88	13.73
o-xylene	0.62	100	0.01			16	0.43	12.16
total m+p-xylene	1.95	100	0.02			23	0.87	47.76

A1-8. Franklin County, Fairgrounds (2003-2009)

Source Related VOC Sampling Franklin					Total Risk	2003-2009		
	Ohio EPA Central District Office							
Location:	Franklin County							
	Ohio State Fairgrounds							
	Korbel Ave.							
	Columbus							
	39-049-0034							
AQS # :	RM ESI-EPA017587							
Compounds	**Average (µg/m3) with half detections**	**RBC**	**Hazard Index**	**UR**	**RISK**	**Frequency**	**Minimum value detected (µg/m3)**	**Maximum value detected (µg/m3)**
acetone	13.59	31000	0.00			207	2.85	87.89
acetonitrile	0.42	60	0.01			111	0.34	3.02
acrylonitrile	0.37	2	0.19	6.80E-05	2.53E-05	38	0.43	3.91
benzene	0.90	30	0.03	7.80E-06	7.01E-06	178	0.32	4.15
bromodichloromethane	0.42					1	0.67	0.67
1,3-butadiene	0.17	2	0.08	3.00E-05	5.09E-06	18	0.24	1.53
n-butane	3.25					211	0.12	19.25
2-butanone	2.46	5000	0.00			135	0.00	14.45
carbon disulfide	0.83	700	0.00			5	2.86	4.98
carbon tetrachloride	0.49	40	0.01	1.50E-05	7.34E-06	58	0.63	1.26
chlorodifluoromethane	1.23	50000	0.00			195	0.50	4.24
chloroform	0.30	98	0.00	2.30E-05	6.90E-06	1	0.59	0.59
chloromethane	1.07	90	0.01	1.80E-06	1.92E-06	213	0.50	2.07
cyclohexane	0.22	6000	0.00			3	0.38	0.59
decane	0.39					2	0.87	6.40
1,4-dichlorobenzene (para)	0.40	800	0.00	1.00E-05	3.98E-06	7	0.66	2.65
dichlorodifluoromethane	2.62					210	1.38	3.71
cis-1,2-dichloroethene	0.23					1	0.40	0.40
n-dodecane	0.36					1	2.44	2.44
ethylbenzene	0.35	1000	0.00			32	0.43	1.65
4-ethyltoluene	0.31					3	0.59	0.64
n-heptane	0.30					24	0.41	1.11
hexachlorobutadiene	0.66			2.20E-05	1.45E-05	1	1.39	1.39
hexane	0.67	700	0.00			70	0.70	2.93
methylene chloride	0.32	1000	0.00	4.70E-07	1.51E-07	58	0.35	1.49
4-methyl-2-pentanone	0.27					11	0.41	0.74
naphthalene	0.38	3	0.13			18	0.52	2.25
n-nonane	0.33					2	0.68	1.15
n-octane	0.29					2	0.47	0.61
n-pentane	1.75					209	0.41	8.56
propylene	1.04					105	0.43	9.29
styrene	0.26	1000	0.00	5.70E-07	1.51E-07	3	0.47	0.81
tetrachloroethylene	0.42			9.50E-07	4.00E-07	2	0.75	0.95
toluene	1.84	5000	0.00			176	0.19	12.81
1,1,1-trichloroethane	0.34					2	0.93	0.93
trichloroethene	0.33	60	0.01	2.00E-06	6.65E-07	1	0.54	0.54
trichlorofluoromethane	1.47					205	1.01	2.47
1,1,2-trichloro-1,2,2-trifluoroethane	0.50					13	0.77	1.00
1,2,4-trimethylbenzene	0.43					33	0.49	2.61
1,3,5-trimethylbenzene	0.31					2	0.64	0.64
n-undecane	0.42					2	0.70	6.07
vinyl acetate	1.18	200	0.01			93	0.70	14.44
o-xylene	0.37	100	0.00			35	0.43	1.91
total m+p-xylene	0.90	100	0.01			52	0.87	7.82

A1-9. Hamilton County, Reading Pool (2000-2002)

Urban Air Monitoring: VOC Sampling				**Total Risk**	**2000-2002**			
Annual Average Concentrations								
Location:	HAMILTON							
	Hamilton County							
	Reading Pool							
	1601 West St.							
	Reading, Cincinnati							
AIRS #:								
Sampler:								
Concentrations:		µg/m3						

Compounds	Average (µg/m3) with half detections	RBC	Hazard Index	UR	RISK	Frequency	Minimum value detected (µg/m3)	Maximum value detected (µg/m3)
acetone	7.76	31000	0.00			39	3.56	30.88
acetonitrile	0.26	60	0.00			8	0.42	1.16
acrylonitrile	0.31	2	0.16	6.80E-05	2.13E-05	3	0.46	1.09
benzene	2.27	30	0.08	7.80E-06	1.77E-05	49	0.45	12.14
1,3-butadiene	0.30	2	0.15	3.00E-05	8.90E-06	17	0.24	1.61
n-butane	18.79					50	1.40	309.02
2-butanone	1.57	5000	0.00			40	0.59	6.49
carbon disulfide	1.45	700	0.00			11	1.71	8.10
carbon tetrachloride	0.51	40	0.01	1.50E-05	7.65E-06	15	0.63	1.07
chlorodifluoromethane	0.87	50000	0.00			37	0.46	2.62
chloroethane	0.22	10000	0.00			3	0.29	0.40
chloroform	0.81	98	0.01	2.30E-05	1.87E-05	17	0.54	5.86
chloromethane	1.62	90	0.02	1.80E-06	2.91E-06	50	0.62	2.89
cumene	0.39	400	0.00			3	0.54	1.92
cyclohexane	0.27	6000	0.00			10	0.34	1.27
decane	0.53					3	0.87	5.12
1,4-dichlorobenzene (para)	0.61	800	0.00	1.00E-05	5.33E-06	7	0.66	4.57
dichlorodifluoromethane	3.10					50	1.09	4.30
n-dodecane	0.48					1	0.77	0.77
ethanol	31.66					17	5.65	471.06
ethylbenzene	1.71	1000	0.00			25	0.43	29.53
4-ethyltoluene	0.90					7	0.69	13.77
n-heptane	0.82					14	0.45	9.43
hexane	2.82	700	0.00			41	0.49	26.08
methylene chloride	0.71	1000	0.00	4.70E-07	3.92E-07	27	0.38	3.82
a-methylstyrene	0.47					8	0.48	1.50
4-methyl-2-pentanone	0.51					4	0.57	8.60
naphthalene	0.83	3	0.28			11	0.52	8.39
n-nonane	0.45					4	0.63	5.25
n-octane	0.59					6	0.47	7.94
n-pentane	14.94					50	0.62	171.15
propylene	0.75					13	0.53	7.23
n-propyl benzene	0.69					4	0.49	8.36
styrene	1.16	1000	0.00	5.70E-07	6.59E-07	24	0.47	5.54
1, 1, 2, 2-tetrachloroethane	0.48			5.80E-05	2.80E-05	1	2.75	2.75
tetrachloroethylene	0.46			9.50E-07	4.37E-07	2	0.81	1.09
toluene	7.37	5000	0.00			49	0.64	41.45
1,1,1-trichloroethane	0.38					1	1.58	1.58
trichlorofluoromethane	2.09					38	0.79	32.03
1,1,2-trichloro-1,2,2-trifluoroethane	0.59					7	0.77	1.15
1,2,4-trimethylbenzene	2.85					33	0.49	49.16
1,3,5-trimethylbenzene	0.95					9	0.64	19.17
n-undecane	0.68					3	0.90	1.53
o-xylene	2.04	100	0.02			29	0.43	34.30
total m+p-xylene	4.93	100	0.05			35	0.43	56.44

A1-10. Hamilton County, Cincinnati State (2003-2007)

Source Related VOC Sampling Cuyahoga					**Total Risk**	**2003-2007**		
Location:	HAMILTON							
	Hamilton County							
	Cincinnati State community College							
	10100 Reading Rd.							
	Reading, Cincinnati							
AIRS#:								

Compounds	Average (µg/m3) with half detections	RBC	Hazard Index	UR	RISK	Frequency	Minimum value detected (µg/m3)	Maximum value detected (µg/m3)
acetone	17.97	31000	0.00			98	5.23	78.39
acetonitrile	0.69	60	0.01			65	0.34	11.42
acrylonitrile	0.29	2	0.14	6.80E-05	1.96E-05	11	0.43	2.02
benzene	5.07	30	0.17	7.80E-06	3.96E-05	98	0.54	47.92
1,3-butadiene	0.13	2	0.06	3.00E-05	3.78E-06	9	0.22	0.55
n-butane	2.51					96	0.71	10.93
2-butanone	2.95	5000	0.00			91	1.47	10.32
carbon disulfide	0.84	700	0.00			3	1.68	2.27
carbon tetrachloride	0.56	40	0.01	1.50E-05	8.40E-06	63	0.63	1.01
chlorobenzene	0.43	1000	0.00			30	0.46	2.49
chlorodifluoromethane	1.03	50000	0.00			45	0.95	21.22
chloroethane	0.14	10000	0.00			2	0.26	1.77
chloroform	0.25	98	0.00	2.30E-05	5.70E-06	2	0.54	0.98
chloromethane	1.19	90	0.01	1.80E-06	2.15E-06	95	0.70	8.26
cyclohexane	0.18	6000	0.00			4	0.34	0.62
decane	1.93					47	0.58	23.28
1,4-dichlorobenzene (para)	0.66	800	0.00	1.00E-05	6.64E-06	19	0.60	18.64
dichlorodifluoromethane	2.17					94	1.58	3.71
1,1-dichloroethane	0.20					1	0.40	0.40
cis-1,2-dichloroethene	0.20					1	0.44	0.44
trans-1,2-dichloroethene	0.20					1	0.44	0.44
n-dodecane	0.36					1	0.70	0.70
ethylbenzene	0.25	1000	0.00			10	0.48	0.82
4-ethyltoluene	0.33					9	0.25	2.26
n-heptane	0.24					14	0.41	0.78
hexachlorobutadiene	0.55			2.20E-05	1.20E-05	1	2.67	2.67
hexane	0.59	700	0.00			25	0.74	4.93
methylene chloride	0.38	1000	0.00	4.70E-07	1.79E-07	54	0.35	1.74
4-methyl-2-pentanone	0.24					16	0.41	0.86
naphthalene	0.33	3	0.11			10	0.52	1.99
n-nonane	1.06					25	0.52	19.93
n-octane	0.26					7	0.56	1.35
n-pentane	1.36					97	0.44	9.15
propylene	1.24					84	0.55	3.96
n-propyl benzene	0.30					6	0.54	1.52
styrene	0.24	1000	0.00	5.70E-07	1.39E-07	8	0.51	1.24
toluene	1.52	5000	0.00			93	0.38	9.80
1,1,1-trichloroethane	43.68					91	18.55	152.79
trichlorofluoromethane	2.27					99	1.24	21.35
1,1,2-trichloro-1,2,2-trifluoroethane	0.49					21	0.77	1.07
1,2,4-trimethylbenzene	1.27					47	0.49	9.83
1,3,5-trimethylbenzene	0.39					11	0.59	3.29
n-undecane	1.18					29	0.64	10.23
vinyl acetate	1.33	200	0.01			46	0.35	8.45
o-xylene	0.30	100	0.00			17	0.56	1.22
total m+p-xylene	0.57	100	0.01			24	0.87	4.34

A1-11. Jefferson County (2004-2009)

Source Related VOC Sampling Steubenville						**Total Risk 2004-2009**		
Location:	SEDO							
	Jefferson County							
	Steubenville Trailer							
	618 Logan Street							
	Steubenville							
AIRS #	39-081-0017							
Compounds	**Average (µg/m3) with half detections**	**RBC**	**Hazard Index**	**UR**	**RISK**	**Frequency**	**Minimum value detected (µg/m3)**	**Maximum value detected (µg/m3)**
acetone	11.70	31000	0.00			194	2.38	140.15
acetonitrile	0.55	60	0.01			119	0.34	4.03
acrylonitrile	0.79	2	0.40	6.80E-05	5.37E-05	77	0.43	6.08
benzene	7.11	30	0.24	7.80E-06	5.55E-05	178	0.32	89.45
1,3-butadiene	0.19	2	0.10	3.00E-05	5.70E-06	23	0.22	0.75
n-butane	3.80					193	0.36	26.15
2-butanone	1.75	5000	0.00			105	0.74	17.10
carbon disulfide	1.29	700	0.00			38	1.56	11.21
carbon tetrachloride	0.53	40	0.01	1.50E-05	7.95E-06	42	0.63	1.01
chlorodifluoromethane	1.03	50000	0.00			172	0.46	3.89
chloroform / trichloromethane	0.34	98	0.00	2.30E-05	7.82E-06	1	0.49	0.49
chloromethane / methyl chloride	1.13	90	0.01	1.80E-06	2.03E-06	193	0.47	2.02
cyclohexane	0.25	6000	0.00			5	0.34	1.10
decane	0.46					6	0.58	6.98
1,4-dichlorobenzene (para)	0.48	800	0.00	1.00E-05	4.80E-06	8	1.08	2.71
dichlorodifluoromethane	2.68					193	1.38	3.81
n-dodecane	0.35					1	0.77	0.77
1,2-Dichloroethane	0.29			2.60E-05	7.54E-06	1	1.09	1.09
ethylbenzene	0.44	1000	0.00			36	0.43	3.21
4-ethyltoluene	0.35					1	0.54	0.54
n-heptane	0.51					55	0.41	7.38
hexachlorobutadiene	0.76			2.20E-05	1.67E-05	1	3.52	3.52
hexane	0.69	700	0.00			68	0.70	5.64
methylene chloride / dichloromethane	0.30	1000	0.00	4.70E-07	1.41E-07	28	0.35	1.81
4-methyl-2-pentanone	0.58					36	0.41	9.83
napthalene	5.80	3	1.93			109	0.52	162.49
n-nonane	0.38					1	2.05	2.05
n-octane	0.33					2	0.47	0.51
n-pentane	1.84					190	0.32	15.05
propylene	1.09					95	0.53	7.06
n-propyl benzene	0.35					1	0.84	0.84
styrene	0.35	1000	0.00	5.70E-07	2.00E-07	17	0.43	1.96
toluene	2.85	5000	0.00			164	0.41	21.48
1,1,1-trichloroethane	0.38					1	0.55	0.55
Trichloroethene	0.39	60	0.01	2.00E-06	7.80E-07	2	1.45	1.77
trichlorofluoromethane	5.00					193	1.12	67.43
1,1,2-trichloro-1,2,2-trifluoroethane	0.57					15	0.77	1.00
1,2,4-trimethylbenzene	0.53					39	0.49	2.70
1,3,5-trimethylbenzene	0.35					5	0.49	0.84
n-undecane	0.48					1	6.01	6.01
vinyl acetate	1.57	200	0.01			112	0.74	14.44
o-xylene	0.46	100	0.00			40	0.48	2.74
total m+p-xylene	1.38	100	0.01			66	0.87	13.03

A1-12. Lake County (2000-2004)

Source Related VOC Sampling Paineville								
Location:	E COUNTY GHD							
	Lake County							
	Lake County Hospital							
	71 E. High St							
	Paineville							
AIRS SITE #:	39-085-3002							
				Total Risk 2000-2004				

Compounds	Average (µg/m3) with half detections	RBC	Hazard Index	UR	RISK	Frequency	Minimum value detected (µg/m3)	Maximum value detected (µg/m3)
acetone	9.37	31000	0.00			118	2.61	38.01
acetonitrile	0.30	60	0.01			37	0.34	1.16
acrylonitrile	0.41	2	0.21	6.80E-05	2.79E-05	34	0.43	1.54
benzene	0.77	30	0.03	7.80E-06	6.01E-06	124	0.32	2.59
1,3-butadiene	0.14	2	0.07	3.00E-05	4.20E-06	7	0.22	0.73
n-butane	2.89					131	0.64	9.03
2-butanone	1.04	5000	0.00			57	0.65	10.62
carbon disulfide	1.00	700	0.00			18	0.40	4.67
carbon tetrachloride	0.50	40	0.01	1.50E-05	7.50E-06	59	0.63	1.01
chlorodifluoromethane	17.75	50000	0.00			113	0.57	1060.98
chloroethane / ethyl chloride	0.17	10000	0.00			1	0.92	0.92
chloroform / trichloromethane	0.25	98	0.00	2.30E-05	5.75E-06	2	0.49	1.12
chloromethane / methyl chloride	1.06	90	0.01	1.80E-06	1.91E-06	125	0.45	2.07
3-chloropropene	0.16					1	0.31	0.31
decane	0.34					2	0.93	1.45
dichlorodifluoromethane	2.83					131	0.25	5.44
ethanol	1.46					3	1.36	11.49
ethylbenzene	0.27	1000	0.00			10	0.48	1.22
4-ethyltoluene	0.32					2	0.84	1.62
n-heptane	0.25					9	0.41	0.49
hexachlorobutadiene	0.60			2.20E-05	1.32E-05	1	1.07	1.07
hexane	0.56	700	0.00			45	0.39	2.40
methylene chloride / dichloromethane	0.27	1000	0.00	4.70E-07	1.27E-07	21	0.35	2.95
4-methyl-2-pentanone	0.22					2	0.45	1.47
a-methylstyrene	0.27					1	0.63	0.63
napthalene	0.41	3	0.14			7	0.26	5.77
n-pentane	1.53					127	0.41	4.72
propylene	0.53					32	0.55	4.99
n-propyl benzene	0.31					1	0.74	0.74
styrene	0.21	1000	0.00	5.70E-07	1.20E-07	1	0.43	0.43
tetrachloroethylene	0.39			9.50E-07	3.71E-07	3	0.68	1.29
toluene	1.88	5000	0.00			125	0.38	9.80
1,1,1-trichloroethane	0.52					31	0.55	16.92
trichloroethene	0.27	60	0.00	2.00E-06	5.40E-07	1	0.59	0.59
trichlorofluoromethane	1.53					111	1.12	2.98
1,1,2-trichloro-1,2,2-trifluoroethane	0.51					20	0.77	1.84
1,2,4-trimethylbenzene	0.30					11	0.49	1.77
n-undecane	0.45					1	1.53	1.53
vinyl acetate	0.61	200	0.00			17	1.02	4.93
o-xylene	0.31	100	0.00			17	0.22	1.43
total m+p-xylene	0.83	100	0.01			51	0.22	21.71

A1-13. Montgomery County (2003-2004)

Source Related VOC Sampling Dayton					Total Risk 2003-2004			
Location:	RAPCA							
	Montgomery County							
	Downtown Dayton Library							
	215 E. Third Str.							
	Dayton							
AIRS SITE #:	39-113-0032							

Compounds	Average (µg/m3) with half detections	RBC	Hazard Index	UR	RISK	Frequency	Minimum value detected (µg/m3)	Maximum value detected (µg/m3)
acetone	9.62	31000	0.00			42	26.13	10.91
acetonitrile	0.36	60	0.01			21	0.82	0.60
acrylonitrile	1.13	2	0.57	6.80E-05	7.68E-05	34	6.08	1.51
benzene	1.41	30	0.05	7.80E-06	1.10E-05	48	3.51	1.41
1,3-butadiene	0.18	2	0.09	3.00E-05	5.40E-06	12	0.69	0.37
n-butane	3.41					48	12.84	3.41
2-butanone	1.51	5000	0.00			25	6.78	2.77
carbon disulfide	5.89	700	0.01			41	20.86	6.76
carbon tetrachloride	0.67	40	0.02	1.50E-05	1.01E-05	41	0.88	0.73
chlorodifluoromethane	1.63	50000	0.00			48	15.56	1.63
chloroform / trichloromethane	0.29	98	0.00	2.30E-05	6.67E-06	6	0.78	0.63
chloromethane / methyl chloride	0.92	90	0.01	1.80E-06	1.66E-06	47	1.24	0.94
cyclohexane	0.19	6000	0.00			4	0.41	0.38
decane	0.65					5	13.39	3.76
dichlorodifluoromethane	3.01					47	18.79	3.07
cis-1,2-dichloroethene	0.21					2	0.48	0.46
n-dodecane	0.37					1	1.18	1.18
ethylbenzene	0.29	1000	0.00			7	1.13	0.71
4-ethyltoluene	0.30					3	2.21	1.15
n-heptane	0.31					12	1.07	0.62
hexane	1.11	700	0.00			31	3.42	1.52
methylene chloride / dichloromethane	0.43	1000	0.00	4.70E-07	2.02E-07	27	1.29	0.63
4-methyl-2-pentanone	0.22					3	0.45	0.42
napthalene	0.33	3	0.11			6	1.05	0.79
n-nonane	0.37					4	3.15	1.52
n-octane	0.30					3	2.29	1.32
n-pentane	1.96					48	8.26	1.96
propylene	0.97					19	5.68	2.31
n-propyl benzene	0.26					1	0.93	0.93
tetrachloroethylene	0.45			9.50E-07	4.28E-07	9	1.22	0.93
toluene	2.64	5000	0.00			48	10.93	2.64
trichloroethene	0.37	60	0.00	2.00E-06	7.40E-07	7	1.50	0.96
trichlorofluoromethane	2.99					48	9.55	2.99
1,1,2-trichloro-1,2,2-trifluoroethane	0.48					11	1.07	0.82
1,2,4-trimethylbenzene	0.52					10	5.90	1.56
1,3,5-trimethylbenzene	0.32					3	3.24	1.47
n-undecane	0.58					3	11.51	4.45
vinyl acetate	0.71	200	0.00			8	4.93	2.51
o-xylene	0.33	100	0.00			10	1.61	0.78
total m+p-xylene	0.81	100	0.01			17	4.78	1.88

A1-14. Scioto County (2000-2003)

Source Related VOC Sampling New Boston **Total Risk 2000-2003**

Location:	Scioto Co. Portsmouth Water Treatment Plant 4862 Gallia New Boston, OH
AIRS #	39-145-0013
Sampler:	Xon.J23414

Compounds	Average (µg/m3) with half detections	RBC	Hazard Index	UR	RISK	Frequency	Minimum value detected (µg/m3)	Maximum value detected (µg/m3)
acetone	7.69	31000.00	0.00			80	2.38	9.82
acetonitrile	0.26	60.00	0.00			14	0.34	0.55
acrylonitrile	0.45	2.00	0.23	6.80E-05	3.06E-05	21	0.48	1.17
benzene	4.90	30.00	0.16	7.80E-06	3.82E-05	102	0.42	5.00
1,3-butadiene	0.22	2.00	0.11	3.00E-05	6.60E-06	20	0.27	0.55
n-butane	6.93					101	1.00	7.13
2-butanone	1.46	5000.00	0.00			57	0.56	2.54
carbon disulfide	3.11	700.00	0.00			48	0.78	5.84
carbon tetrachloride	0.48	40.00	0.01	1.50E-05	7.20E-06	36	0.63	0.78
chlorobenzene	0.40	1000.00	0.00			1	11.05	11.05
chlorodifluoromethane	0.63	50000.00	0.00			67	0.42	0.83
chloroethane / ethyl chloride	0.22	10000.00	0.00			1	0.92	0.92
chloroform / trichloromethane	0.49	98.00	0.01	2.30E-05	1.13E-05	14	0.49	2.05
chloromethane / methyl chloride	1.09	90.00	0.01	1.80E-06	1.96E-06	95	0.52	1.17
cumene	0.34	400.00	0.00			6	0.54	0.67
cyclohexane	0.25	6000.00	0.00			3	0.59	2.75
decane	0.41					4	0.76	1.21
1,4-dichlorobenzene (para)	0.40	800.00	0.00	1.00E-05	4.00E-06	1	0.96	0.96
dichlorodifluoromethane	2.67					103	1.04	2.69
1,2-dichloro-1,1,2,2-tetrafluoroethane	0.46					1	0.70	0.70
n-dodecane	0.46					3	0.70	0.95
ethanol	13.59					19	0.96	21.93
ethylbenzene	0.32	1000.00	0.00			12	0.43	0.58
4-ethyltoluene	0.41					1	1.57	1.57
n-heptane	0.28					6	0.45	0.64
hexane	0.54	700.00	0.00			34	0.18	0.98
methylene chloride / dichloromethane	0.29	1000.00	0.00	4.70E-07	1.36E-07	20	0.38	0.80
a-methylstyrene	0.32					1	0.82	0.82
napthalene	1.93	3.00	0.64			47	0.52	3.72
n-octane	0.31					1	0.98	0.98
n-pentane	2.11					99	0.35	2.20
propylene	0.77					14	1.29	4.71
styrene	0.48	1000.00	0.00	5.70E-07	2.74E-07	4	0.47	7.27
toluene	1.73	5000.00	0.00			91	0.45	1.93
1,2,4-trichlorobenzene	0.70					1	0.82	0.82
1,1,1-trichloroethane	0.36					1	1.36	1.36
trichloroethene	0.29	60.00	0.00			2	1.07	1.34
trichlorofluoromethane	40.58					101	1.12	41.77
1,1,2-trichloro-1,2,2-trifluoroethane	0.60					15	0.77	1.09
1,2,4-trimethylbenzene	0.40					26	0.49	0.87
n-undecane	0.63					4	0.77	1.26
vinyl acetate	1.33	200.00	0.01			14	0.77	7.65
o-xylene	0.38	100.00	0.00			20	0.43	0.76
total m+p-xylene	0.79	100.00	0.01			31	0.56	1.60

A1-15. Summit County (2000-2003)

Source Related VOC Sampling	**Akron**			**Total Risk 2000-2003**				
Location:	Akron Region Air Quality Management District							
	Summit County							
	East High School							
	80 Brittain Rd.							
	Akron							
AIRS SITE #:	39-153-0017							
Compounds	**Average (µg/m3) with half detections**	**RBC**	**Hazard Index**	**UR**	**RISK**	**Frequency**	**Minimum value detected (µg/m3)**	**Maximum value detected (µg/m3)**
acetone	9.00	31000	0.00			42	3.80	35.63
acetonitrile	0.27	60	0.00			9	0.34	0.87
acrylonitrile	0.34	2	0.17	6.80E-05	2.31E-05	6	0.50	2.08
benzene	1.33	30	0.04	7.80E-06	1.04E-05	60	0.42	4.15
1,3-butadiene	0.29	2	0.15	3.00E-05	8.70E-06	22	0.24	1.24
n-butane	6.09					57	0.95	52.30
2-butanone	2.50	5000	0.00			46	1.18	20.05
carbon disulfide	6.61	700	0.01			37	1.59	121.45
carbon tetrachloride	0.49	40	0.01	1.50E-05	7.35E-06	24	0.63	1.01
chlorodifluoromethane	1.67	50000	0.00			46	0.35	19.45
chloroethane / ethyl chloride	0.21	10000	0.00			3	0.29	0.58
chloromethane / methyl chloride	0.99	90	0.01	1.80E-06	1.78E-06	51	0.68	1.76
cyclohexane	0.32	6000	0.00			11	0.34	2.44
decane	0.48					6	0.81	3.08
1,4-dichlorobenzene (para)	0.41	800	0.00	1.00E-05	4.10E-06	2	1.20	1.62
dichlorodifluoromethane	3.81					58	1.09	47.97
n-dodecane	0.50					4	0.84	2.44
ethanol	176.86					14	5.46	753.70
ethylbenzene	0.67	1000	0.00			34	0.43	2.95
4-ethyltoluene	0.38					2	0.59	0.79
n-heptane	0.48					13	0.41	4.51
hexane	1.78	700	0.00			51	0.35	10.57
methylene chloride / dichloromethane	0.55	1000	0.00	4.70E-07	2.59E-07	31	0.38	2.54
4-methyl-2-pentanone	0.25					5	0.45	1.47
a-methylstyrene	0.33					4	0.53	0.92
napthalene	0.48	3	0.16			4	0.52	1.21
n-nonane	0.32					5	0.63	1.26
n-octane	0.33					4	0.47	1.68
n-pentane	14.34					58	0.94	268.53
propylene	0.33					4	0.64	4.82
n-propyl benzene	0.38					1	0.69	0.69
styrene	0.98	1000	0.00	5.70E-07	5.59E-07	33	0.47	8.52
tetrachloroethylene	0.44			9.50E-07	4.18E-07	2	0.88	1.02
toluene	3.86	5000	0.00			57	0.45	64.06
1,1,1-trichloroethane	501.21					60	3.98	9822.33
trichlorofluoromethane	24.91					57	1.12	252.85
1,1,2-trichloro-1,2,2-trifluoroethane	0.51					4	0.84	0.92
1,2,4-trimethylbenzene	0.64					24	0.49	5.41
1,3,5-trimethylbenzene	0.27					3	0.64	0.84
n-undecane	0.65					6	0.64	2.43
vinyl acetate	0.45	200	0.00			5	0.70	2.71
o-xylene	0.61	100	0.01			34	0.43	4.34
total m+p-xylene	1.98	100	0.02			46	0.91	14.33

A1-16. Washington County, Washington County Career Center (2000-2008)

Source Related VOC Sampling Marietta					**Total Risk 2000-2008**			
Location:	SEDO							
	Washington County							
	Washington County Career Center							
	Rt 2							
	Marietta							
AIRS # :	39-167-0008							
Compounds	**Average (µg/m3) with half detections**	**RBC**	**Hazard Index**	**UR**	**RISK**	**Frequency**	**Maximum value detected (µg/m3)**	**Minimum value detected (µg/m3)**
acetone	9.32	31000	0.00			234	59.39	1.95
acetonitrile	0.40	60	0.01			73	3.02	0.34
acrylonitrile	1.59	2	0.79	6.80E-05	1.08E-04	162	8.90	0.43
benzene	0.59	30	0.02	7.80E-06	4.57E-06	186	8.31	0.32
bromomethane	0.23	5	0.05			1	0.85	0.85
1,3-butadiene	0.14	2	0.07	3.00E-05	4.32E-06	10	0.80	0.24
n-butane	4.55					253	23.06	0.36
2-butanone	1.29	5000	0.00			131	12.09	0.56
carbon disulfide	3.17	700	0.00			178	29.27	1.59
carbon tetrachloride	0.46	40	0.01	1.50E-05	6.87E-06	74	1.26	0.63
chlorobenzene	0.54	1000	0.00			35	6.45	0.46
chlorodifluoromethane	1.58	50000	0.00			216	45.98	0.39
chloroethane / ethyl chloride	0.18	10000	0.00			3	1.56	0.58
chloroform / trichloromethane	0.26	98	0.00	2.30E-05	5.97E-06	1	0.54	0.54
chloromethane / methyl chloride	1.24	90	0.01	1.80E-06	2.24E-06	247	6.40	0.47
cyclohexane	0.31	6000	0.00			51	3.27	0.34
decane	0.37					5	3.14	0.58
1,4-dichlorobenzene (para)	0.44	800	0.00	1.00E-05	4.43E-06	8	7.22	0.60
dichlorodifluoromethane	2.59					253	6.43	0.99
n-dodecane	0.39					2	0.84	0.77
ethanol	1.04					14	154.51	1.09
ethylbenzene	0.29	1000	0.00			7	3.00	0.61
4-ethyltoluene	0.33					3	1.57	0.64
n-heptane	0.36					42	10.66	0.41
hexane	0.74	700	0.00			98	16.21	0.39
methanol	0.66					1	129.73	129.73
methylene chloride / dichloromethane	0.26	1000	0.00	4.70E-07	1.22E-07	28	3.34	0.35
4-methyl-2-pentanone	0.23					9	1.11	0.45
a-methylstyrene	0.29					1	0.63	0.63
napthalene	0.41	3	0.14			15	3.09	0.52
n-nonane	0.29					4	1.26	0.52
n-octane	0.30					14	1.64	0.47
n-pentane	1.77					247	16.23	0.30
propylene	1.09					84	9.47	0.62
n-propyl benzene	0.32					1	0.79	0.79
styrene	0.32	1000	0.00	5.70E-07	1.83E-07	20	4.09	0.51
toluene	0.98	5000	0.00			133	28.26	0.38
1,2,4-trichlorobenzene	0.52					1	0.89	0.89
1,1,1-trichloroethane	0.33					2	0.93	0.60
trichlorofluoromethane	10.12					253	84.28	1.12
1,1,2-trichloro-1,2,2-trifluoroethane	0.61					67	2.68	0.77
1,2,4-trimethylbenzene	0.35					10	9.83	0.49
1,3,5-trimethylbenzene	0.28					4	2.51	0.54
n-undecane	0.48					4	3.07	0.90
vinyl acetate	1.10	200	0.01			83	16.90	0.70
o-xylene	0.31	100	0.00			9	3.82	0.43
total m+p-xylene	0.59	100	0.01			17	14.76	0.56

Appendix A2, Average Total Heavy Metals Site Risk Per County

A2-1. Butler County (2004-2009)

MIDDLETOWN HEAVY METALS DATA, 2004-2009								
Ohio Bell								
3901 Lefferson Rd.								
AQS: 39-017-0015								
Butler								
Compound	**Average µg/m³**	**RBC**	**HI**	**UR**	**RISK**	**Frequency**	**Min µg/m³**	**Max µg/m³**
beryllium	0.000	0.02	0.00	2.40E-03	1.30E-07	65	0.000	0.000
chromium	0.004	0.10	0.04	1.00E-02	4.28E-05	71	0.001	0.010
lead	0.007	1.50	0.00	1.20E-05	8.06E-08	72	0.002	0.021
manganese	0.054	0.05	1.09			72	0.012	0.150
nickel	0.004	0.20	0.02	2.40E-04	8.43E-07	22	0.001	0.013
zinc	0.071					72	0.019	0.630
arsenic	0.001			4.30E-03	5.79E-06	71	0.001	0.003
cadmium	0.000			1.80E-03	6.27E-07	72	0.000	0.003
iron	0.254					45	0.120	1.100

A2-2. Butler County, Oneida School (2000-2002)

Middletown Heavy Metals Data, 2000-2002								
Oneida School I								
Yankee Rd.								
AIRS: 39-017-0014, units -- µg/m³								
Butler County, Parameters								
Compound	**Average µg/m³**	**RBC**	**HI**	**UR**	**RISK**	**Frequency**	**Min µg/m³**	**Max µg/m³**
beryllium	0.000	0.02	0.00	2.40E-03	1.42E-07	30	0.000	0.000
chromium	0.005	0.10	0.05	1.80E-02	8.29E-05	30	0.002	0.009
lead	0.012	1.50	0.01	1.20E-05	1.50E-07	30	0.005	0.077
manganese	0.079	0.05	1.57			30	0.017	0.160
nickel	0.005	0.20	0.03	2.40E-04	1.21E-06	30	0.003	0.008
zinc	0.049					30	0.015	0.121
arsenic	0.002			4.30E-03	7.34E-06	30	0.000	0.004

A2-3. Coshocton County, Johnson Plumbing (2000-2002)

Coshocton County HEAVY METALS DATA, 2000-2002								
1840 Otsego Ave.								
AIRS: 39-031-0005								
Coshocton County, units -- µg/m³								
Compound	**Average µg/m³**	**RBC**	**HI**	**UR**	**RISK**	**Frequency**	**Min µg/m³**	**Max µg/m³**
beryllium	0.000	0.02	0.00	2.40E-03	8.06E-08	19	0.000	0.000
chromium	0.005	0.10	0.05	1.00E-02	4.81E-05	19	0.003	0.010
lead	0.012	1.50	0.01	1.20E-05	1.48E-07	19	0.006	0.028
manganese	0.050	0.05	0.99			19	0.008	0.590
nickel	0.006	0.20	0.03	2.40E-04	1.54E-06	19	0.004	0.013
zinc	0.095					19	0.045	0.240
arsenic	0.002			4.30E-03	6.99E-06	19	0.001	0.004

A2-4. Columbiana County, Maryland Ave. (2001-2009)

EAST LIVERPOOL HEAVY METALS DATA, 2001 - 2009								
500 Maryland Ave.								
AIRS: 39-029-0022, units -- µg/m³								
Columbiana - designated								
Compound	**Average µg/m³**	**RBC**	**HI**	**UR**	**RISK**	**Frequency**	**Min µg/m³**	**Max µg/m³**
beryllium	0.000	0.02	0.00	2.40E-03	9.54E-08	82	0.000	0.000
chromium	0.004	0.10	0.04	1.00E-02	4.21E-05	98	0.001	0.031
lead	0.015	1.50	0.01	1.20E-05	1.77E-07	98	0.003	0.081
manganese	0.205	0.05	4.09			98	0.009	1.000
nickel	0.005	0.20	0.03	2.40E-04	1.28E-06	97	0.000	0.028
zinc	0.094					98	0.011	0.490
arsenic	0.003			4.30E-03	1.49E-05	96	0.001	0.150
cadmium	0.001			1.80E-03	1.77E-06	98	0.000	0.005
mercury	0.000	0.30	0.00			96	0.000	0.001
iron	0.148					59	0.011	0.600
potassium	0.324					47	0.125	0.600

A2-5. Columbiana County, Port Authority (2000-2009)

EAST LIVERPOOL HEAVY METALS DATA, 2000 - 2009								
Port Authority								
1250 St. George St.:								
AIRS: 39-029-0019, units -- µg/m³								
Columbiana County, Parameters								
Compound	**Average µg/m³**	**RBC**	**HI**	**UR**	**RISK**	**Frequency**	**Min µg/m³**	**Max µg/m³**
beryllium	0.000	0.02	0.01	2.40E-03	2.96E-07	94	0.000	0.000
chromium	0.006	0.10	0.06	1.00E-02	5.57E-05	95	0.001	0.043
lead	0.017	1.50	0.01	1.20E-05	2.00E-07	95	0.002	0.076
manganese	0.372	0.05	7.44			95	0.018	1.900
nickel	0.003	0.20	0.02	2.40E-04	7.86E-07	95	0.000	0.024
zinc	0.116					95	0.022	0.540
arsenic	0.002			4.30E-03	1.06E-05	95	0.001	0.009
cadmium	0.001			1.80E-03	2.00E-06	95	0.000	0.007
mercury	0.000	0.30	0.00			92	0.000	0.002
iron	0.211					47	0.075	0.900

A2-6. Columbiana County, Water Plant (2000-2009)

East Liverpool Hetal Metals Data, 2000 - 2009								
Water Plant								
2220 Michigan Ave.								
AIRS: 39-029-0019								
Columbiana								
Compound	**Average µg/m³**	**RBC**	**HI**	**UR**	**RISK**	**Frequency**	**Min µg/m³**	**Max µg/m³**
beryllium	0.000	0.02	0.00	2.40E-03	1.33E-07	86	0.000	0.001
chromium	0.016	0.10	0.16	1.20E-02	1.93E-04	96	0.001	0.087
lead	0.016	1.50	0.01	1.20E-05	1.94E-07	96	0.002	0.067
manganese	1.602	0.05	32.05			96	0.100	6.800
nickel	0.011	0.20	0.06	2.40E-04	2.65E-06	96	0.002	0.088
zinc	0.109					96	0.018	0.720
arsenic	0.003			4.30E-03	1.21E-05	96	0.001	0.020
cadmium	0.001			1.80E-03	1.94E-06	96	0.000	0.005
mercury	0.000	0.30	0.00			94	0.000	0.001
iron	0.300					47	0.130	1.600

A2-7. Cuyahoga County, St. Theodosius Church (2000-2009)

CLEVELAND, 2000-2009
Cuyahoga County
St. Theodosius Church
2547 St. Tikhon Ave.
AIRS: 39-035-0038

Compound	Average µg/m³	RBC	HI	UR	RISK	Frequency	Min µg/m³	Max µg/m³
beryllium	0.000	0.02	0.00	2.40E-03	1.03E-07	93	0.000	0.000
chromium	0.003	0.10	0.03	1.20E-02	3.81E-05	91	0.000	0.008
lead	0.018	1.50	0.01	1.20E-05	2.14E-07	119	0.004	0.061
manganese	0.043	0.05	0.86			119	0.006	0.160
nickel	0.003	0.20	0.02	2.40E-04	8.12E-07	77	0.000	0.010
zinc	0.077					119	0.025	0.240
arsenic	0.002			4.30E-03	6.57E-06	119	0.000	0.004

A2-8. Cuyahoga County, Ferro "A"/Ferro “B” (2000-2009)

CLEVELAND, 2000-2009
Cuyahoga County
FERRO "A"
4150 East 56th Street
AIRS: 39-035-0049

Compound	Average µg/m³	RBC	HI	UR	RISK	Frequency	Min µg/m³	Max µg/m³
beryllium	0.000	0.02	0.00	2.40E-03	1.39E-07	100	0.000	0.000
chromium	0.005	0.10	0.05	1.20E-02	6.59E-05	110	0.000	0.015
lead	0.104	1.50	0.07	1.20E-05	1.25E-06	120	0.015	0.450
manganese	0.129	0.05	2.57			120	0.020	0.710
nickel	0.060	0.20	0.30	2.40E-04	1.43E-05	119	0.002	1.860
zinc	0.116					120	0.044	0.820
arsenic	0.002			4.30E-03	7.58E-06	120	0.001	0.004
cadmium	0.001			1.80E-03	1.24E-06	120	0.000	0.002

CLEVELAND, 2000-2009
Cuyahoga County
FERRO "B"
4150 East 56th Street
AIRS: 39-035-0049

Compound	Average µg/m³	RBC	HI	UR	RISK	Frequency	Min µg/m³	Max µg/m³
beryllium	0.000	0.02	0.00	2.40E-03	1.53E-07	55	0.000	0.000
chromium	0.006	0.10	0.06	1.20E-02	7.06E-05	58	0.002	0.012
lead	0.106	1.50	0.07	1.20E-05	1.27E-06	60	0.011	0.263
manganese	0.135	0.05	2.70			60	0.009	0.303
nickel	0.052	0.20	0.26	2.40E-04	1.26E-05	60	0.005	0.340
zinc	0.118					60	0.034	0.300
arsenic	0.002			4.30E-03	7.06E-06	60	0.001	0.003
cadmium	0.001			1.80E-03	1.34E-06	60	0.000	0.002

A2-9. Cuyahoga County, Asphalt Plant A (2001-2009)

CLEVELAND, 2001-2009								
Cuyahoga County								
Asphalt Plant								
West 3rd St.								
AIRS: 39-035-0061								
Compound	**Average µg/m³**	**RBC**	**HI**	**UR**	**RISK**	**Frequency**	**Min µg/m³**	**Max µg/m³**
beryllium	0.000	0.02	0.00	2.40E-03	1.54E-07	91	0.000	0.001
chromium	0.004	0.10	0.04	1.00E-02	3.65E-05	85	0.000	0.014
lead	0.032	1.50	0.02	1.20E-05	3.80E-07	107	0.004	0.560
manganese	0.059	0.05	1.18			107	0.007	0.310
nickel	0.004	0.20	0.02	2.40E-04	8.51E-07	65	0.000	0.014
zinc	0.091					107	0.025	0.500
arsenic	0.001			4.30E-03	6.37E-06	107	0.000	0.004

A2-10. Cuyahoga County, Fortran Printing Inc. (2000-2009)

Cuyahoga County								
Fortran Printing Inc								
5777 GRANT AVE.								
AIRS: 39-035-0050								
Fortran Printing Inc								
Compound	**Average µg/m³**	**RBC**	**HI**	**UR**	**RISK**	**Frequency**	**Min µg/m³**	**Max µg/m³**
beryllium	0.000	0.02	0.00	2.40E-03	9.36E-08	86	0.000	0.000
chromium	0.004	0.10	0.04	1.00E-02	4.28E-05	100	0.000	0.012
lead	0.033	1.50	0.02	1.20E-05	3.93E-07	119	0.000	0.140
manganese	0.067	0.05	1.35			120	0.010	0.220
nickel	0.013	0.20	0.07	2.40E-04	3.17E-06	112	0.000	0.078
zinc	0.072					120	0.023	0.183
arsenic	0.002			4.30E-03	8.60E-06	120	0.001	0.005
cadmium	0.001			1.80E-03	1.09E-06	120	0.000	0.002

A2-11. Cuyahoga County, Fire "4A", "4B" (2000-2009)

Cuyahoga County, 2000-2009
FIRE "4A"
3136 Lorain Ave.
AIRS: 39-035-0042

Compound	Average µg/m³	RBC	HI	UR	RISK	Frequency	Min µg/m³	Max µg/m³
beryllium	0.000	0.02	0.00	2.40E-03	5.59E-08	63	0.000	0.000
chromium	0.002	0.10	0.02	1.20E-02	2.95E-05	69	0.000	0.005
lead	0.016	1.50	0.01	1.20E-05	1.91E-07	120	0.000	0.049
manganese	0.019	0.05	0.37			118	0.000	0.050
nickel	0.004	0.20	0.02	2.40E-04	8.99E-07	71	0.000	0.019
zinc	0.060					118	0.000	0.160
arsenic	0.001			4.30E-03	5.93E-06	117	0.000	0.004
cadmium	0.001			1.80E-03	1.03E-06	118	0.000	0.009

CLEVELAND, 2000-2009
Cuyahoga County
FIRE "4B"
3136 Lorain Ave.
AIRS: 39-035-0042

Compound	Average µg/m³	RBC	HI	UR	RISK	Frequency	Min µg/m³	Max µg/m³
beryllium	0.000	0.02	0.00	2.40E-03	6.68E-08	40	0.000	0.000
chromium	0.003	0.10	0.03	1.20E-02	3.81E-05	56	0.001	0.007
lead	0.016	1.50	0.01	1.20E-05	1.95E-07	71	0.004	0.049
manganese	0.019	0.05	0.38			69	0.003	0.058
nickel	0.007	0.20	0.03	2.40E-04	1.58E-06	56	0.001	0.068
zinc	0.057					69	0.019	0.150
arsenic	0.001			4.30E-03	5.42E-06	68	0.001	0.004
cadmium	0.001			1.80E-03	9.24E-07	69	0.000	0.007

A2-12. Cuyahoga County, IMT (2000-2003)

CLEVELAND, 2000-2003
Cuyahoga County
IMT
511 West 164th
AIRS: 39-035-1003

Compound	Average µg/m³	RBC	HI	UR	RISK	Frequency	Min µg/m³	Max µg/m³
beryllium	0.000	0.02	0.002	2.40E-03	9.20E-08	36	0.000	0.000
chromium	0.004	0.10	0.041	1.20E-02	4.98E-05	36	0.003	0.007
lead	0.015	1.50	0.010	1.20E-05	1.86E-07	36	0.003	0.036
manganese	0.047	0.05	0.950			36	0.016	0.126
nickel	0.005	0.20	0.027	2.40E-04	1.32E-06	36	0.004	0.009
zinc	0.116					36	0.032	0.439
arsenic	0.002			4.30E-03	7.75E-06	36	0.001	0.005
cadmium	0.001			1.80E-03	1.09E-06	36	0.000	0.002

A2-13. Cuyahoga County, Gate A (2001-2003)

CLEVELAND, 2001-2003
Cuyahoga County
Gate "A"
2850 3rd St.
AIRS: 39-035-0063

Compound	Average µg/m³	RBC	HI	UR	RISK	Frequency	Min µg/m³	Max µg/m³
beryllium	0.000	0.02	0.00	2.40E-03	2.10E-07	27	0.000	0.000
chromium	0.004	0.10	0.04	1.20E-02	5.07E-05	27	0.000	0.008
lead	0.078	1.50	0.05	1.20E-05	9.33E-07	27	0.010	0.380
manganese	0.060	0.05	1.19			27	0.014	0.180
nickel	0.005	0.20	0.02	2.40E-04	1.10E-06	27	0.000	0.006
zinc	0.114					27	0.032	0.260
arsenic	0.002			4.30E-03	7.67E-06	27	0.001	0.004
cadmium	0.001			1.80E-03	1.77E-06	27	0.000	0.004

A2-14. Cuyahoga County, Fire Station #11 (2004-2006)

CLEVELAND, 2004-2006
Cuyahoga County
Fire Station #11
7629 Broadway Ave.
AIRS: 39-035-0068

Compound	Average µg/m³	RBC	HI	UR	RISK	Frequency	Min µg/m³	Max µg/m³
beryllium	0.000	0.02	0.00	2.40E-03	7.32E-08	8	0.000	0.000
chromium	0.004	0.10	0.04	1.20E-02	4.76E-05	3	0.004	0.008
lead	0.023	1.50	0.02	1.20E-05	2.77E-07	34	0.007	0.052
manganese	0.033	0.05	0.67			35	0.009	0.070
nickel	0.007	0.20	0.03	2.40E-04	1.58E-06	8	0.005	0.020
zinc	0.083					36	0.024	0.180
arsenic	0.002			4.30E-03	8.22E-06	35	0.001	0.006
cadmium	0.001			1.80E-03	1.38E-06	36	0.000	0.003

A2-15. Cuyahoga County, Fire Station #22 (2004-2006)

CLEVELAND, 2004-2006
Cuyahoga County
Fire Station #22
7300 Superior Ave.
AIRS: 39-035-0069

Compound	Average µg/m³	RBC	HI	UR	RISK	Frequency	Min µg/m³	Max µg/m³
beryllium	0.000	0.02	0.0012	2.40E-03	5.76E-08	1	0.000	0.000
lead	0.016	1.50	0.010636	1.20E-05	1.91E-07	28	0.007	0.047
manganese	0.022	0.05	0.431429			35	0.008	0.040
zinc	0.063					35	0.032	0.130
arsenic	0.002			4.30E-03	7.75E-06	35	0.001	0.005
cadmium	0.001			1.80E-03	9.04E-07	35	0.000	0.001

A2-16. Franklin County, Ann/Woodrow (2000-2009)

COLUMBUS, 2000-2009								
Franklin County								
E. Woodrow Ave./Ann St.								
AIRS: 39-049-0025								
Compound	**Average µg/m³**	**RBC**	**HI**	**UR**	**RISK**	**Frequency**	**Min µg/m³**	**Max µg/m³**
beryllium	0.000	0.02	0.00	2.40E-03	4.25E-08	48	0.000	0.000
chromium	0.002	0.10	0.02	1.00E-02	2.40E-05	71	0.000	0.006
lead	0.034	1.50	0.02	1.20E-05	4.03E-07	115	0.004	0.300
manganese	0.014	0.05	0.29			120	0.004	0.130
nickel	0.003	0.20	0.01	2.40E-04	7.17E-07	70	0.000	0.007
zinc	0.063					120	0.024	0.200
arsenic	0.002			4.30E-03	8.64E-06	119	0.001	0.014
cadmium	0.000			1.80E-03	7.21E-07	120	0.000	0.002
mercury	0.000					18	0.000	0.001
iron	0.044					24	0.080	0.400

A2-17. Fulton County, Van Buren St. (2000-2009)

NWDO HEAVY METALS DATA, 2000-2009								
Delta, 200 Van Buren St.								
AIRS: 39-051-0001, units -- µg/m³								
Fulton County, Parameters								
Compound	**Average µg/m³**	**RBC**	**HAZARD INDEX**	**UR**	**RISK**	**Frequency**	**Min µg/m³**	**Max µg/m³**
beryllium	0.000	0.02	0.00	2.40E-03	6.84E-08	96	0.000	0.000
chromium	0.003	0.10	0.03	1.00E-02	2.53E-05	69	0.001	0.016
lead	0.175	1.50	0.12	1.20E-05	2.10E-06	130	0.006	1.100
manganese	0.010	0.05	0.20			130	0.002	0.032
nickel	0.004	0.20	0.02	2.40E-04	9.52E-07	78	0.000	0.130
zinc	1.042					130	0.016	17.000
arsenic	0.001			4.30E-03	4.04E-06	125	0.000	0.003
cadmium	0.002			1.80E-03	2.91E-06	130	0.000	0.019

A2-18. Logan County, Bellefontaine (2000-2009)

SWDO HEAVY METALS DATA, 2000-2009								
Bellefontaine								
1222 Superior Ave.								
AIRS: 39-091-0003, units -- µg/m³								
Logan County, Parameters								
Compound	**Average µg/m³**	**RBC**	**HAZARD INDEX**	**UR**	**RISK**	**Frequency**	**Min µg/m³**	**Max µg/m³**
beryllium	0.000	0.02	0.00	2.40E-03	5.30E-08	45	0.000	0.000
chromium	0.003	0.10	0.03	1.00E-02	2.52E-05	89	0.001	0.021
lead	0.067	1.50	0.04	1.20E-05	8.08E-07	142	0.000	0.368
manganese	0.005	0.05	0.09			140	0.000	0.014
nickel	0.003	0.20	0.02	2.40E-04	7.91E-07	137	0.001	0.017
zinc	0.022					141	0.011	0.063
arsenic	0.001			4.30E-03	3.83E-06	139	0.000	0.008
cadmium	0.000			1.80E-03	3.11E-07	139	0.000	0.003
iron	0.054					66	0.048	0.510

A2-19. Marion County, Marion Steel (2000-2009)

NWDO HEAVY METALS DATA, 2000-2009								
Marion Steel								
635 Bellfontaine / Gill Ave. RIGHT								
designated, units -- µg/m³								
Marion County, Parameters								
Compound	**Average µg/m³**	**RBC**	**HAZARD INDEX**	**UR**	**RISK**	**Frequency**	**Min µg/m³**	**Max µg/m³**
beryllium	0.000	0.02	0.00	2.40E-03	4.87E-08	27	0.000	0.000
chromium	0.004	0.10	0.04	1.00E-02	3.79E-05	81	0.000	0.012
lead	0.034	1.50	0.02	1.20E-05	4.05E-07	85	0.000	0.430
manganese	0.054	0.05	1.07			85	0.000	0.190
nickel	0.004	0.20	0.02	2.40E-04	8.98E-07	26	0.000	0.011
zinc	0.180					85	0.000	3.200
arsenic	0.001			4.30E-03	5.27E-06	85	0.000	0.004
cadmium	0.001			1.80E-03	1.16E-06	85	0.000	0.010
mercury	0.000	0.30	0.00			86	0.000	0.003

NWDO HEAVY METALS DATA, 2000-2009								
Marion Steel								
635 Bellfontaine / Gill Ave. LEFT								
Left/ co-located, units - µg/m³								
Marion County, Parameters								
Compound	**Average µg/m³**	**RBC**	**HAZARD INDEX**	**UR**	**RISK**	**Frequency**	**Min µg/m³**	**Max µg/m³**
beryllium	0.000	0.02	0.00	2.40E-03	5.44E-08	26	0.000	0.000
chromium	0.004	0.10	0.04	1.00E-02	3.88E-05	66	0.000	0.011
lead	0.024	1.50	0.02	1.20E-05	2.87E-07	73	0.000	0.140
manganese	0.049	0.05	0.99			73	0.000	0.200
nickel	0.004	0.20	0.02	2.40E-04	1.06E-06	26	0.000	0.013
zinc	0.125					73	0.000	0.550
arsenic	0.001			4.30E-03	5.08E-06	72	0.000	0.003
cadmium	0.000			1.80E-03	8.29E-07	73	0.000	0.003

A2-20. Marion County, Whitmore (2000-2009)

NWDO HEAVY METALS DATA, 2002-2009								
Marion Steel								
441 Whitmore St. LEFT								
AIRS: units - µg/m³								
Parameters								
Compound	Average µg/m³	RBC	HAZARD INDEX	UR	RISK	Frequency	Min µg/m³	Max µg/m³
beryllium	0.000	0.02	0.00	2.40E-03	6.55E-08	33	0.000	0.000
chromium	0.007	0.10	0.07	1.00E-02	6.95E-05	72	0.000	0.042
lead	0.076	1.50	0.05	1.20E-05	9.08E-07	126	0.006	0.670
manganese	0.152	0.05	3.03			139	0.007	1.200
nickel	0.004	0.20	0.02	2.40E-04	9.43E-07	40	0.001	0.015
zinc	0.460					139	0.018	9.400
arsenic	0.016			4.30E-03	6.68E-05	136	0.001	0.340
cadmium	0.001			1.80E-03	2.10E-06	138	0.000	0.010
mercury	0.000	0.30	0.00			137	0.000	0.006

NWDO HEAVY METALS DATA, 2002-2009								
Marion Steel								
441 Whitmore St. RIGHT								
AIRS: units - µg/m³								
Parameters								
Compound	Average µg/m³	RBC	HAZARD INDEX	UR	RISK	Frequency	Min µg/m³	Max µg/m³
beryllium	0.000	0.02	0.00	2.40E-03	6.18E-08	41	0.000	0.000
chromium	0.007	0.10	0.07	1.00E-02	6.82E-05	89	0.000	0.047
lead	0.081	1.50	0.05	1.20E-05	9.68E-07	135	0.003	0.690
manganese	0.156	0.05	3.11			152	0.006	1.600
nickel	0.004	0.20	0.02	2.40E-04	9.64E-07	56	0.001	0.031
zinc	0.427					152	0.018	3.200
arsenic	0.015			4.30E-03	6.59E-05	149	0.001	0.390
cadmium	0.001			1.80E-03	2.12E-06	152	0.000	0.009
mercury	0.000	0.30	0.00			149	0.000	0.004

A2-21. Ottawa County, Brush Wellman (2000-2009)

NWDO HEAVY METALS DATA, 2002-2009								
Brush Wellman 32								
AIRS: units - µg/m³								
Parameters								
Compounds	Average µg/m³	RBC	HAZARD INDEX	UR	RISK	Frequency	Min µg/m³	Max µg/m³
beryllium	0.000	0.02	0.01	2.40E-03	7.13E-07	67	0.000	0.013
chromium	0.000	0.10	0.00	1.00E-02	3.18E-06	30	0.000	0.001
lead	0.003	1.50	0.00	1.20E-05	3.77E-08	67	0.001	0.007
manganese	0.004	0.05	0.08			67	0.001	0.022
nickel	0.001	0.20	0.00	2.40E-04	1.58E-07	66	0.000	0.002
zinc	0.013					67	0.005	0.027
arsenic	0.001			4.30E-03	2.40E-06	67	0.000	0.001
cadmium	0.000			1.80E-03	1.93E-07	67	0.000	0.000

A2-22. Washington County, Career Center (2000-2009)

Washington County HEAVY METALS DATA, 2000-2009								
Rt. 676 Marietta, Career Center								
AIRS: 39-167-0008								
Washinton County								
Compounds	Average µg/m³	RBC	HAZARD INDEX	UR	RISK	Frequency	Min µg/m³	Max µg/m³
beryllium	0.000	0.02	0.00	2.40E-03	4.75E-08	50	0.000	0.000
chromium	0.002	0.10	0.02	1.00E-02	2.22E-05	70	0.000	0.009
lead	0.006	1.50	0.00	1.20E-05	6.92E-08	108	0.000	0.056
manganese	0.145	0.05	2.90			108	0.007	1.400
nickel	0.003	0.20	0.01	2.40E-04	6.15E-07	81	0.000	0.012
zinc	0.026					108	0.002	0.110
arsenic	0.001			4.30E-03	4.69E-06	106	0.000	0.003
cadmium	0.001			1.80E-03	1.81E-06	108	0.000	0.014
mercury	0.000	0.30	0.00			44	0.000	0.000

APPENDIX B1, OHIO YEARLY AVERAGES

VOLATILE ORGANIC COMPOUNDS

B-1-1. Butler County (2000-2009)

Urban Air Monitoring: VOC Sampling										
Annual Average Concentrations						**2000-2009**				
Location:	**HAMILTON**									
	Butler County									
	Verity School									
	1900 St. John's Rd.									
	Middletown									
AIRS #:	**39-017-0003**									
Sampler:	**Xon - J23398**									
Concentrations:	$\mu g/m^3$									
Compounds	**2000**	**2001**	**2002**	**2003**	**2004**	**2005**	**2006**	**2007**	**2008**	**2009**
acetone	7.49	8.10	5.39	4.52	13.38	11.84	14.25	9.76	8.79	9.26
acetonitrile	0.36	0.19	0.19	ND	0.42	0.73	0.59	0.68	0.21	0.24
acrylonitrile	ND	0.27	ND	ND	0.58	0.60	0.30	0.82	0.25	0.24
benzene	0.72	0.91	1.01	0.88	1.11	0.88	1.05	0.83	0.66	0.76
1,3-butadiene	0.25	0.19	0.14	0.13	0.13	ND	0.14	0.45	ND	ND
n-butane	3.55	3.83	2.55	2.49	2.96	2.77	2.87	2.84	2.04	2.64
2-butanone	1.32	0.50	0.58	0.84	2.36	2.12	1.99	2.66	1.25	1.33
carbon disulfide	0.88	1.60	1.49	0.82	0.86	ND	1.00	ND	ND	ND
carbon tetrachloride	0.46	0.44	0.59	0.37	0.60	0.48	0.54	0.70	ND	ND
chlorodifluoromethane	0.78	1.09	0.73	0.81	1.06	1.09	1.36	1.73	1.45	1.50
chloroethane	ND	0.15	ND	ND	ND	ND	ND	ND	ND	ND
chloroform	ND	ND	ND	ND	ND	0.25	0.28	0.59	ND	ND
chloromethane / methyl chloride	0.84	1.18	1.29	0.93	0.88	0.93	1.09	1.10	1.17	ND
3-chloropropene	ND	ND	0.25	ND	ND	ND	ND	ND	ND	ND
cyclohexane	0.24	0.20	ND	ND	0.19	ND	ND	ND	ND	ND
decane	1.03	0.36	0.30	ND	0.33	ND	0.31	ND	ND	ND
dibromochloromethane	ND	0.62	ND	ND	ND	ND	ND	ND	ND	ND
1,4-dichlorobenzene (para)	ND	0.37	ND	0.32	ND	ND	0.36	0.80	0.54	ND
dichlorodifluoromethane	2.28	3.10	3.24	2.30	2.47	2.44	2.74	2.63	2.64	2.98
trans-1,2-dichloroethene	ND	ND	ND	ND	ND	ND	ND	1.03	ND	ND
n-dodecane	ND	0.59	ND	ND	ND	ND	ND	ND	ND	ND
ethanol	7.47	ND	ND	ND	ND	ND	ND	ND	ND	ND
ethylbenzene	0.44	0.30	0.26	ND	0.29	ND	ND	0.63	ND	ND
4-ethyltoluene	0.73	ND	ND	ND	0.25	0.26	0.26	ND	ND	ND
n-heptane	0.61	0.22	ND	ND	0.28	0.22	0.23	0.80	ND	ND
hexane	0.64	0.61	0.41	0.41	0.80	0.58	0.49	1.38	0.19	0.43
methylene chloride / dichloromethane	0.48	0.23	0.21	0.21	0.26	0.23	0.24	0.82	0.17	0.50
a-methylstyrene	ND	ND	0.26	ND	ND	ND	ND	ND	ND	ND
4-methyl-2-pentanone	0.22	ND	ND	ND	0.24	0.22	0.21	ND	ND	ND
naphthalene	ND	0.29	0.34	0.27	0.27	ND	0.35	0.68	ND	ND
n-nonane	0.65	ND	0.27	ND	ND	ND	ND	1.60	ND	ND
n-octane	ND	ND	ND	0.25	ND	ND	ND	ND	ND	ND
n-pentane	1.74	1.64	1.20	1.19	1.59	1.69	1.26	1.53	0.59	1.40
propylene	0.61	0.13	0.48	0.33	1.36	1.22	1.62	1.27	0.55	0.28
toluene	1.32	1.99	1.33	0.95	1.78	0.76	1.30	1.34	0.38	1.21
1,1,1-trichloroethane	1.02	1.29	1.33	0.94	0.55	0.53	0.74	0.82	ND	ND
trichloroethene	ND	ND	ND	ND	0.32	ND	ND	2.93	ND	ND
trichlorofluoromethane	25.34	54.86	29.05	2.19	2.99	2.29	2.63	2.93	0.89	1.77
1,1,2-trichloro-1,2,2-trifluoroethane	0.77	0.40	0.52	ND	0.49	ND	0.46	0.84	ND	ND
1,2,4-trimethylbenzene	0.48	0.45	0.32	0.29	0.36	0.27	0.41	0.96	ND	ND
1,3,5-trimethylbenzene	0.28	ND	ND	ND	0.26	ND	ND	ND	ND	ND
n-undecane	1.65	0.54	ND	ND	0.35	ND	0.33	ND	ND	ND
vinyl acetate	ND	ND	0.51	0.37	1.49	1.30	1.66	2.55	0.54	1.32
o-xylene	0.45	0.31	0.27	0.24	0.31	0.25	0.28	0.72	ND	ND
total m+p-xylene	0.94	0.66	0.45	0.34	0.58	0.35	0.55	1.98	ND	0.91

B-1-2. Coshocton County (2000-2002)

Urban Air Monitoring: VOC Sampling **2000-2002**
Annual Average Concentrations

Location: SEDO
Coshocton County
Coshocton Co. Health Dept.
724 South 7th Street
Coshocton
AIRS#: N/A
Sampler: Xon - J23388
Concentrations: µg/m3

Compounds	2000	2001	2002
acetone	6.21	7.36	6.08
acetonitrile	0.22	0.18	ND
acrylonitrile	0.36	0.24	ND
benzene	0.71	0.92	2.53
1,3-butadiene	0.14	0.21	0.35
n-butane	5.45	7.03	12.56
2-butanone	1.03	0.49	0.55
carbon disulfide	5.85	1.77	2.25
carbon tetrachloride	0.79	0.39	0.71
chlorodifluoromethane	0.65	1.61	0.83
chloroethane	0.18	0.15	ND
chloromethane	1.01	0.99	1.53
cyclohexane	0.19	0.19	0.39
decane	0.39	0.32	0.40
1,3-dichlorobenzene (meta)	0.35	ND	ND
1,4-dichlorobenzene (para)	0.35	0.33	ND
dichlorodifluoromethane	2.48	2.56	3.79
1,1-dichloroethene	ND	ND	ND
ethanol	1.02	ND	ND
ethylbenzene	0.32	0.34	0.62
4-ethyltoluene	ND	ND	0.32
n-heptane	0.41	0.40	0.89
hexane	1.02	1.00	1.27
methylene chloride	1.13	0.23	0.34
4-methyl-2-pentanone	ND	ND	0.27
a-methylstyrene	0.34	0.25	0.34
naphthalene	0.28	1.18	ND
n-nonane	0.43	0.39	0.39
n-octane	0.52	0.37	0.65
n-pentane	2.39	2.95	4.34
propylene	0.21	0.20	2.85
styrene	ND	ND	0.27
tetrachloroethylene	1.40	0.75	ND
toluene	2.46	3.50	6.25
1,2,4-trichlorobenzene	0.52	ND	ND
1,1,1-trichloroethane	6.97	3.27	1.27
trichlorofluoromethane	8.44	12.59	10.01
1,1,2-trichloro-1,2,2-trifluoroethane	0.40	ND	0.50
1,2,4-trimethylbenzene	0.35	0.55	0.81
1,3,5-trimethylbenzene	0.26	ND	0.35
n-undecane	0.33	ND	0.38
vinyl acetate	0.38	0.36	ND
o-xylene	0.36	0.40	0.63
total m+p-xylene	1.09	1.17	2.23

B1-3. Cuyahoga County, G. T. Craig (2000-2003)

Urban Air Monitoring: VOC Sampling				**2000-2003**
Annual Average Concentrations				
Location:	**CLEVELAND**			
	Cuyahoga County			
	G.T. Craig			
	E. 14th & Orange			
AIRS #:	**39-035-0060**			
Sampler:	**Xon. 911-J23460**			
Concentrations:		μg/m³		
Compounds	**2000**	**2001**	**2002**	**2003**
acetone	10.47	12.42	9.30	4.56
acetonitrile	0.76	0.96	1.03	ND
acrylonitrile	0.63	0.77	0.75	ND
benzene	1.08	1.02	1.30	1.12
bromomethane	ND	ND	ND	0.78
1,3-butadiene	0.36	0.28	0.50	0.29
n-butane	9.14	9.94	15.52	8.48
2-butanone	1.51	2.02	2.57	2.25
carbon disulfide	3.69	3.36	2.15	1.82
carbon tetrachloride	0.63	0.70	0.81	0.65
chlorodifluoromethane	0.84	0.87	1.12	0.86
chloroethane	ND	0.40	0.37	ND
chloromethane	1.11	1.15	1.23	1.00
cyclohexane	0.76	ND	0.34	0.34
decane	0.78	ND	ND	1.11
1,4-dichlorobenzene (para)	ND	1.92	0.78	ND
dichlorodifluoromethane	2.49	3.05	3.10	2.50
ethanol	4.90	ND	ND	ND
ethylbenzene	1.13	0.77	0.76	0.86
n-heptane	1.17	0.78	0.83	0.61
hexane	1.85	1.33	1.50	1.39
methyl-butyl ether	1.84	ND	ND	ND
methylene chloride	1.61	0.72	0.86	0.79
4-methyl-2-pentanone	0.70	0.61	ND	2.62
naphthalene	0.60	ND	0.63	0.94
n-nonane	0.60	0.68	ND	0.94
n-octane	0.68	ND	ND	0.61
n-pentane	4.52	3.52	4.07	3.84
propylene	1.07	1.17	3.45	1.92
styrene	0.51	ND	ND	ND
tetrachloroethylene	0.98	1.15	0.79	ND
toluene	3.22	2.35	2.52	2.16
1,1,1-trichloroethane	0.64	0.89	0.74	0.64
trichloroethene	0.91	ND	0.86	ND
trichlorofluoromethane	1.63	1.59	2.08	1.33
1,1,2-trichloro-1,2,2-trifluoroethane	ND	ND	0.99	ND
1,2,4-trimethylbenzene	1.00	0.67	0.94	0.82
1,3,5-trimethylbenzene	ND	ND	0.54	ND
n-uNDecane	0.70	ND	ND	ND
vinyl acetate	0.70	1.46	1.55	ND
o-xylene	0.92	0.70	0.83	0.98
total m+p-xylene	3.46	1.62	1.89	2.42

B1-4. Cuyahoga County, Fire Station #11 (2004-2009)

Urban Air Monitoring: VOC Sampling			**2004-2009**			
Annual Average Concentrations						
Location:	**CLEVELAND**					
	Cuyhoga County					
	Fire Station #11					
	7629 Broadway Ave.					
AIRS#:	**39-035-0068**					
Sampler:	**1201-651**					
Concentrations:			µg/m^3			
Compounds	**2004**	**2005**	**2006**	**2007**	**2008**	**2009**
acetone	13.04	13.07	16.55	7.19	7.36	9.73
acetonitrile	0.49	1.12	0.88	0.33	0.20	0.41
acrylonitrile	ND	ND	0.27	0.22	ND	ND
benzene	1.66	1.24	1.75	0.78	0.85	0.72
bromodichloromethane	0.35	0.35	ND	ND	ND	ND
bromomethane	ND	0.21	ND	ND	ND	ND
1,3-butadiene	0.22	0.13	0.16	0.13	ND	ND
n-butane	4.49	4.94	4.40	4.11	3.77	4.20
2-butanone	2.37	2.49	2.47	1.56	1.48	1.26
carbon disulfide	0.81	ND	0.83	ND	ND	ND
carbon tetrachloride	0.67	0.61	0.58	0.35	0.52	ND
chlorodifluoromethane	1.08	1.60	1.64	1.02	1.47	2.98
chloroethane	0.14	0.14	ND	ND	ND	ND
chloroform	0.26	0.29	ND	ND	ND	ND
chloromethane	0.94	0.97	1.19	1.01	ND	1.30
3-chloropropene	0.21	0.18	ND	ND	ND	ND
cyclohexane	0.23	0.19	0.20	0.24	ND	ND
decane	ND	0.38	0.49	0.34	0.46	ND
dibromomethane	0.37	0.37	ND	ND	ND	ND
1,4-dichlorobenzene (para)	0.31	0.44	0.34	ND	ND	ND
dichlorodifluoromethane	2.96	2.64	2.91	2.66	3.12	3.10
1,1-dichloroethane	0.21	0.22	ND	ND	ND	ND
1,2-dichloroethane	0.21	0.22	ND	ND	ND	ND
1,1-dichloroethene	0.21	0.21	ND	ND	ND	ND
cis-1,2-dichloroethene	0.21	0.21	ND	ND	ND	ND
trans-1,2-dichloroethene	0.21	0.21	ND	ND	ND	ND
1,2-dichloropropane	ND	0.24	ND	ND	ND	ND
1,2-dichloro-1,1,2,2-tetrafluoro	ND	0.37	ND	ND	ND	ND
ethylbenzene	0.37	0.39	0.52	0.32	0.43	0.43
4-ethyltoluene	0.27	0.28	0.26	0.27	0.37	0.49
n-heptane	0.50	0.43	0.60	0.41	0.56	0.41
hexachlorobutadiene	0.56	ND	ND	ND	ND	1.07
hexane	1.32	1.36	1.15	0.82	0.86	0.66
methylene chloride	1.08	1.74	2.68	1.16	1.93	1.15
4-methyl-2-pentanone	0.43	0.21	0.23	0.28	0.38	ND
naphthalene	0.31	0.29	0.44	0.28	ND	ND
n-nonane	0.28	0.33	0.33	0.27	ND	ND
n-octane	ND	0.25	0.41	0.25	0.38	ND
n-pentane	2.86	3.01	2.81	2.66	2.61	2.20
propylene	2.50	2.18	2.74	0.98	1.05	ND
n-propyl benzene	ND	0.27	ND	ND	ND	ND
styrene	ND	0.24	0.22	ND	ND	ND
tetrachloroethylene	0.48	0.46	0.46	0.35	0.67	ND
toluene	3.12	2.47	4.08	2.42	2.71	1.71
1,1,1-trichloroethane	0.29	0.29	ND	ND	ND	ND
1,1,2-trichloroethane	ND	0.28	ND	ND	ND	ND
trichloroethene	0.52	0.35	0.30	0.28	ND	ND
trichlorofluoromethane	2.25	1.88	2.31	1.87	1.64	2.13
1,1,2-trichloro-1,2,2-trifluoroeth	0.51	0.47	0.49	0.48	0.62	ND
1,2,4-trimethylbenzene	0.49	0.54	0.89	0.39	0.49	ND
1,3,5-trimethylbenzene	0.27	0.31	0.27	ND	ND	ND
n-uNDecane	ND	0.36	0.34	0.32	0.50	ND
vinyl acetate	ND	0.46	1.01	0.88	0.83	0.85
vinyl chloride	ND	0.13	ND	ND	ND	ND
o-xylene	0.38	0.43	0.57	0.33	0.39	ND
total m+p-xylene	1.18	1.18	1.71	0.93	1.08	0.92

B1-5. Cuyahoga County, Fire Station #22 (2004-2009)

Urban Air Monitoring: VOC Sampling					**2004-2009**	
Annual Average Concentrations						
Location:	**CLEVELAND**					
	Cuyahoga County					
	Fire Station #22					
	7300 Superior Ave.					
	Cleveland					
AIRS #:	**39-035-0069**					
Sampler:	**302-652**					
Concentrations:			μg/m^3			
Compounds	**2004**	**2005**	**2006**	**2007**	**2008**	**2009**
acetone	12.17	15.14	15.17	9.49	10.52	16.44
acetonitrile	0.46	1.03	0.57	0.27	0.36	0.33
acrylonitrile	0.27	ND	ND	ND	0.32	0.42
benzene	1.84	1.92	1.81	1.20	0.99	1.35
1,3-butadiene	0.23	0.16	ND	0.18	0.25	ND
n-butane	5.28	6.36	6.52	7.34	5.51	5.86
2-butanone	1.79	2.62	1.80	1.89	1.55	1.11
carbon disulfide	0.85	ND	0.84	ND	0.82	ND
carbon tetrachloride	0.63	0.57	0.52	0.38	0.56	ND
chlorobenzene	ND	ND	0.27	ND	ND	ND
chlorodifluoromethane	1.07	2.70	1.44	1.25	1.49	1.1
chloroethane	ND	0.14	ND	ND	ND	ND
chloroform	ND	0.26	0.34	ND	ND	ND
chloromethane	0.99	0.98	1.14	1.10	1.20	1.3
cyclohexane	0.23	ND	0.25	0.25	0.34	ND
decane	0.33	0.37	0.50	0.35	0.50	ND
1,4-dichlorobenzene (para)	0.34	0.32	0.38	0.32	0.50	ND
dichlorodifluoromethane	2.74	2.56	2.67	2.66	2.85	2.96
1,1-dichloroethane	ND	0.21	ND	ND	ND	ND
1,2-dichloroethane	ND	0.21	ND	ND	ND	ND
1,1-dichloroethene	ND	0.21	ND	ND	ND	ND
cis-1,2-dichloroethene	ND	0.22	ND	ND	ND	ND
trans-1,2-dichloroethene	ND	0.21	ND	ND	ND	ND
1,2-dichloro-1,1,2,2-tetrafluoro	ND	0.36	ND	ND	ND	ND
ethylbenzene	0.37	0.48	0.46	0.42	0.45	ND
4-ethyltoluene	0.28	0.30	ND	0.28	0.42	ND
n-heptane	0.46	0.55	0.57	0.51	0.50	ND
hexane	1.65	1.65	2.16	1.73	0.94	0.74
methylene chloride	0.45	0.45	1.70	0.41	0.50	0.55
4-methyl-2-pentanone	0.31	0.22	0.23	0.24	2.44	ND
naphthalene	0.31	0.27	0.36	0.31	0.66	ND
n-nonane	0.29	0.31	0.29	0.29	0.44	ND
n-octane	ND	0.27	0.28	0.26	0.39	ND
n-pentane	3.48	3.72	4.64	3.65	3.31	2.73
propylene	2.32	1.86	2.36	1.13	1.15	ND
n-propyl benzene	0.27	0.26	ND	0.26	ND	ND
tetrachloroethylene	0.41	0.53	0.40	0.44	0.65	ND
toluene	4.45	3.21	4.03	4.44	2.61	1.55
1,1,1-trichloroethane	ND	0.30	ND	ND	ND	ND
trichloroethene	0.42	0.29	0.34	0.31	0.46	ND
trichlorofluoromethane	1.80	1.89	1.64	1.71	1.45	1.62
1,1,2-trichloro-1,2,2-trifluoroet	0.51	0.45	0.44	0.40	0.65	ND
1,2,4-trimethylbenzene	0.49	0.68	0.82	0.46	0.53	ND
1,3,5-trimethylbenzene	0.28	0.36	0.27	0.27	0.41	ND
n-undecane	ND	0.34	0.41	ND	0.66	ND
vinyl acetate	ND	0.43	0.60	1.51	0.79	0.69
o-xylene	0.40	0.54	0.55	0.43	0.45	ND
total m+p-xylene	1.19	1.57	2.09	1.25	1.17	ND

B1-6. Cuyahoga County, Tri-C College (2006-2008)

			2006-2008
Annual Average Concentrations			
Location:	**CLEVELAND**		
	Cuyahoga Co.		
	Tri-C College		
	2900 Community College Ave.		
	Cleveland		
AIRS #:	**39-035-0071**		
Sampler:	**AVOCS-0102-652**		
Concentrations:	µg/m³		
Compounds	**2006**	**2007**	**2008**
acetone	32.26	192.84	37.94
acetonitrile	0.59	0.22	0.94
acrylonitrile	**ND**	**ND**	0.30
benzene	1.82	1.08	1.42
1,3-butadiene	0.22	0.12	0.50
n-butane	7.07	4.12	13.94
2-butanone	10.10	12.07	3.71
carbon disulfide	**ND**	**ND**	0.92
carbon tetrachloride	0.65	0.42	0.55
chlorodifluoromethane	2.14	1.06	2.04
chloroethane	**ND**	0.15	**ND**
chloromethane	1.40	1.21	1.37
cyclohexane	0.20	0.20	0.90
decane	1.05	0.63	1.37
1,4-dichlorobenzene (para)	2.27	0.40	1.76
dichlorodifluoromethane	3.00	2.55	4.27
1,2-dichloroethane	**ND**	**ND**	0.51
ethylbenzene	0.50	0.30	1.09
4-ethyltoluene	**ND**	0.28	0.44
n-heptane	0.66	0.58	1.14
hexane	1.35	0.90	1.92
methyl-butyl ether	**ND**	**ND**	0.38
methylene chloride	0.74	0.58	0.66
4-methyl-2-pentanone	0.88	1.50	2.24
a-methylstyrene	0.27	**ND**	**ND**
naphthalene	0.42	0.29	0.81
n-nonane	0.34	0.34	0.51
n-octane	0.34	0.31	0.40
n-pentane	5.03	3.82	15.01
propylene	2.33	6.74	2.83
n-propyl benzene	**ND**	**ND**	0.45
styrene	**ND**	**ND**	0.41
tetrachloroethylene	0.41	0.38	0.65
toluene	4.91	2.63	6.51
trichloroethene	0.47	0.32	1.01
trichlorofluoromethane	1.98	1.75	1.98
1,1,2-trichloro-1,2,2-trifluoro	0.76	0.49	0.67
1,2,4-trimethylbenzene	0.92	0.54	0.56
1,3,5-trimethylbenzene	**ND**	0.28	0.43
n-undecane	0.67	0.45	2.12
vinyl acetate	0.55	2.93	1.32
o-xylene	0.52	0.31	1.04
total m+p-xylene	1.53	0.96	3.37

B1-7. Cuyahoga County, St. Theodosius (2006-2009)

Urban Air Monitoring: VOC Sampling			**2006-2009**	
Annual Average Concentrations				
Location:	**CLEVELAND**			
	Cuyahoga Co.			
	St. Theodosious			
	2547 St. Tikon			
	Cleveland			
AIRS #:	**39-035-0038**			
Sampler:				
Concentrations:	μg/m^3			
Compounds	**2006**	**2007**	**2008**	**2009**
acetone	20.03	8.26	6.04	23.67
acetonitrile	0.42	0.33	0.18	0.76
acrylonitrile	0.32	0.23	**ND**	0.25
benzene	1.22	0.75	0.63	0.70
1,3-butadiene	**ND**	0.12	**ND**	0.27
n-butane	4.57	5.00	4.64	5.65
2-butanone	1.48	1.57	1.18	1.85
carbon tetrachloride	0.59	0.35	0.49	**ND**
chlorodifluoromethane	1.24	0.58	1.38	1.06
chloromethane	1.37	1.10	1.09	1.35
cumene	**ND**	**ND**	**ND**	0.52
decane	0.80	0.40	0.58	25.20
1,4-dichlorobenzene (para)	0.53	**ND**	**ND**	**ND**
dichlorodifluoromethane	2.83	2.43	2.73	2.87
n-dodecane	0.47	**ND**	**ND**	**ND**
ethylbenzene	0.35	0.39	0.35	0.59
4-ethyltoluene	**ND**	**ND**	**ND**	1.18
n-heptane	1.79	0.37	0.32	1.46
hexane	0.95	0.90	0.67	1.63
methylene chloride	0.98	0.54	0.63	1.06
4-methyl-2-pentanone	0.23	**ND**	**ND**	1.18
naphthalene	0.35	**ND**	**ND**	**ND**
n-nonane	0.39	0.28	0.51	22.36
n-octane	**ND**	**ND**	**ND**	1.83
n-pentane	2.70	2.82	2.67	2.63
n-propylbenzene	**ND**	**ND**	**ND**	1.25
propylene	1.71	0.88	0.98	**ND**
toluene	4.23	1.74	1.23	3.32
1,1,1-trichloroethane	**ND**	0.30	**ND**	**ND**
trichloroethene	**ND**	0.31	0.41	0.57
trichlorofluoromethane	8.21	4.30	1.27	1.58
1,1,2-trichloro-1,2,2-trifluoroethane	0.47	0.47	0.56	**ND**
1,2,4-trimethylbenzene	0.66	0.28	**ND**	5.42
1,3,5-trimethylbenzene	0.28	**ND**	**ND**	1.69
n-undecane	1.04	0.35	**ND**	8.64
vinyl acetate	0.94	0.83	0.42	1.52
o-xylene	0.41	0.33	0.31	1.39
total m+p-xylene	1.07	1.25	0.66	3.25

B1-8. Franklin County (2003-2009)

Urban Air Monitoring: VOC Sampling					**2003-2009**		
Annual Average Concentrations							
Location:	Ohio EPA Central District Office						
	Franklin County						
	Ohio State Fairgrounds						
	Korbel Ave.						
	Columbus						
	39-049-0034						
AQS # :	RM ESI-EPA017587						
Concentrations:	µg/m³						
Compounds	**2003**	**2004**	**2005**	**2006**	**2007**	**2008**	**2009**
acetone	8.31	17.71	16.35	22.18	9.10	11.07	10.45
acetonitrile	0.19	0.46	0.89	0.48	0.30	0.36	0.29
acrylonitrile	0.28	0.42	0.65	0.40	0.25	0.34	0.26
benzene	1.03	1.13	0.99	1.07	0.62	0.64	0.81
bromodichloromethane	0.35	**ND**	**ND**	**ND**	**ND**	**ND**	**ND**
1,3-butadiene	0.16	0.17	0.12	0.19	0.12	0.20	**ND**
n-butane	3.51	3.16	3.53	3.25	2.76	2.87	3.66
2-butanone	1.70	3.35	2.90	2.98	2.32	2.41	1.54
carbon disulfide	0.85	0.89	**ND**	**ND**	0.98	**ND**	**ND**
carbon tetrachloride	0.45	0.60	0.40	0.49	0.35	**ND**	**ND**
chlorodifluoromethane	1.05	0.97	1.37	1.16	1.06	1.46	1.51
chloroform	0.26	**ND**	**ND**	**ND**	**ND**	**ND**	**ND**
chloromethane	1.04	0.92	0.93	1.06	1.00	1.15	1.37
cyclohexane	0.18	0.18	0.18	0.19	**ND**	**ND**	**ND**
decane	0.52	0.31	**ND**	0.31	**ND**	**ND**	**ND**
1,4-dichlorobenzene (para)	0.32	**ND**	0.33	0.37	0.32	**ND**	0.66
dichlorodifluoromethane	2.74	2.56	2.50	2.53	2.34	2.75	2.90
cis-1,2-dichloroethene	0.21	**ND**	**ND**	**ND**	**ND**	**ND**	**ND**
n-dodecane	0.43	**ND**	**ND**	**ND**	**ND**	**ND**	**ND**
ethylbenzene	0.35	0.32	0.31	0.38	0.26	**ND**	0.45
4-ethyltoluene	0.27	0.26	**ND**	**ND**	**ND**	**ND**	**ND**
n-heptane	0.28	0.28	0.28	0.30	0.21	**ND**	**ND**
hexachlorobutadiene	0.56	**ND**	**ND**	**ND**	**ND**	**ND**	**ND**
hexane	0.78	0.78	0.78	0.73	0.48	0.56	0.60
methylene chloride	0.25	0.27	0.28	0.29	0.23	0.40	0.52
4-methyl-2-pentanone	0.23	0.27	**ND**	0.23	**ND**	**ND**	**ND**
naphthalene	0.53	0.35	**ND**	0.30	0.29	**ND**	**ND**
n-nonane	0.30	0.28	**ND**	**ND**	**ND**	**ND**	**ND**
n-octane	0.25	**ND**	**ND**	0.24	**ND**	**ND**	**ND**
n-pentane	1.83	1.77	1.93	1.85	1.40	1.59	1.89
propylene	**ND**	1.91	1.69	2.19	0.67	0.59	**ND**
styrene	**ND**	0.22	0.23	**ND**	**ND**	**ND**	**ND**
tetrachloroethylene	**ND**	0.35	**ND**	0.36	**ND**	**ND**	**ND**
toluene	2.32	1.94	1.66	2.26	1.37	1.48	1.80
1,1,1-trichloroethane	0.28	**ND**	**ND**	**ND**	0.29	**ND**	**ND**
trichloroethene	0.28	**ND**	**ND**	**ND**	**ND**	**ND**	**ND**
trichlorofluoromethane	1.22	1.51	1.54	1.46	1.46	1.38	1.71
1,1,2-trichloro-1,2,2-trifluoroethane	0.40	0.45	**ND**	0.47	0.41	**ND**	**ND**
1,2,4-trimethylbenzene	0.52	0.37	0.39	0.56	0.26	**ND**	**ND**
1,3,5-trimethylbenzene	0.26	0.26	**ND**	**ND**	**ND**	**ND**	**ND**
n-undecane	0.53	**ND**	0.33	**ND**	**ND**	**ND**	**ND**
vinyl acetate	0.40	1.31	0.94	1.53	1.56	1.36	1.17
o-xylene	0.42	0.32	0.35	0.40	0.26	0.36	0.46
total m+p-xylene	1.05	0.81	0.91	1.01	0.68	0.83	1.02

B1-9. Hamilton County, Reading Pool (2000-2002)

Urban Air Monitoring: VOC Sampling			**2000-2002**
Annual Average Concentrations			
Location:	**HAMILTON**		
	Hamilton County		
	Reading Pool		
	1601 West St.		
	Reading, Cincinnati		
AIRS #:			
Sampler:			
Concentrations:	μg/m^3		
Compounds	**2000**	**2001**	**2002**
acetone	10.86	6.14	6.27
acetonitrile	0.43	0.19	**ND**
acrylonitrile	0.44	0.28	**ND**
benzene	1.36	3.88	1.58
1,3-butadiene	0.35	0.36	0.18
n-butane	8.98	42.35	5.03
2-butanone	2.03	1.20	1.48
carbon disulfide	1.57	0.95	1.82
carbon tetrachloride	**ND**	0.42	0.80
chlorodifluoromethane	0.88	1.06	0.66
chloroethane	0.39	0.15	**ND**
chloroform	0.84	0.88	0.72
chloromethane	1.38	1.66	1.81
cumene	**ND**	0.43	**ND**
cyclohexane	0.26	0.37	**ND**
decane	0.59	0.72	**ND**
1,4-dichlorobenzene (para)	0.63	0.91	**ND**
dichlorodifluoromethane	2.39	3.17	3.75
n-dodecane	**ND**	0.38	**ND**
ethanol	94.22	**ND**	**ND**
ethylbenzene	0.68	4.01	0.44
4-ethyltoluene	0.76	1.70	**ND**
n-heptane	0.65	1.62	**ND**
hexane	2.19	5.48	0.80
methylene chloride	0.85	0.79	0.48
a-methylstyrene	0.52	0.37	0.53
4-methyl-2-pentanone	**ND**	**ND**	1.14
naphthalene	1.05	1.00	0.45
n-nonane	0.28	0.81	**ND**
n-octane	0.48	1.06	**ND**
n-pentane	8.27	33.51	3.05
propylene	0.75	0.56	0.94
n-propyl benzene	0.73	1.08	**ND**
styrene	1.08	1.88	0.51
1, 1, 2, 2-tetrachloroethane	0.77	**ND**	**ND**
tetrachloroethylene	0.70	**ND**	**ND**
toluene	3.80	13.03	5.28
1,1,1-trichloroethane	0.59	**ND**	**ND**
trichlorofluoromethane	2.43	1.68	2.15
1,1,2-trichloro-1,2,2-trifluoroet	0.77	**ND**	0.61
1,2,4-trimethylbenzene	1.01	6.73	0.81
1,3,5-trimethylbenzene	0.35	2.20	0.29
n-undecane	1.26	0.47	**ND**
o-xylene	0.82	4.79	**ND**
total m+p-xylene	2.19	11.04	1.55

B1-10. Hamilton County, Cincinnati State Community College (2003-2007)

Urban Air Monitoring: VOC Sampling					**2003-2007**
Annual Average Concentrations					
Location:	**HAMILTON**				
	Hamilton County				
	Cincinnati State community College				
	10100 Reading Rd.				
	Reading, Cincinnati				
AIRS #:					
Sampler:					
Concentrations:		µg/m^3			
Compounds	**2003**	**2004**	**2005**	**2006**	**2007**
acetone	9.24	24.92	23.04	21.93	10.74
acetonitrile	0.20	0.46	1.56	0.96	0.26
acrylonitrile	**ND**	0.42	0.31	0.24	0.25
benzene	1.03	11.04	3.33	6.24	3.72
1,3-butadiene	**ND**	0.13	0.12	0.14	0.13
n-butane	2.10	2.51	2.67	2.80	2.47
2-butanone	1.63	4.05	3.87	3.27	1.94
carbon disulfide	1.03	0.85	**ND**	**ND**	**ND**
carbon tetrachloride	0.62	0.64	0.53	0.51	0.50
chlorobenzene	**ND**	0.53	0.24	0.59	0.58
chlorodifluoromethane	**ND**	0.29	2.40	1.39	0.89
chloroethane	**ND**	**ND**	0.20	**ND**	**ND**
chloroform	**ND**	**ND**	0.27	0.25	0.24
chloromethane	0.67	1.14	1.19	1.68	1.28
cyclohexane	**ND**	0.19	0.18	0.19	**ND**
decane	4.01	0.52	0.55	2.94	1.63
1,4-dichlorobenzene (para)	**ND**	**ND**	1.90	0.52	**ND**
dichlorodifluoromethane	0.67	2.46	2.44	2.70	2.60
1,1-dichloroethane	**ND**	0.21	**ND**	**ND**	**ND**
cis-1,2-dichloroethene	**ND**	0.21	**ND**	**ND**	**ND**
trans-1,2-dichloroethene	**ND**	0.21	**ND**	**ND**	**ND**
n-dodecane	0.41	**ND**	**ND**	**ND**	**ND**
ethylbenzene	**ND**	0.26	0.29	0.25	**ND**
4-ethyltoluene	0.49	0.27	**ND**	0.41	**ND**
n-heptane	**ND**	0.24	0.27	0.26	0.23
hexachlorobutadiene	**ND**	0.61	**ND**	**ND**	**ND**
hexane	0.47	0.69	0.66	0.68	0.44
methylene chloride	0.31	0.42	0.47	0.33	0.37
4-methyl-2-pentanone	**ND**	0.38	**ND**	0.24	**ND**
naphthalene	0.42	0.31	0.34	0.31	**ND**
n-nonane	1.70	0.28	0.35	2.11	0.87
n-octane	**ND**	**ND**	0.25	0.37	**ND**
n-pentane	0.93	1.39	1.73	1.62	1.12
propylene	**ND**	1.88	1.58	1.73	0.94
n-propyl benzene	0.40	**ND**	**ND**	0.34	**ND**
styrene	**ND**	0.29	0.25	0.26	**ND**
toluene	1.33	1.80	1.69	1.66	1.11
1,1,1-trichloroethane	**ND**	58.41	45.42	61.66	52.66
trichlorofluoromethane	1.50	2.08	3.09	2.73	1.97
1,1,2-trichloro-1,2,2-trifluoro	**ND**	0.53	**ND**	0.50	0.64
1,2,4-trimethylbenzene	2.91	0.42	0.47	1.79	0.75
1,3,5-trimethylbenzene	0.61	0.28	**ND**	0.53	0.28
n-undecane	2.69	0.51	0.44	1.30	0.98
vinyl acetate	**ND**	1.63	1.30	1.86	1.52
o-xylene	0.30	0.29	0.34	0.34	**ND**
total m+p-xylene	0.52	0.48	0.71	0.67	0.49

B1-11. Jefferson County (2004-2008)

Urban Air Monitoring: VOC Sampling					**2004-2009**	
Annual Average Concentrations						
Location:	**SEDO**					
	Jefferson Co.					
	Steubenville trailer					
	618 Logan Street					
	Steubenville					
AIRS #:						
Sampler:						
Concentrations:	**µg/m³**					
Compounds	**2004**	**2005**	**2006**	**2007**	**2008**	**2009**
acetone	24.03	13.10	13.83	7.83	9.60	10.53
acetonitrile	0.65	1.18	0.81	0.35	0.28	0.35
acrylonitrile	0.44	0.55	1.91	1.21	0.46	0.36
benzene	6.97	7.39	4.64	3.46	9.01	9.35
1,3-butadiene	0.18	0.14	0.20	0.14	0.20	ND
n-butane	4.53	2.78	4.14	3.08	3.81	4.37
2-butanone	2.06	2.62	1.90	1.44	1.38	1.56
carbon disulfide	0.85	0.84	1.58	2.58	1.05	0.86
carbon tetrachloride	0.62	0.59	0.46	0.34	ND	ND
chlorodifluoromethane	0.83	0.88	1.02	0.83	1.09	1.23
chloroform	ND	ND	0.25	ND	ND	ND
chloromethane	0.81	0.97	1.08	1.02	1.13	1.36
cyclohexane	0.21	ND	0.18	0.20	ND	ND
decane	ND	0.55	0.38	ND	ND	ND
1,4-dichlorobenzene (para)	ND	0.43	ND	0.34	ND	0.70
dichlorodifluoromethane	2.57	2.45	2.58	2.43	2.62	3.03
n-dodecane	ND	0.36	ND	ND	ND	ND
1,2-dichloroethane	ND	ND	ND	ND	ND	0.42
ethylbenzene	0.44	0.45	0.62	0.27	0.44	0.45
4-ethyltoluene	ND	0.26	ND	ND	ND	ND
n-heptane	0.55	0.32	0.53	0.58	0.50	0.54
hexachlorobutadiene	ND	ND	ND	ND	0.95	ND
hexane	1.23	0.66	0.83	0.61	0.68	0.56
methylene chloride	0.24	0.20	0.22	0.24	0.31	0.43
4-methyl-2-pentanone	1.35	0.71	0.89	0.40	0.36	ND
naphthalene	6.48	9.97	5.87	3.92	4.95	5.07
n-nonane	ND	0.32	ND	ND	ND	ND
n-octane	ND	0.24	ND	0.24	ND	ND
n-pentane	2.23	1.55	1.85	1.57	2.16	1.89
propylene	2.64	1.74	1.93	0.74	1.13	0.21
n-propyl benzene	ND	ND	0.27	ND	ND	ND
styrene	ND	0.33	0.27	0.24	0.45	0.45
toluene	2.94	2.13	3.01	1.93	3.65	3.24
1,1,1-trichloroethane	0.29	ND	ND	ND	ND	ND
trichloroethene	ND	ND	ND	ND	ND	0.57
trichlorofluoromethane	22.50	5.95	6.20	3.91	1.94	2.27
1,1,2-trichloro-1,2,2-trifluoroethane	0.53	0.40	0.47	0.44	ND	ND
1,2,4-trimethylbenzene	0.47	0.51	0.88	0.33	0.50	ND
1,3,5-trimethylbenzene	ND	0.28	0.27	ND	ND	ND
n-undecane	ND	0.52	ND	ND	ND	ND
vinyl acetate	1.47	1.57	1.62	1.67	1.34	1.62
o-xylene	0.52	0.46	0.65	0.28	0.46	0.46
total m+p-xylene	1.90	1.58	2.16	0.82	1.33	1.14

B1-12. Lake County (2000-2004)

Urban Air Monitoring: VOC Sampling					**2000-2004**
Annual Average Concentrations					
Location:	**PAINESVILLE**				
	Lake County				
	Lake County Hospital				
	71 E. High St				
AIRS #:	**39-085-3002**				
Sampler:	**Xontech-J23462**				
Concentrations:	**µg/m³**				
Compounds	**2000**	**2001**	**2002**	**2003**	**2004**
acetone	8.12	10.79	6.53	6.99	13.50
acetonitrile	0.37	0.25	0.21	0.21	0.46
acrylonitrile	0.49	0.23	0.28	0.27	0.78
benzene	0.61	0.63	0.88	0.80	0.83
1,3-butadiene	0.31	0.12	ND	ND	0.11
n-butane	2.51	3.47	3.38	2.58	2.44
2-butanone	1.36	0.36	0.65	1.20	1.61
carbon disulfide	0.89	1.40	1.06	0.88	ND
carbon tetrachloride	0.44	0.40	0.59	0.42	0.60
chlorodifluoromethane	0.73	0.92	2.15	69.58	6.61
chloroethane	ND	0.16	ND	ND	ND
chloroform	ND	ND	ND	0.27	0.25
chloromethane	1.08	1.10	1.28	0.95	0.91
3-chloropropene	ND	ND	ND	ND	0.16
decane	ND	ND	ND	0.35	ND
dichlorodifluoromethane	2.38	3.32	3.31	2.69	2.37
ethanol	1.46	ND	ND	ND	ND
ethylbenzene	0.45	0.28	0.24	0.27	ND
4-ethyltoluene	ND	ND	ND	0.29	0.26
n-heptane	0.41	0.23	0.22	0.23	0.23
hexachlorobutadiene	ND	ND	ND	0.55	ND
hexane	0.92	0.46	0.55	0.51	0.52
methylene chloride	0.42	0.24	0.26	0.20	0.29
4-methyl-2-pentanone	ND	ND	ND	0.25	0.21
a-methylstyrene	ND	ND	ND	0.26	ND
naphthalene	ND	0.53	ND	0.27	0.26
n-pentane	1.80	1.89	1.40	1.41	1.33
propylene	0.42	0.13	0.73	0.11	1.10
n-propyl benzene	ND	ND	ND	ND	0.26
styrene	ND	ND	0.22	ND	ND
tetrachloroethylene	0.71	0.35	0.37	ND	ND
toluene	2.03	2.11	1.99	1.63	1.74
1,1,1-trichloroethane	0.81	1.03	0.40	0.30	0.28
trichloroethene	ND	ND	ND	ND	0.28
trichlorofluoromethane	1.49	1.77	1.72	1.26	1.43
1,1,2-trichloro-1,2,2-trifluoroethane	ND	0.49	0.56	0.42	0.46
1,2,4-trimethylbenzene	0.29	0.36	0.27	0.31	0.28
n-undecane	ND	ND	ND	0.36	ND
vinyl acetate	ND	ND	0.50	0.50	1.15
o-xylene	0.46	0.31	0.25	0.37	0.24
total m+p-xylene	1.09	1.60	0.52	0.83	0.34

B1-13. Montgomery County (2003-2004)

Location: RAPCA (VANDALIA / TIPP CITY)		
	Montgomery County	
	Downtown Dayton Library	
	215 E. Third St.	
	Dayton	
AIRS#:	**39-113-0032**	
Sampler:	**Xontech - J23413**	
Concentrations:	**µg/m^3**	
Compounds	**2003**	**2004**
acetone	3.93	12.74
acetonitrile	0.19	0.45
acrylonitrile	0.42	1.52
benzene	1.51	1.36
1,3-butadiene	0.17	0.18
n-butane	3.90	3.14
2-butanone	0.77	1.92
carbon disulfide	7.06	5.25
carbon tetrachloride	0.59	0.71
chlorodifluoromethane	2.33	1.25
chloroform	0.36	0.25
chloromethane	0.91	0.92
cyclohexane	0.18	0.19
decane	1.10	0.41
dichlorodifluoromethane	3.44	2.77
cis-1,2-dichloroethene	ND	0.21
n-dodecane	0.40	ND
ethylbenzene	0.34	0.26
4-ethyltoluene	0.38	0.26
n-heptane	0.33	0.30
hexane	1.29	1.00
methylene chloride	0.49	0.39
4-methyl-2-pentanone	ND	0.23
naphthalene	0.40	0.29
n-nonane	0.46	0.32
n-octane	0.25	0.33
n-pentane	2.11	1.87
propylene	0.20	1.38
n-propyl benzene	0.29	ND
tetrachloroethylene	0.46	0.44
toluene	3.11	2.38
trichloroethene	ND	0.42
trichlorofluoromethane	3.96	2.46
1,1,2-trichloro-1,2,2-trifluoroethane	ND	0.54
1,2,4-trimethylbenzene	0.80	0.36
1,3,5-trimethylbenzene	0.44	0.26
n-undecane	1.01	0.34
vinyl acetate	ND	0.91
o-xylene	0.45	0.27
total m+p-xylene	1.30	0.54

B1-14. Scioto County (2000-2003)

Urban Air Monitoring: VOC Sampling				**2000-2003**
Annual Average Concentrations				
Location:	**PORTSMOUTH**			
	Scioto County			
	Portsmouth Water Treatment Plant			
	4862 Gallia			
	New Boston, OH			
AIRS #	**39-145-0013**			
Sampler:	N/A			
Concentrations:	**µg/m³**			
Compounds	**2000**	**2001**	**2002**	**2003**
acetone	11.17	8.51	7.11	4.33
acetonitrile	0.41	0.26	0.19	0.17
acrylonitrile	0.87	0.36	0.27	0.24
benzene	6.77	10.01	4.33	1.34
bromodichloromethane	ND	ND	0.35	0.46
1,3-butadiene	0.33	0.36	0.15	0.12
n-butane	7.99	8.81	7.30	4.63
2-butanone	1.58	0.19	1.61	1.73
carbon disulfide	5.56	3.75	1.96	1.46
carbon tetrachloride	0.33	0.34	0.72	0.44
chlorobenzene	ND	ND	ND	0.59
chlorodifluoromethane	0.63	0.73	0.56	0.66
chloroethane / ethyl chloride	0.41	ND	ND	ND
chloroform	0.26	ND	0.35	0.97
chloromethane / methyl chlor	0.84	1.07	1.53	0.92
cumene	ND	0.28	0.29	0.27
cyclohexane	0.42	ND	ND	ND
decane	0.6	ND	0.37	0.31
1,4-dichlorobenzene (para)	ND	ND	ND	0.32
dichlorodifluoromethane	1.87	2.91	3.61	2.46
1,2-dichloro-1,1,2,2-tetrafluor	ND	ND	0.36	ND
n-dodecane	0.71	ND	ND	0.37
ethanol	13.59	ND	ND	ND
ethylbenzene	0.44	0.23	0.27	0.28
4-ethyltoluene	ND	ND	ND	0.29
n-heptane	0.45	ND	0.22	0.21
hexane	0.76	0.42	0.46	0.45
methylene chloride / dichloror	0.43	0.25	0.29	ND
a-methylstyrene	0.49	ND	ND	ND
naphthlene	2.45	4.22	1.97	0.36
n-octane	ND	ND	ND	0.26
n-pentane	2.86	2.15	2.10	1.31
propylene	0.9	0.29	1.49	0.14
styrene	ND	2.37	0.22	ND
toluene	1.93	2.73	1.73	1.07
1,2,4-trichlorobenzene	1.46	ND	ND	ND
1,1,1-trichloroethane	0.57	ND	ND	ND
trichloroethene	ND	ND	ND	0.34
trichlorofluoromethane	112.87	31.29	6.16	4.33
1,1,2-trichloro-1,2,2-trifluoroe	0.82	ND	0.67	ND
1,2,4-trimethylbenzene	0.37	0.63	0.37	0.36
n-undecane	1.31	ND	0.36	0.33
vinyl acetate	0.38	ND	0.58	3.50
o-xylene	0.46	0.31	0.28	0.42
total m+p-xylene	0.95	0.80	0.53	0.87

B1-15. Summit County (2000-2003)

Urban Air Monitoring: VOC Sampling				**2000-2003**
Annual Average Concentrations				
Location:	**Akron**			
	Summit County			
	Akron Region Air Quality			
	Management District			
	East High School			
	80 Brittain Rd.			
AIRS #:	**39-153-0017**			
Sampler:	**SIS 0799-01**			
Concentrations:	**µg/m^3**			
Compounds	**2000**	**2001**	**2002**	**2003**
acetone	9.56	14.36	5.72	5.00
acetonitrile	0.38	0.31	0.20	ND
acrylonitrile	ND	0.28	0.35	0.29
benzene	1.28	1.27	1.37	1.42
1,3-butadiene	0.35	0.32	0.21	0.28
n-butane	6.83	8.82	3.28	5.23
2-butanone	2.83	2.46	2.64	1.90
carbon disulfide	4.37	16.44	2.32	1.12
carbon tetrachloride	ND	0.59	0.59	0.41
chlorodifluoromethane	0.63	1.24	1.38	4.21
chloroethane	ND	0.16	0.16	ND
chloromethane	0.76	1.19	1.03	0.91
cyclohexane	0.30	0.41	0.24	0.35
decane	0.76	0.52	0.33	ND
1,4-dichlorobenzene (para)	ND	0.43	ND	ND
dichlorodifluoromethane	2.32	5.97	3.04	3.68
n-dodecane	0.85	0.43	ND	ND
ethanol	176.86	ND	ND	ND
ethylbenzene	0.45	0.78	0.87	0.49
4-ethyltoluene	ND	0.28	0.27	ND
n-heptane	0.51	0.58	0.39	0.43
hexane	1.68	2.44	1.28	1.68
methylene chloride	0.47	0.57	0.82	0.24
4-methyl-2-pentanone	0.25	0.26	0.28	ND
a-methylstyrene	ND	0.26	0.33	ND
naphthalene	1.06	0.33	ND	0.29
n-nonane	0.33	0.38	0.29	ND
n-octane	0.50	0.32	0.26	0.25
n-pentane	3.29	4.67	39.58	5.36
propylene	0.36	0.60	0.18	ND
n-propyl benzene	ND	0.27	ND	ND
styrene	0.24	0.96	1.92	0.57
tetrachloroethylene	ND	ND	0.37	0.40
toluene	2.11	4.09	5.79	2.94
1,1,1-trichloroethane	47.66	1249.11	438.12	61.35
trichlorofluoromethane	1.61	42.08	39.19	8.09
1,1,2-trichloro-1,2,2-trifluoroet	ND	ND	0.50	ND
1,2,4-trimethylbenzene	0.55	1.02	0.39	0.54
1,3,5-trimethylbenzene	0.28	0.30	ND	ND
n-undecane	1.31	0.61	0.34	ND
vinyl acetate	ND	0.40	0.64	ND
o-xylene	0.49	0.82	0.53	0.56
total m+p-xylene	1.15	2.84	1.96	1.81

B1-16. Washington County (2000-2008)

Urban Air Monitoring: VOC Sampling					**2000-2008**				
Annual Average Concentrations									
Location:	**SEDO**								
	Washington County								
	Washington County Career Center								
	Rt 2								
	Marietta								
AIRS # :	**39-167-0008**								
Sampler:	**Xon - J23416**								
Concentrations:	**µg/m³**								
Compounds	**2000**	**2001**	**2002**	**2003**	**2004**	**2005**	**2006**	**2007**	**2008**
acetone	7.54	8.06	9.10	3.72	11.24	14.15	14.46	7.66	7.97
acetonitrile	0.34	0.22	**ND**	**ND**	0.46	1.01	0.71	0.30	**ND**
acrylonitrile	0.83	0.49	0.82	0.46	2.37	2.74	3.19	1.99	1.40
benzene	0.51	0.42	0.55	0.56	0.89	0.55	1.08	0.37	0.34
bromomethane	**ND**	**ND**	**ND**	**ND**	**ND**	**ND**	**ND**	0.21	**ND**
1,3-butadiene	0.23	0.17	0.13	0.12	0.13	**ND**	**ND**	0.12	**ND**
n-butane	4.90	5.61	4.50	4.83	4.10	3.31	5.66	3.94	4.10
2-butanone	1.31	0.32	0.84	0.67	1.26	2.53	1.70	1.70	1.24
carbon disulfide	5.89	4.34	2.60	1.69	3.07	2.27	2.64	3.43	2.61
carbon tetrachloride	0.38	0.39	0.55	0.37	0.60	0.50	0.47	0.34	0.53
chlorobenzene	0.60	0.41	0.45	0.82	0.46	0.40	0.27	0.52	0.91
chlorodifluoromethan	1.17	1.95	0.93	0.68	0.90	0.93	5.82	0.93	0.89
chloroethane	**ND**	0.19	0.15	**ND**	**ND**	0.15	**ND**	**ND**	**ND**
chloroform	**ND**	**ND**	**ND**	**ND**	0.25	**ND**	**ND**	**ND**	**ND**
chloromethane	1.06	1.22	1.50	1.46	1.04	1.09	1.12	1.26	1.45
cyclohexane	0.51	0.36	0.20	0.28	0.26	0.25	0.27	0.33	0.30
decane	0.60	0.37	0.31	0.39	**ND**	**ND**	0.30	**ND**	**ND**
1,4-dichlorobenzene	**ND**	0.53	0.46	**ND**	**ND**	0.40	0.60	**ND**	**ND**
dichlorodifluorometha	2.14	2.97	2.98	2.46	2.45	2.37	2.68	2.46	2.77
n-dodecane	**ND**	0.37	**ND**	0.37	**ND**	**ND**	**ND**	**ND**	**ND**
ethanol	6.29	**ND**	**ND**	**ND**	**ND**	**ND**	**ND**	**ND**	**ND**
ethylbenzene	0.47	0.24	0.26	**ND**	**ND**	**ND**	0.44	**ND**	**ND**
4-ethyltoluene	0.76	**ND**	**ND**	**ND**	**ND**	**ND**	0.28	**ND**	**ND**
n-heptane	0.46	0.31	0.25	0.30	0.31	0.27	0.46	0.56	0.35
hexane	1.16	0.73	0.55	0.59	0.80	0.63	1.18	0.54	0.50
methanol	3.86	**ND**	**ND**	**ND**	**ND**	**ND**	**ND**	**ND**	**ND**
methylene chloride / (	0.43	0.22	0.25	0.19	0.20	0.19	0.32	0.20	0.34
4-methyl-2-pentanon	**ND**	**ND**	0.22	**ND**	0.29	0.22	0.24	0.21	**ND**
a-methylstyrene	**ND**	**ND**	0.26	**ND**	**ND**	**ND**	**ND**	**ND**	**ND**
naphthalene	1.04	0.29	0.28	0.37	0.29	0.31	0.41	0.27	**ND**
n-nonane	0.27	0.30	**ND**	0.29	**ND**	**ND**	**ND**	**ND**	**ND**
n-octane	0.47	0.28	0.25	0.34	**ND**	**ND**	0.27	0.26	**ND**
n-pentane	1.95	2.15	1.63	1.40	1.76	1.40	2.57	1.49	1.61
propylene	**ND**	0.20	**ND**	**ND**	2.13	2.03	3.02	1.06	0.81
n-propyl benzene	0.74	**ND**	**ND**	**ND**	**ND**	**ND**	**ND**	**ND**	**ND**
styrene	0.26	0.23	0.26	0.67	0.28	0.27	0.24	0.26	0.42
toluene	1.05	0.83	0.95	0.67	1.05	0.47	2.66	0.62	0.48
1,2,4-trichlorobenzen	1.47	**ND**	**ND**	**ND**	**ND**	**ND**	**ND**	**ND**	**ND**
1,1,1-trichloroethane	**ND**	0.30	**ND**	**ND**	0.28	**ND**	**ND**	**ND**	**ND**
trichlorofluoromethan	12.46	24.57	16.46	5.28	4.17	3.18	2.14	14.45	8.37
1,1,2-trichloro-1,2,2-t	0.87	0.93	0.64	0.48	0.59	0.40	0.47	0.46	**ND**
1,2,4-trimethylbenzer	0.52	0.41	0.26	**ND**	0.26	0.27	0.53	**ND**	**ND**
1,3,5-trimethylbenzer	0.31	0.27	**ND**	**ND**	**ND**	**ND**	0.27	**ND**	**ND**
n-undecane	**ND**	0.43	**ND**	0.40	**ND**	**ND**	0.34	**ND**	0.58
vinyl acetate	**ND**	0.38	0.62	0.98	1.22	2.19	1.18	1.88	1.09
o-xylene	0.51	0.26	0.25	0.22	0.23	**ND**	0.51	**ND**	**ND**
total m+p-xylene	1.10	0.39	0.47	0.37	0.30	**ND**	1.29	**ND**	**ND**

APPENDIX B2, OHIO YEARLY Averages
Heavy Metals

B 2-1. Butler/Ohio Bell, Heavy Metals (2004-2009)

MIDDLETOWN HEAVY METALS DATA,
Ohio Bell **2004-2009**
3901 Lefferson Rd.
AQS: 39-017-0015, units -- µg/m³
Butler - designated, Parameters

Year	beryllium	chromium	lead	manganese	nickel	zinc	arsenic	cadmium	iron
2004	0.0000	0.0041	0.0085	0.0468	0.0043	0.0843	0.0013	0.0005	0.0025
2005	0.0001	0.0054	0.0080	0.0595	0.0043	0.0848	0.0017	0.0005	0.0025
2006	0.0001	0.0051	0.0070	0.0678	0.0043	0.1070	0.0014	0.0005	0.4029
2007	0.0001	0.0041	0.0056	0.0582	0.0043	0.0527	0.0012	0.0002	0.4325
2008	0.0001	0.0041	0.0065	0.0564	0.0027	0.0555	0.0013	0.0003	0.3860
2009	0.0001	0.0046	0.0071	0.0578	0.0040	0.0769	0.0014	0.0004	0.2453

B 2-2. Butler/Oneida, Heavy Metals (2000-2002)

Middletown Heavy Metals Data,
Oneida School I **2000-2002**
Yankee Rd.
AIRS: 39-017-0014, units -- µg/m³
Butler County, Parameters

Year	beryllium	chromium	lead	manganese	nickel	zinc	arsenic	cadmium
2000	0.0001	0.0042	0.0156	0.0677	0.0052	0.0482	0.0016	0.0002
2001	0.0001	0.0050	0.0117	0.0919	0.0050	0.0542	0.0020	0.0003
2002	0.0001	0.0045	0.0078	0.0737	0.0049	0.0400	0.0012	0.0002

B 2-3. Columbia/Maryland Ave, Heavy Metals (2001-2009)

EAST LIVERPOOL HEAVY METALS DATA,
500 Maryland Ave. **2001 - 2009**
AIRS: 39-029-0022, units -- µg/m³
Columbiana - designated

Year	beryllium	chromium	lead	manganese	nickel	zinc	arsenic	cadmium	mercury	iron	potassium
2001	0.0001	0.0285	0.0180	0.6800	0.0175	0.0445	0.0023	0.0004	0.0000	0.0025	0.3300
2003	0.0001	0.0039	0.0192	0.1438	0.0228	0.0916	0.0022	0.0010	0.0000	0.0025	0.3300
2004	0.0000	0.0038	0.0110	0.2383	0.0025	0.0553	0.0019	0.0008	0.0000	0.0025	0.3300
2005	0.0000	0.0041	0.0117	0.1848	0.0027	0.1026	0.0018	0.0009	0.0000	0.0025	0.3300
2006	0.0000	0.0039	0.0180	0.3198	0.0031	0.1327	0.0145	0.0013	0.0000	0.2777	0.2854
2007	0.0000	0.0053	0.0178	0.3585	0.0051	0.1498	0.0020	0.0017	0.0001	0.3300	0.2979
2008	0.0000	0.0033	0.0163	0.1666	0.0019	0.0893	0.0019	0.0011	0.0001	0.0893	0.3438
2009	0.0000	0.0026	0.0116	0.0731	0.0013	0.0696	0.0018	0.0006	0.0000	0.2523	0.3375

B 2-4. Columbia/Port Authority, Heavy Metals (2000-2009)

EAST LIVERPOOL HEAVY METALS DATA,
Port Authority **2000 - 2009**
1250 St. George St.
AIRS: 39-029-0019, units -- µg/m³
Columbiana County, Parameters

Year	beryllium	chromium	lead	manganese	nickel	zinc	arsenic	cadmium	mercury	iron	potassium
2000	0.0002	0.0123	0.0166	0.6690	0.0076	0.0644	0.0038	0.0009	0.0004	0.0025	0.2438
2001	0.0002	0.0123	0.0166	0.6690	0.0076	0.0644	0.0038	0.0009	0.0004	0.0025	0.2438
2003	0.0001	0.0045	0.0182	0.2996	0.0020	0.1973	0.0026	0.0014	0.0001	0.0025	0.2438
2004	0.0001	0.0057	0.0165	0.5333	0.0020	0.0843	0.0027	0.0007	0.0001	0.0025	0.2438
2005	0.0002	0.0049	0.0135	0.3009	0.0020	0.1132	0.0021	0.0010	0.0001	0.0025	0.2438
2006	0.0002	0.0050	0.0203	0.4882	0.0034	0.1585	0.0023	0.0015	0.0001	0.4119	0.2940
2007	0.0001	0.0051	0.0184	0.4326	0.0040	0.1367	0.0023	0.0013	0.0001	0.4142	0.2758
2008	0.0001	0.0030	0.0165	0.2074	0.0023	0.0945	0.0023	0.0014	0.0001	0.4206	0.3025
2009	0.0001	0.0030	0.0130	0.1351	0.0017	0.0788	0.0022	0.0007	0.0001	0.4171	0.3213

B 2-5. Columbia/Columbia/Water Plant Heavy Metals (2000-2009)

2220 Michigan Ave.						**2000 - 2009**					
AIRS: 39-029-0020, units -- µg/m³											
Columbiana County, Parameters											
Year	**beryllium**	**chromium**	**lead**	**manganese**	**nickel**	**zinc**	**arsenic**	**cadmium**	**mercury**	**iron**	**potassium**
2000	0.0001	0.0170	0.0204	1.5277	0.0104	0.0881	0.0037	0.0008	0.0003	0.0025	0.1969
2001	0.0009	0.0235	0.0235	0.6500	0.0180	0.0940	0.0025	0.0011	0.0000	0.0025	0.1969
2003	0.0001	0.0262	0.0159	0.9576	0.0090	0.1215	0.0021	0.0010	0.0001	0.0025	0.1969
2004	0.0000	0.0204	0.0156	2.1258	0.0083	0.0578	0.0042	0.0006	0.0001	0.0025	0.1969
2005	0.0003	0.0218	0.0188	1.3153	0.0114	0.0904	0.0031	0.0009	0.0001	0.0025	0.1969
2006	0.0000	0.0162	0.0223	2.2158	0.0185	0.1878	0.0028	0.0017	0.0001	0.6277	0.2132
2007	0.0002	0.0208	0.0194	1.4654	0.0126	0.1066	0.0031	0.0010	0.0001	0.1067	0.1996
2008	0.0000	0.0086	0.0124	1.4508	0.0185	0.0945	0.0020	0.0012	0.0001	0.5850	0.3158

B 2-6. Coshocton County Heavy Metals (2000-2002)

Coshocton County HEAVY METALS DATA								
1840 Otsego Ave.								
AIRS: 39-031-0005				**2000-2002**				
Coshocton County, units -- µg/m³								
Year	**beryllium**	**chromium**	**lead**	**manganese**	**nickel**	**zinc**	**arsenic**	**cadmium**
2000	0.0000	0.0056	0.0100	0.0107	0.0076	0.0910	0.0010	0.0007
2001	0.0000	0.0042	0.0118	0.0170	0.0056	0.0865	0.0015	0.0004
2002	0.0000	0.0059	0.0147	0.1436	0.0079	0.1188	0.0021	0.0005

B 2-7. Cuyahoga/Ferro A & Ferro B, Heavy Metals (2000-2009)

CLEVELAND
Cuyahoga County **2000-2009**
FERRO "A"
4150 East 56th Street
AIRS: 39-035-0049 units -- µg/m³

YEAR	beryllium	chromium	lead	manganese	nickel	zinc	arsenic	cadmium
2000	0.0001	0.0063	0.1218	0.1624	0.1977	0.1563	0.0018	0.0011
2001	0.0001	0.0055	0.0928	0.1136	0.0255	0.1260	0.0015	0.0008
2002	0.0001	0.0054	0.0988	0.1030	0.0175	0.0887	0.0014	0.0006
2003	0.0001	0.0040	0.1528	0.1203	0.0239	0.1128	0.0018	0.0008
2004	0.0001	0.0058	0.0992	0.1347	0.0258	0.0979	0.0020	0.0008
2005	0.0001	0.0063	0.1026	0.1192	0.0249	0.1088	0.0023	0.0007
2006	0.0000	0.0051	0.0703	0.1045	0.0666	0.1027	0.0021	0.0005
2007	0.0000	0.0056	0.0990	0.1770	0.1008	0.1590	0.0016	0.0004
2008	0.0000	0.0058	0.1111	0.1380	0.0610	0.1139	0.0019	0.0007
2009	0.0000	0.0052	0.0898	0.1138	0.0542	0.0986	0.0013	0.0003

CLEVELAND
Cuyahoga County **2000-2008**
FERRO "B"
4150 East 56th Street
AIRS: 39-035-0049 units -- µg/m³

YEAR	beryllium	chromium	lead	manganese	nickel	zinc	arsenic	cadmium
2000	0.0001	0.0065	0.1182	0.1665	0.0565	0.1553	0.0017	0.0011
2001	0.0001	0.0056	0.0983	0.1199	0.0272	0.1341	0.0016	0.0008
2002	0.0001	0.0056	0.0951	0.1107	0.0170	0.0902	0.0015	0.0006
2007	0.0000	0.0058	0.1019	0.1318	0.0992	0.0976	0.0016	0.0004
2008	0.0001	0.0058	0.1147	0.1458	0.0619	0.1125	0.0018	0.0007

B 2-8. Cuyahoga/Asphalt A Heavy Metals (2000-2009)

CLEVELAND Cuyahoga County Asphalt Plant West 3rd St. AIRS: 39-035-0061 units -- µg/m³				2001-2009				
Year	beryllium	chromium	lead	manganese	nickel	zinc	arsenic	cadmium
2001	0.0001	0.0047	0.0425	0.0728	0.0050	0.1363	0.0015	0.0007
2002	0.0000	0.0038	0.0339	0.0295	0.0051	0.0814	0.0013	0.0007
2003	0.0001	0.0018	0.1046	0.0477	0.0009	0.0959	0.0019	0.0011
2004	0.0001	0.0041	0.0221	0.0724	0.0030	0.0927	0.0016	0.0008
2005	0.0001	0.0041	0.0149	0.0712	0.0032	0.0967	0.0016	0.0007
2006	0.0001	0.0028	0.0176	0.0592	0.0036	0.0807	0.0017	0.0004
2007	0.0000	0.0036	0.0186	0.0592	0.0041	0.0733	0.0013	0.0003

B 2-9 Cuyahoga/Fortran Printing, Heavy Metals (2000-2009)

Cuyahoga County					2000 - 2009			
Fortran Printing Inc								
5777 GRANT AVE.								
AIRS: 39-035-0050		units -- µg/m³						
Year	beryllium	chromium	lead	manganese	nickel	zinc	arsenic	cadmium
2000	0.0000	0.0045	0.0527	0.0677	0.0102	0.0923	0.0019	0.0006
2001	0.0001	0.0051	0.0299	0.0591	0.0071	0.0825	0.0018	0.0007
2002	0.0000	0.0054	0.0390	0.0735	0.0067	0.0602	0.0017	0.0006
2003	0.0000	0.0023	0.0303	0.0556	0.0026	0.0629	0.0018	0.0006
2004	0.0000	0.0043	0.0349	0.0638	0.0069	0.0673	0.0021	0.0008
2005	0.0000	0.0052	0.0429	0.0790	0.0103	0.0857	0.0027	0.0007
2006	0.0000	0.0042	0.0218	0.0723	0.0195	0.0693	0.0026	0.0005
2007	0.0000	0.0046	0.0286	0.0848	0.0342	0.0713	0.0019	0.0006
2008	0.0000	0.0037	0.0223	0.0673	0.0178	0.0669	0.0018	0.0005
2009	0.0000	0.0035	0.0253	0.0521	0.0169	0.0629	0.0017	0.0004

B 2-10. Cuyahoga/St. Theodosius, Heavy Metals (2000-2009)

CLEVELAND, 2000-2009								
Cuyahoga County					**2000-2009**			
St. Theodosius Church								
2547 St. Tikhon Ave.								
AIRS: 39-035-0038		**units -- µg/m³**						
Year	**beryllium**	**chromium**	**lead**	**manganese**	**nickel**	**zinc**	**arsenic**	**cadmium**
2000	0.0001	0.0041	0.0202	0.0603	0.0050	0.0910	0.0015	0.0006
2001	0.0001	0.0044	0.0207	0.0526	0.0051	0.1127	0.0015	0.0008
2002	0.0000	0.0037	0.0168	0.0262	0.0050	0.0604	0.0013	0.0006
2003	0.0000	0.0007	0.0195	0.0388	0.0000	0.0779	0.0016	0.0009
2004	0.0000	0.0033	0.0202	0.0403	0.0028	0.0675	0.0016	0.0009
2005	0.0000	0.0036	0.0196	0.0553	0.0031	0.0963	0.0021	0.0012
2006	0.0000	0.0035	0.0193	0.0480	0.0035	0.0884	0.0020	0.0006
2007	0.0000	0.0029	0.0155	0.0365	0.0036	0.0656	0.0015	0.0004
2008	0.0000	0.0030	0.0164	0.0513	0.0034	0.0656	0.0012	0.0005
2009	0.0000	0.0026	0.0103	0.0191	0.0024	0.0468	0.0010	0.0003

B 2-11. Cuyahoga/Fire Station 11, Heavy Metals (2004-2006)

CLEVELAND,								
Cuyahoga County					**2004-2006**			
Fire Station #11								
7629 Broadway Ave.								
AIRS: 39-035-0068		**units -- µg/m³**						
YEAR	**beryllium**	**chromium**	**lead**	**manganese**	**nickel**	**zinc**	**arsenic**	**cadmium**
2004	0.0000	0.0040	0.0304	0.0362	0.0053	0.0936	0.0022	0.0009
2005	0.0000	0.0041	0.0230	0.0341	0.0088	0.0891	0.0019	0.0009
2006	0.0000	0.0037	0.0159	0.0300	0.0058	0.0677	0.0016	0.0005

B 2-12. Cuyahoga/Fire “4A” & “4B”, Heavy Metals (2000-2009)

CLEVELAND,								
Cuyahoga County					**2000-2009**			
FIRE "4A"								
3136 Lorain Ave.								
AIRS: 39-035-0042	**units -- µg/m³**							
Year	**beryllium**	**chromium**	**lead**	**manganese**	**nickel**	**zinc**	**arsenic**	**cadmium**
2000	0.0000	0.0038	0.0235	0.0249	0.0061	0.0650	0.0012	0.0004
2001	0.0000	0.0040	0.0212	0.0261	0.0054	0.0825	0.0015	0.0006
2002	0.0000	0.0041	0.0132	0.0144	0.0055	0.0588	0.0012	0.0012
2003	0.0000	0.0000	0.0124	0.0156	0.0034	0.0533	0.0013	0.0005
2004	0.0000	0.0024	0.0153	0.0151	0.0029	0.0535	0.0016	0.0005
2005	0.0000	0.0022	0.0191	0.0200	0.0029	0.0774	0.0018	0.0010
2006	0.0000	0.0022	0.0143	0.0192	0.0033	0.0660	0.0016	0.0005
2007	0.0000	0.0022	0.0174	0.0207	0.0035	0.0608	0.0013	0.0004
2008	0.0000	0.0021	0.0141	0.0185	0.0023	0.0473	0.0012	0.0004
2009	0.0000	0.0016	0.0088	0.0120	0.0022	0.0372	0.0010	0.0003

CLEVELAND,								
Cuyahoga County					**2000-2009**			
FIRE "4B"								
3136 Lorain Ave.								
AIRS: 39-035-0042	**units -- µg/m³**							
Year	**beryllium**	**chromium**	**lead**	**manganese**	**nickel**	**zinc**	**arsenic**	**cadmium**
2000	0.0000	0.0038	0.0225	0.0198	0.0142	0.0555	0.0011	0.0006
2001	0.0000	0.0044	0.0205	0.0255	0.0058	0.0802	0.0016	0.0006
2002	0.0000	0.0045	0.0152	0.0182	0.0060	0.0683	0.0013	0.0011
2007	0.0000	0.0027	0.0171	0.0204	0.0088	0.0585	0.0014	0.0003
2008	0.0000	0.0022	0.0136	0.0181	0.0025	0.0461	0.0011	0.0003
2009	0.0000	0.0016	0.0087	0.0117	0.0022	0.0365	0.0011	0.0002

B 2-13. Cuyahoga/Fire 22 , Heavy Metals (2004-2006)

CLEVELAND,								
Cuyahoga County			**2004-2006**					
Fire Station #22								
7300 Superior Ave.								
AIRS: 39-035-0069		**units -- µg/m³**						
YEAR	**beryllium**	**chromium**	**lead**	**manganese**	**nickel**	**zinc**	**arsenic**	**cadmium**
2004	0.0000	0.0035	0.0210	0.0239	0.0045	0.0728	0.0020	0.0007
2005	0.0000	0.0035	0.0133	0.0213	0.0045	0.0615	0.0018	0.0005
2006	0.0000	0.0035	0.0133	0.0195	0.0045	0.0532	0.0016	0.0003

B 2-14. Cuyahoga/Gate A , Heavy Metals (2001-2003)

CLEVELAND								
Cuyahoga County								
Gate "A"					**2001-2003**			
2850 3rd St.								
AIRS: 39-035-0063		**units -- µg/m³**						
YEAR	**beryllium**	**chromium**	**lead**	**manganese**	**nickel**	**zinc**	**arsenic**	**cadmium**
2001	0.0001	0.0047	0.0536	0.0863	0.0052	0.1392	0.0017	0.0008
2002	0.0000	0.0040	0.0630	0.0366	0.0051	0.0908	0.0016	0.0008
2003	0.0001	0.0031	0.2333	0.0443	0.0000	0.1087	0.0028	0.0023

B 2-15. Fulton /Delta, Heavy Metals (2000-2009)

NWDO HEAVY METALS DATA,								
Delta					**2000-2009**			
200 Van Buren St.								
AIRS: 39-051-0001, units -- µg/m³								
Fulton County, Parameters								
Year	**beryllium**	**chromium**	**lead**	**manganese**	**nickel**	**zinc**	**arsenic**	**cadmium**
2000	0.0000	0.0040	0.2047	0.0101	0.0054	0.4669	0.0011	0.0006
2001	0.0000	0.0053	0.1278	0.0086	0.0064	0.4008	0.0011	0.0008
2002	0.0000	0.0042	0.0966	0.0107	0.0056	0.2422	0.0010	0.0013
2003	0.0000	0.0022	0.1506	0.0122	0.0024	0.3825	0.0010	0.0006
2004	0.0000	0.0022	0.1155	0.0115	0.0024	0.4213	0.0009	0.0005
2005	0.0000	0.0022	0.1946	0.0088	0.0024	1.0223	0.0011	0.0015
2006	0.0000	0.0022	0.1681	0.0075	0.0024	1.0931	0.0007	0.0026
2007	0.0000	0.0022	0.3879	0.0115	0.0138	3.8624	0.0011	0.0057
2008	0.0000	0.0014	0.2788	0.0102	0.0012	2.4883	0.0008	0.0031
2009	0.0000	0.0012	0.0918	0.0098	0.0010	0.4751	0.0008	0.0006

B 2-16. Cuyahoga /IMT (2000-2002)

CLEVELAND, 2000-2002								
Cuyahoga County								
IMT					**2000-2002**			
511 West 164th								
AIRS: 39-035-1003		**units -- µg/m³**						
YEAR	**beryllium**	**chromium**	**lead**	**manganese**	**nickel**	**zinc**	**arsenic**	**cadmium**
2000	0.0000	0.0041	0.0189	0.0598	0.0055	0.1629	0.0016	0.0009
2001	0.0000	0.0043	0.0156	0.0518	0.0057	0.1065	0.0022	0.0005
2002	0.0000	0.0040	0.0120	0.0309	0.0053	0.0795	0.0017	0.0004

B 2-17. Franklin/Woodrow, Heavy Metals (2000-2009)

COLUMBUS, 2000-2002										
Franklin County						2000-2009				
580 E. Woodrow Ave.										
AIRS: 39-049-0025		units -- µg/m³								
Year	**beryllium**	**chromium**	**lead**	**manganese**	**nickel**	**zinc**	**arsenic**	**cadmium**	**mercury**	**iron**
2000	0.0000	0.0039	0.0264	0.0130	0.0052	0.0602	0.0013	0.0003	0.0000	0.0025
2001	0.0000	0.0037	0.0222	0.0133	0.0049	0.0503	0.0013	0.0003	0.0000	0.0025
2002	0.0000	0.0045	0.0183	0.0146	0.0059	0.0563	0.0013	0.0003	0.0000	0.0025
2003	0.0000	0.0000	0.0136	0.0212	0.0000	0.0583	0.0011	0.0003	0.0000	0.0025
2004	0.0000	0.0021	0.0134	0.0127	0.0028	0.0727	0.0010	0.0004	0.0000	0.0025
2005	0.0000	0.0021	0.0074	0.0137	0.0028	0.0720	0.0021	0.0004	0.0000	0.0025
2006	0.0000	0.0024	0.0080	0.0160	0.0028	0.0753	0.0012	0.0004	0.0000	0.0025
2007	0.0000	0.0021	0.2088	0.0143	0.0028	0.0603	0.0084	0.0010	0.0003	0.0025
2008	0.0000	0.0018	0.0089	0.0136	0.0017	0.0662	0.0011	0.0004	0.0000	0.2175
2009	0.0000	0.0014	0.0092	0.0105	0.0010	0.0624	0.0012	0.0002	0.0000	0.1983

B 2-18. Logan/Bellfontaine, Heavy Metals (2000-2009)

SWDO HEAVY METALS DATA,									
Bellefontaine						2000-2009			
1222 Superior Ave.									
AIRS: 39-091-0003, units -- µg/m³									
Logan County, Parameters									
Year	**beryllium**	**chromium**	**lead**	**manganese**	**nickel**	**zinc**	**arsenic**	**cadmium**	**iron**
2000	0.0000	0.0058	0.1814	0.0056	0.0056	0.0259	0.0008	0.0002	0.0025
2001	0.0000	0.0054	0.1135	0.0054	0.0084	0.0245	0.0010	0.0003	0.0025
2002	0.0001	0.0040	0.0808	0.0045	0.0053	0.0185	0.0012	0.0001	0.0025
2003	0.0000	0.0019	0.0001	0.0000	0.0039	0.0257	0.0008	0.0004	0.0025
2004	0.0000	0.0019	0.1053	0.0058	0.0027	0.0233	0.0009	0.0002	0.0025
2005	0.0000	0.0019	0.0783	0.0055	0.0043	0.0218	0.0009	0.0002	0.0025
2006	0.0000	0.0019	0.0472	0.0046	0.0030	0.0201	0.0008	0.0001	0.1049
2007	0.0000	0.0019	0.0799	0.0066	0.0025	0.0247	0.0011	0.0001	0.1438
2008	0.0000	0.0013	0.0348	0.0048	0.0013	0.0198	0.0008	0.0001	0.0908
2009	0.0000	0.0016	0.0287	0.0045	0.0009	0.0225	0.0008	0.0001	0.0943

B 2-19. Marion/Marion Steel (Left and Right), Heavy Metals (2002-2009)

NWDO HEAVY METALS DATA,									
Marion Steel						2002-2009			
441 Whitmore St. LEFT									
AIRS: units - µg/m³									
Parameters									
Year	**beryllium**	**chromium**	**lead**	**manganese**	**nickel**	**zinc**	**arsenic**	**cadmium**	**mercury**
2002	0.0000	0.0093	0.1211	0.1778	0.0089	0.6073	0.0028	0.0012	0.0008
2003	0.0000	0.0066	0.0782	0.1505	0.0035	0.5005	0.0027	0.0011	0.0003
2004	0.0000	0.0077	0.0630	0.1483	0.0030	0.3209	0.0021	0.0008	0.0002
2005	0.0000	0.0060	0.0532	0.1287	0.0033	0.3356	0.0023	0.0009	0.0004
2007	0.0000	0.0030	0.0078	0.0979	0.0031	0.5500	0.1703	0.0010	0.0017
2008	0.0000	0.0094	0.1424	0.2183	0.0039	0.5928	0.0023	0.0030	0.0004
2009	0.0000	0.0058	0.0365	0.1290	0.0027	0.1663	0.0015	0.0006	0.0002

NWDO HEAVY METALS DATA, 2002-2009									
Marion Steel									
441 Whitmore St. RIGHT									
AIRS: units - µg/m³									
Parameters									
Year	**beryllium**	**chromium**	**lead**	**manganese**	**nickel**	**zinc**	**arsenic**	**cadmium**	**mercury**
2002	0.0000	0.0089	0.0619	0.1178	0.0090	0.3623	0.0018	0.0008	0.0005
2003	0.0000	0.0081	0.1215	0.2270	0.0042	0.5529	0.0038	0.0015	0.0003
2004	0.0000	0.0054	0.0564	0.0920	0.0027	0.2520	0.0019	0.0006	0.0001
2005	0.0000	0.0057	0.0435	0.1015	0.0030	0.2773	0.0019	0.0007	0.0002
2006	0.0000	0.0067	0.0862	0.1323	0.0028	0.4517	0.0021	0.0018	0.0006
2007	0.0000	0.0022	0.0083	0.0977	0.0037	0.5583	0.1797	0.0005	0.0018
2008	0.0000	0.0069	0.0659	0.1308	0.0026	0.3533	0.0019	0.0019	0.0003
2009	0.0000	0.0053	0.0515	0.1108	0.0023	0.2269	0.0015	0.0008	0.0002

B 2-20. Marion/Bellfontaine (Left and Right), Heavy Metals (2000-2007)

NWDO HEAVY METALS DATA,									
Marion Steel					2000-2007				
635 Bellfontaine / Gill Ave. RIGHT									
designated, units -- µg/m³									
Marion County, Parameters									
Year	beryllium	chromium	lead	manganese	nickel	zinc	arsenic	cadmium	mercury
2000	0.0000	0.0056	0.0195	0.0349	0.0059	0.0909	0.0013	0.0003	0.0002
2001	0.0000	0.0037	0.0310	0.0461	0.0049	0.1319	0.0012	0.0004	0.0004
2002	0.0000	0.0039	0.0250	0.0457	0.0047	0.1586	0.0010	0.0004	0.0007
2003	0.0000	0.0028	0.0451	0.0622	0.0031	0.1608	0.0012	0.0008	0.0002
2004	0.0000	0.0028	0.0206	0.0412	0.0031	0.1191	0.0010	0.0004	0.0001
2005	0.0000	0.0030	0.0200	0.0502	0.0031	0.1380	0.0015	0.0004	0.0002
2006	0.0000	0.0036	0.0329	0.0542	0.0031	0.1678	0.0012	0.0006	0.0002
2007	0.0000	0.0055	0.0709	0.0885	0.0031	0.4617	0.0012	0.0016	0.0002

NWDO HEAVY METALS DATA									
Marion Steel					2000-2007				
635 Bellfontaine / Gill Ave. LEFT									
Left/ co-located, units - µg/m³									
Marion County, Parameters									
Year	beryllium	chromium	lead	manganese	nickel	zinc	arsenic	cadmium	mercury
2000	0.0000	0.0057	0.0218	0.0509	0.0066	0.1150	0.0012	0.0004	0.0002
2001	0.0000	0.0038	0.0281	0.0444	0.0050	0.1144	0.0012	0.0004	0.0003
2002	0.0000	0.0039	0.0189	0.0382	0.0049	0.0909	0.0010	0.0003	0.0000
2003	0.0000	0.0033	0.0191	0.0389	0.0038	0.0931	0.0010	0.0004	0.0001
2004	0.0000	0.0034	0.0230	0.0468	0.0038	0.1386	0.0014	0.0005	0.0001
2005	0.0000	0.0032	0.0217	0.0512	0.0038	0.1449	0.0013	0.0004	0.0002
2007	0.0000	0.0044	0.0326	0.0726	0.0038	0.1643	0.0010	0.0008	0.0002

B 2-21. Ottawa/Brush Wellman 32, Heavy Metals (2002-2009)

NWDO HEAVY METALS DATA,								
Brush Wellman 32					**2002-2009**			
AIRS: units - µg/m³								
Parameters								
	beryllium	**chromium**	**lead**	**manganese**	**nickel**	**zinc**	**arsenic**	**cadmium**
2002	0.0001	0.0007	0.0034	0.0045	0.0011	0.0137	0.0006	0.0001
2004	0.0001	0.0003	0.0026	0.0027	0.0007	0.0115	0.0005	0.0001
2005	0.0001	0.0003	0.0035	0.0038	0.0007	0.0152	0.0007	0.0001
2006	0.0001	0.0003	0.0034	0.0036	0.0008	0.0137	0.0006	0.0001
2008	0.0012	0.0004	0.0033	0.0056	0.0006	0.0147	0.0005	0.0001
2009	0.0000	0.0003	0.0028	0.0032	0.0004	0.0098	0.0005	0.0001

B 2-22. Washington/Career Center, Heavy Metals (2000-2009)

Washington County HEAVY METALS DATA,									
Rt. 676 Marietta, Career Center						**2000-2009**			
AIRS: 39-167-0008									
Washinton County, units -- µg/m³									
Parameters									
Year	**beryllium**	**chromium**	**lead**	**manganese**	**nickel**	**zinc**	**arsenic**	**cadmium**	**mercury**
2000	0.0000	0.0033	0.0056	0.0635	0.0044	0.0310	0.0012	0.0020	0.0000
2001	0.0000	0.0045	0.0123	0.3164	0.0057	0.0430	0.0016	0.0010	0.0000
2002	0.0000	0.0045	0.0075	0.1596	0.0060	0.0272	0.0011	0.0005	0.0000
2003	0.0000	0.0024	0.0072	0.2141	0.0023	0.0302	0.0017	0.0037	0.0000
2004	0.0000	0.0020	0.0048	0.1031	0.0026	0.0238	0.0012	0.0017	0.0000
2005	0.0000	0.0013	0.0035	0.1235	0.0022	0.0257	0.0010	0.0008	0.0000
2006	0.0000	0.0013	0.0042	0.0711	0.0013	0.0186	0.0009	0.0003	0.0000
2007	0.0000	0.0013	0.0044	0.1063	0.0013	0.0227	0.0008	0.0003	0.0000
2008	0.0000	0.0014	0.0038	0.1288	0.0006	0.0199	0.0007	0.0005	0.0000
2009	0.0000	0.0009	0.0039	0.0827	0.0006	0.0181	0.0007	0.0002	0.0000

White House Environmental Justice Advisory Council (WHEJAC)

Public Comments presented by Laura Olah – January 26, 2022

My name is Laura Olah, I am a member of the Mole Lake Sokaogon Ojibwa tribe in Wisconsin and acknowledge my great-grandmothers today. I am the Executive Director of Citizens for Safe Water Around Badger and national organizer for the Cease Fire Campaign.

Right now, as we are speaking today, in communities across America and its territories, the Department of Defense, Department of Energy, and industry are permitted by EPA and authorized states to conduct routine **open air** burning and detonation of hazardous waste – causing the uncontrolled release of toxic heavy metals, energetic compounds, perchlorate, nitrogen oxides, hexavalent chromium, PFAS, dioxins and other carcinogens to the environment, and permanently polluting our water. These burn pits are not just overseas, they are here at home.

My neighbors and I organized Citizens for Safe Water Around Badger in 1990 when rural families near the Badger Army Ammunition Plant learned that their drinking water wells were polluted with chemicals from an open burning area that was more than 3 miles away.

Today, more than 30 years later, the Army is still testing our drinking water wells for the explosive dinitrotoluene (DNT) – having spent more than $250 million on remediation efforts.

There are three major groundwater contaminants plumes that have migrated off-site and two of them are coming from open burning areas inside the plant. In September 2020, total DNT was detected in groundwater at concentrations as high as 1,286,900 ng/L – a concentration that is 25,000 times higher than the WI Groundwater Enforcement Standard of 50 ng/L for total DNT.

At active bases like the Crane Naval Surface Warfare Center in Indiana, the military has been allowed to open burn and detonate as much as 109 million pounds of hazardous waste every year, for decades. The McAlester Army Ammunition Plant in Oklahoma is permitted to burn nearly 10 million pounds per year. At industrial sites like Clean Harbors Colfax in Louisiana, the facility is permitted to open burn nearly half-a-million pounds of hazardous waste every year, often enveloping nearby homes in thick black smoke.

Emerging contaminants like PFAS are part of this waste stream. Military countermeasure flares may contain as much as 45% PFAS. PFAS are intentionally added to improve the performance and stability of military explosives and munitions. The military is actively ramping up the production of insensitive and polymer-bonded explosives, so this is a threat that is not going away, in fact it's just going to get bigger.

PFAS are not destroyed in an open fire or even by incineration and instead are continually dispersed to the air and the surrounding community where they accumulate in people, in fish and wildlife – and contaminating our drinking water and groundwater resources.

Even though safer alternative treatment technologies have been successfully deployed by military and the private sector in some communities, but this devasting practice *continues* almost exclusively in communities without the resources to defend themselves from this *relentless* source of toxic exposure and harm.

These are our "domestic" burn pits" – they are here at home in the Unites States and its territories, and almost exclusively in communities that are the most vulnerable to harm. We need to break the cycle, we need your help to make that happen.

Chi-miigwech. Thank you.

Citizens for Safe Water Around Badger | E12629 Weigand's Bay South | Merrimac, WI 53561
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Blue Grass Army Depot *(Richmond, KY)*

Source: Kentucky Environmental Foundation

Clean Harbors Colfax *(Colfax, LA)*

Source: Louisiana Department of Environmental Quality

Former Atlantic Fleet Weapons Training Area *(Vieques, PR)*

Source: Cease Fire Campaign

Holston Army Ammunition Plant *(Kingsport, TN)*

Source: Volunteers for Environmental Health and Justice

Radford Army Ammunition Plant *(Radford, VA)*

Source: EPA

Andersen Air Force Base Open Detonation Disposal Training (*Yigo, Guam)*

Source: Department of Defense

CSWAB Fact Sheet:

Recognizing the Potential for PFAS at OB/OD Munitions Disposal Sites

Source: U.S. Army | GAO-15-538

Some regulators and military officials are starting to recognize the potential for PFAS at Open Burning/Open Detonation (OB/OD) munitions disposal sites. Here are a few examples but there are still hundreds of current and former OB/OD sites that have not been tested for PFAS.

• Naval Weapons Station Yorktown, Virginia

The U.S. Navy Pentagon has identified the former Explosive Ordnance Burning and Disposal area (SWMU 2) and Burn Pad (Site 22) at the Naval Weapons Station in Yorktown as potential source of per- and polyfluoroalkyl substances (PFAS) contamination.[1]

The Burn Pad site at the Yorktown is a 9-acre area that once contained a circular array of steel burning pads that were used for burning waste explosives and spent solvents generated from loading operations. Contamination of groundwater is likely due to releases of chemicals that occurred during these waste handling and burning activities on the ground surface. The Burn Pad area was operational from the early 1940s until 1995.[2]

The remedy at the Yorktown Burn Pad Area has not yet been implemented. A pre-design investigation identified the need to delineate solvent plumes and to define the extent of 1,4-dioxane at the site. The investigation also determined that sampling for PFAS and perchlorate in groundwater is needed.[3] As the potential use of Aqueous Film Forming Foam (AFFF) in controlling the individual burn pads could not be ruled out, the Navy will be conducting analysis for PFAS.

• Seal Beach Naval Weapons Station, California

The Explosives Burning Ground, which operated from 1945-1971[4] at Seal Beach Naval Weapons Station has been identified by the military as a potential source of PFAS contamination. The Explosives Burning Ground was an open area of approximately 15 acres that was used for open burning of ordnance-contaminated wastes. Burned wastes included Explosive-D, Explosive-D sludge from the primary settling basin, waste black powder, waste smokeless powder, black powder, smoke pots containing fog oil, and miscellaneous wastes. Limited burning of the following were also reported through 1972: waste Otto Fuel contaminated with Agitene and solids, damaged smoke signals, underwater explosives demolition charges, and firefighting exercise materials (waste lumber and a few tires, Aqueous Fire Fighting Foam or protein-based foam additive mixed with extinguishing water).[5]

• Letterkenny Army Depot, Pennsylvania

At the request of Pennsylvania Department of Environmental Protection, the U.S. Army will conduct groundwater sampling at Letterkenny's Open Burning/Open Detonation (OB/OD) Areas for PFAS.[6] The open burning areas consist of burning cages, burn pans, and rocket static firing tubes used for demilitarization of propellants and rocket motors. The detonation areas consist of open areas where munitions are demilitarized via detonation. Pending groundwater analysis will include 12 PFAS compounds using U.S. EPA SW-846 Method 537.

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[1] U.S. Department of Navy, Office of the Assistant Secretary (Energy, Installations and Environment), Navy Pentagon, Memorandum Subject: Perfluorinated Compounds/Perfluoralkl Substances (PFC/PFAS) – Identification of Potential Areas of Concern and ENCLOSURE 2 – DERP Sites with Potential PFC/PFAS Contamination and Potential PFC/PFAS AOCs, June 20, 2016.

[2] U.S. Department of Navy, Final Fourth Five-Year Review Report for the Naval Weapons Station Yorktown, Section 10.2 Site Background, March 2018.

[3] U.S. Department of Navy, Final Fourth Five-Year Review Report for the Naval Weapons Station Yorktown, Section 10.3.4, Status of Implementation, March 2018.

[4] U.S. Department of Navy, Naval Weapons Station Seal Beach, Newsletter of the Environmental Investigation and Cleanup Program, October 2007.

[5] California Department of Health Services, Federal Facility Site Remediation Agreement for Seal Beach Naval Weapons Station, page 71, 24 September 1991.

[6] U.S. Department of Army, RCRA Subpart X Part B Permit Application for Open Burning/Open Detonation (OB/OD) Areas, Ammonium Perchlorate Rocket Motor Destruction Facility, and Flashing Furnace, Letterkenny Army Depot, April 2018.

Partnership for Southern Equity
TOGETHER WE PROSPER

The Partnership for Southern Equity (PSE) is a 501 c 3 charitable organization whose mission is to advance policies and institutional actions that promote racial equity and shared prosperity for all in the growth of metropolitan Atlanta and the American South.

WHITE HOUSE ENVIRONMENTAL JUSTICE ADVISORY COUNCIL PUBLIC COMMENTS

Contact:
Docket ID No. EPA-HQ-OA-2021-0683

https://www.epa.gov/environmentaljustice/forms/white-house-environmental-justice-advisory-council-whejac-public-comment

Sending comments via email to whejac@epa.gov, for comments with additional materials.

Written comments can be submitted up to two (2) weeks after the meeting date.

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Submission Date:
February 9, 2022

D855

Thank you for the opportunity for PSE staff to comment on the WHEJAC Public Meeting held on January 26-27, 2022. The comments that we have prepared for Docket ID No. EPA-HQ-OA-2021-0683 are intended to serve as an update on the work that we have been doing on the frontlines and a call-to-action for the WHEJAC and the Biden Administration, from the Partnership for Southern Equity (PSE). PSE works "on-the-ground" in the region of the United States that is home to highest concentration of persistent poverty counties, the southern Black Belt and Appalachia. Recent Executive Orders have charged the Office of Management and Budget (OMB), in partnership with the heads of agencies, to identify, by July 2021, effective methods for assessing whether agency policies and actions (e.g., programs, services, processes, and operations) equitably serve all eligible individuals and communities, particularly those underserved. As part of this effort, agencies were directed to consult with members of communities that have been historically underrepresented in the Federal Government and underserved by, or subject to discrimination in, Federal policies and programs, and to evaluate opportunities, as allowable, to increase coordination, communication, and engagement with community-based and civil rights organizations. The public comments that we offer today are a summary of our learnings as we designed and launched the Justice40 Accelerator. Our goal is to help ready frontline communities to prepare for these investments and navigate the complexities of applying for federal, state, and local funding. Lastly, we have posed questions regarding the efficacy of the WHEJAC and seek to better understand the accountability structure of the body.

About PSE

We are a local organization with regional and national influence. PSE's office is in Atlanta, Georgia: 55 Ivan Allen Jr Blvd NW, Suite 530, Atlanta GA 30308 but our work, presence and impact is experienced across the United States with a particular focus on the Southeastern Region. PSE has emerged as a leading voice for equitable growth in Atlanta and the American South, as our founder, Nathaniel Smith, established the organization in 2009. Our organizational profile is on par with more established firms, due to our unwavering commitment to racial equity and authentic community engagement. As a Black-led organization anchored in the American South, we have consistently demonstrated our professional qualifications and have emerged well-equipped to provide expert insights on how government agencies and commissioned entities can become more equitable in their distribution of resources to frontline communities.

In April 2021, Nathaniel Smith published an Op Ed in The Hill[1] entitled "*How to Make a Just Energy Transition, Make it Local*." For over ten years, PSE has advanced policies and institutional actions that promote racial equity and shared prosperity for all in the growth of the American South. PSE strives to bring equitable balance to the social, political, and economic systems which have long been employed to maintain an imbalance of opportunity and quality of life for people and communities of color. To achieve these outcomes, we work to build an equity ecosystem through consensus building, issue framing, training, policy, advocacy, and collective impact organizing. PSE's work supports advancing systemic reforms in areas where disparities often emerge. Our four primary areas; Just Energy, Just Opportunity (economic inclusion), Just Growth (equitable development) and Just Health (health equity).

PSE is serving as an anchor organization for the Justice40 Accelerator[2]. The Biden-Harris Administration's commitment to ensure 40% of the benefits of its clean energy and climate investments into disadvantaged communities is a historic opportunity. The resulting funding levels across energy, water, housing, and transit/transportation will significantly exceed anything possible through philanthropy alone - representing a pivotal opportunity for reparation and

[1] How to implement a 'just' energy transition? Make it local | TheHill

[2] https://www.justice40accelerator.org/

restoration of communities. Realizing these outcomes will require that we better resource frontline community organizations to take the lead. The Justice 40 Accelerator was conceived as a collaborative partnership between CORE Team members: Elevate, Groundswell, Hummingbird, Partnership for Southern Equity, and The Solutions Project for the purpose of supporting organizations that are on the front lines of addressing climate, the environment, and social injustices in their communities.

In our first 9 months of operations, the Justice40 Accelerator our team privately raised $9.3M in philanthropic funds to help frontline communities meet this moment and opportunity. Our inaugural cohort is comprised of [52 organizations] from across the United States and Puerto Rico. We designed a comprehensive approach by providing $25,000 in direct predevelopment support and targeted technical assistance to each cohort member to help organizations identify and apply for federal funding opportunities. Each cohort member is receiving an investment of approximately $70,000 each to help them quickly prepare for this unique opportunity. The Justice40 is about more than building the contracting and procurement muscle of frontline organizations to compete for federal dollars, we designed a community of practice in an effort to disrupt the traditional ways in which resources flow to communities by reimaging this opportunity to use federal dollars to heal and repair harm to Black and historically disinvested communities of color.

PSE is currently developing a GIS data dashboard that will continue to map the various barriers to accessing federal funds and offer users some insights on how to navigate structural barriers to accessing federal funds. In addition to geocoding each project so that people can see in real time where shovel ready projects that align squarely with the objectives of the Justice40 Initiative—we are using this map to highlight the structural barriers and reinforcing loops of inequality. During our initial scan of barriers to access we have highlighted some challenges that include but are not limited to: Access to predevelopment funds to properly plan projects and hire experts to assist with proposal writing, back office support and general compliance tasks; obtaining a SAMS Number, accessing Grants.GOV, Single Audit Requirements, understanding cross cutting regulations 2 CFR; 24 CFR 85; Local Ordinances; Insurance and Bonding; Navigating local municipalities (all of which are different); Meeting Federal Financial Match Requirements; Federal Reimbursement Requirements; Local Procurement Policies (all of which are long, complicated and different); Expenditure Deadlines (all of which are statutory and different); Compliance and Reporting Requirements; Navigating the use of various Federal Systems: LOCCS, IDIS, DRGR, HMIS, etc.;

To deploy a full of government approach to something so bold as the Justice 40 Initiative, there must be a coordinated approach to retraining and upskilling workers who deliver these programs across all areas of government. There must also be clear responsibility and communications to align resources and priorities appropriately. Understanding and application of the Federal Acquisitions Regulations (FAR) requires complex and specialized knowledge that most frontline organizations lack. Moreover, procurement and contracting professionals at the local levels often need retraining and ongoing professional development to help them understand how to apply the rules in a nonpunitive manner. https://www.acquisition.gov/browse/index/far; and 41 U.S.C. chapter 13, A.

Annual scorecards and performance measures that evaluate the impact of federal dollars to frontline communities will help agencies follow the money and evaluate if the program is effective. These scorecards need to be transparent and available to the public to examine and comment. If an agency is trending below targeted goals, then there should be mandatory corrective action plans that give the agency targeted goals and metrics to meet. Currently, less than 1% of federal contracting dollars go to organizations that are Black or BIPOC led or to communities that are historically disinvested. When state or local municipalities are unable to meet statutory mandates to timely deploy funds—there should be a federal sweep of unencumbered dollars and create a mechanism for frontline organizations to directly apply for and draw down funds from a separate agency discretionary source.

Federal government agencies must see themselves as a part of the problem and the solution. Real community impact is often bogged down in bureaucratic process and long-range strategic plans are reduced to compliance documents. Reimaging the resource delivery system as one that is just, fair looks different than one that is compliance driven. The ways in which Federal regulations were amended to distribute trillions of dollars in aid due to the COVID-19 pandemic, government should take that same liberty to redesign an efficient delivery system overall. Government has an endless amount of data and resources available to reimagine the resource delivery system but needs to prioritize this action and follow-through on the implementation of a system that is fair and just.

To solve problems and get tangible results, ask the people who are closest to the problems to help you design the policy solutions. Black and historically disinvested communities of color have several codified examples by which the federal government acted in bad faith with the intentions to harm. Rebuilding that trust must come through trusted frontline organizations that serve as a bridge to federal law makers. Good public policy is not created in a vacuum, rather it is centered on the lived experiences of local people who know how best to get their needs met. During the Justice 40 Accelerator webinars, we polled participants to glean understanding and their familiarity with the federal resource delivery system. Most participants were willing to share this data during this open forum because the responses were anonymous, participants felt like we were trying to help with no underlying agenda. Our CEO, Nathaniel Smith often says the "Change moves at the speed of trust." If the government wants to collect data to build an evidenced based case, you should start with seeking the truth and making public policy decisions that center on the outcomes of people. Stated differently, good public policy happens with people—not to them.

Governmental agencies can build capacity by investing in and democratizing access to information. Training frontline organizations on how to access federal, state, and local funds, write competitive applications that are concise and compliant, and navigate the federal register requires hundreds of hours and several thousand dollars. Most organizations with the best solutions and shovel right projects lack the financial resources to navigate this system in general. Very simply stated, resource front line communities directly because the current system continues to prove ineffective.

Agencies have an opportunity to reimagine the resource delivery system as one that is restorative and reparative by making data driven investments. The way public benefits are framed in our media, policy and academic discourse positions these entitlements as welfare because they support communities and people. However, when these same subsidies and benefits are targeted to businesses, they are viewed as essential for Economic Development. Changing the ways in which we message about federal subsidies for frontline communities shifts the conversation to a problem-solving frame instead of perpetuating moral-based arguments that are rooted in historical racism. This way of understanding federal funds bifurcates the American people into two classes of citizens: those who deserves help (i.e., corporations and affluent) and those who does not (everybody else). Federal dollars are public funds, and they belong to the people, this means that the people closest to the problems should be at the table decision making table to shape the priorities of their communities. Moreover, these federal investments should provide tangible evidence of how the dollars spent and the impact they had to make the community better off. The commodification of federal resource delivery system is doing what it was designed to do. It is privileging those with means and resources while marginalizing everyone else.

The Federal Acquisition Regulations System is established for the codification and publication of uniform policies and procedures for acquisition by all executive agencies. The Federal Acquisition Regulations System consists of the Federal Acquisition Regulation (FAR), which is the primary document, and agency acquisition regulations that implement or supplement the FAR. The FAR System does not include internal agency guidance of the type described in 1.301(a)(2). Navigating these regulations requires skillful and technical knowledge that most procurement or purchasing professionals working at the local levels do not understand. Simplifying the procurement system for both internal staff and external offerors requires a strategic realignment and coordination between all levels of government and systems reform to be more effective.

The use of easy-to-follow checklist, infographics, and technical assistance to frontline communities can help organizations be more precise in preparing bids. Most of the time, bids are disqualified on minor technicalities and typographical errors. The rigid nature of procurement undermines equity. There is little flexibility or discretion given to contracting professionals to use common sense and ask probing questions. The announcements for proposals are typically lengthy and require numerous hours to follow. Larger organizations with more resources automatically have an advantage as they have several staff dedicated to the preparation and delivery of a proposal when in fact a smaller firm may be more uniquely qualified to deliver on a scope of work but lacked capacity to put forward a strong application. When you add the racist and historical ways in which government contracting has evolved, black led, minority firms are at a stark disadvantage.[3]

Through our deep engagement with community, we have been in the posture of listening and co-creating a meaningful experience that connects frontline organizations to the resources needed to navigate the complicated landscape of federal, state, and local funding opportunities. We have been designing strategic on-ramps and guiding cohort members to opportunities that are best aligned for their projects. The metrics that we are tracking fall into three categories: 1) Cohort Experience, 2) Federal Government Influence, and 3) Funding and Media Ecosystem Influence

Cohort Experience

1. Cohort has a positive / regenerative experience
2. Our offerings are responsive to cohort needs
3. Cohort members have developed new relationships with key federal government personnel
4. Cohort learnings make them better equipped to navigate the federal funding process
    - Duns numbers
    - Number of applications submitted
    - Informed decisions to NOT apply for certain funding due to the small award sizes and compliance hurdles to maintain the award
    - Number of cohort members partnered with a strong / aligned ally
    - Number of grant opportunities applied for and awarded
    - Successful grantees have a positive experience with the federal dollars
    - Resourced to navigate grant implementation

[3] https://scholarship.law.gwu.edu/cgi/viewcontent.cgi?article=2569&context=faculty_publications

Federal Government Influence

1. Federal government has a better understanding of what it takes to meaningfully move resources to the under-resourced BIPOC communities
2. Federal government has made investments / commitments to do better

Funding and Media Ecosystem Influence

1. Funding - Philanthropy and private investment are moving more regenerative dollars to BIPOC frontline, under-resourced communities by way of the J40 Accelerator.
2. Narrative - control of narrative (BIPOC led projects are the "**shovel right**" solutions to climate / clean energy needs (so public / private systems need to work collaboratively to ensure these success stories are lifted),
3. Focus on the long arc of change / transformation, clear story of how public and private sector need to get in formation around the 52 and others like the 52).
4. Reframing the ways in which we talk about federal subsidies. Investments in communities and people should no longer be seen as welfare but as economic development.

In summation, PSE asks of WHEJAC, the following:

1. How will WHEJAC be different and more effective than NEJAC- the National Environmental Justice Advisory Council established in 1993 by President Clinton? Erecting prestigious bodies to confer on the injustices bestowed upon frontline communities has proven to be more ceremonious than effective when it comes to getting the environmental justice communities deserve.
2. To what extent will the WHEJAC develop accountability metrics and transparent communications to the community of the progress and activities of the body?
3. How will WHEJAC ensure that the funds being deployed from the Bipartisan Infrastructure Bill adhere to President Biden's Justice40 Initiative? Recognizing that oversight and compliance at the federal level has traditionally excluded historically marginalized EJ communities.
4. What will the WHEJAC do to ensure that the impact of the body stands as more than another vestigial consortium whose outputs culminate in more than a report?
5. What role does the WHEJAC play in supporting the Biden Administration to implement the 10-12-30 provision?
6. How can WHEJAC ensure the Department of Energy actively participates in a whole-of-government response to work with every single Rural Electric Cooperative in the country to chart a technology transition path?

We are very grateful for this opportunity to submit public comments and hope that we have offered perspective and insights that help move our communities forward.

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Fluorine-containing oxidizers for metal fuels in energetic formulations

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ABSTRACT

Fluorine containing oxidizers, primarily polymers, are extensively used in pyrotechnic compositions. Fluorinated oxidizers are less explored for metalized propellants and explosives despite a potential advantage of substantial heat release combined with gaseous combustion products. This review summarizes different types of fluorinated oxidizers used in energetic formulations or of potential interest for such systems, including gases, polymers, and inorganic compounds. Types of energetic formulations employing metals and fluoropolymers are discussed in more detail, including methods used to prepare composites and resulting salient features of the obtained materials. Laboratory experiments characterizing such materials, in particular, electron microscopy and thermal analysis, are discussed, showing characteristic morphologies and reaction sequences observed in different metal-fluorinated oxidizer composites. Striking similarities are noted in reaction sequences for diverse compositions hinting at possible similarities in the respective reaction mechanisms. Experiments probing ignition and combustion of metal-fluorinated oxidizer composites in laboratory conditions are also reviewed, including impact, flash heating and shock ignition. Finally, some practical performance tests for energetic formulations are described following by a brief discussion of the reaction mechanisms expected to govern ignition and combustion in various metal-fluorinated oxidizer composites. The conclusions are combined with recommendation for future research in the area of reactive metal-fluorinated oxidizer composites.
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1. Introduction

Metal powders may serve as excellent fuels due to their high volumetric and gravimetric heat release upon combustion [1–7]. They also generate high combustion temperatures, sometime in excess of 3000 K, attractive for high-efficiency propellants, enhanced blast explosives, and specialized pyrotechnics [4–7]. Applications in dual use materials, such as structural energetic or reactive materials are also explored [8–10]. The thermodynamic benefits of metals, however, are often offset by their relatively long ignition delays, low burn rates, and generation of condensed products, such as oxides, as a result of combustion [11]. Condensed oxides reduce work produced by the burning energetic composition and cause so-called two-phase losses [12–14], which are detrimental for solid propellants and some explosives. In many energetic formulations, metals are combined with aggressive oxidizers, such as sodium or potassium perchlorate [15,16], to increase their reaction rate. However, use of such aggressive oxidizers is associated with difficulties in handling and storing energetics. In addition, it is important to reduce environmentally undesirable emissions, e.g., corrosive chlorinated products. In some cases, metals may be combined with other oxidizing compounds, such as metal oxides in thermite compositions [17–20]. Such compositions are capable of generating very high temperatures, however, the shortcomings associated with the low burn rates and final condensed products remain. An approach of increasing the burn rates for metals by reducing the sizes of metal particles and mixing them with finely divided oxidizers has been actively explored over the last decade [7]. Indeed, the ignition delays were observed to shorten; however, the burn rates commonly are weakly affected because of rapid sintering of nanostructured materials, which thus lose their structural advantages upon ignition [21,22]. Furthermore, the sensitivity of the nano-scale metal-oxidizer systems to various ignition stimuli, including, in particular, electrostatic discharge (ESD), was also reported to become very high [23], making it difficult to handle such materials. In summary, ignition delays, low burn rates, high sensitivity to ignition by ESD, and generation of condensed oxides as combustion products are main impediments

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Abbreviations

FLOX	Fluorine- liquid oxygen
ESD	Electrostatic discharge
AP	Ammonium perchlorate
HTPB	Hydroxyl-terminated polybutadiene
PCTFE	Polychlorotrifluoroethylene
PVDF	Polyvinylidene fluoride
PVF	Polyvinyl fluoride
FEP	Fluorinated thylene-propylene polymer
ECTFE	Ethylene chlorotrofluoroethylene
ETFE	Ethylene-tetrafluoroethylene
PFA	Perfluoroalkoxy polymer
PTFE	Polytetrafluoroethylene
THV	Tetrafluoroethylene hexafluoropropylene vinylidene
PFPE	Perfluorinated polyether
PFSA	Perfluoro sebacic acid
PFPA	Perfluoropentanoic acid
PFNA	Perfluorononanoic acid
PFUDA	Perfluoroundecanoic acid
PFTDA	Perfluorotetradecanoic acid
MTV	Magnesium/Telfon/Viton
HT	High temperature incendiary
TMD	Theoretical maximum density
SEM	Scanning electron microscopy
TEM	Transmission electron microscopy
FTIR	Fourier transform infrared spectroscopy
FESEM	Field emission scanning electron microscopy
DSC	Differential scanning calorimetry
DTA	Differential thermal analysis
XRD	X-ray diffraction
IR	Infrared

to wider use of metal fuels in advanced energetic formulations. Fluorinated oxidizers may alleviate some of the above issues, mostly due to the combination of high heats of formation of metal fluorides, comparable to that of metal oxides, with the relatively high vapor pressure of metal fluorides and oxyfluorides, which volatilize more readily than refractory oxides for most of the energetically interesting metals.

Fluorine is the most electronegative element known, with the electronegativity of 3.98 on Pauling scale [24]. It is more reactive than most non-metals, and is therefore not found in nature in its elemental form. Its unique behavioral traits arise from multiple factors such as relatively small atomic size, strong electron affinity, weak molecular F-F bond, and weak polarizability. These characteristics can be combined in the concept of chemical hardness [25]. Calculated atomic radii and reported values of the chemical hardness are shown in Table 1. Indeed, fluorine is the hardest Lewis base of the typical oxidizing elements. The oxidation strength of fluorine-based species compared to other halogens in aqueous solutions is greater or, in some cases, comparable to chlorine [26].

The product of fluorination, the reaction where fluorine oxidizes a metal/metalloid and sometimes non-metals like oxygen, hydrogen and even inert gases like xenon, is primarily a fluoride. The enthalpy of formation of some fluorides and their corresponding oxides of some reactive metals are compared to each other in Fig. 1. The values are shown normalized by the number of metal ions (a) and by number of anions (b), respectively. Per mole of fuel consumed, formation enthalpies of metal fluorides always considerably exceed those of their respective oxides. However, due to the valence difference between fluorine (1) and oxygen (2), formation enthalpies per fluorine anion are on average about 50 kJ/mol less exothermic than per oxygen anion. Thus, deciding whether use of fluorine as oxidizer is thermodynamically advantageous depends on whether an application is limited by the amount of fuel or oxidizer. Further, the enthalpies of formation of binary fluorides exceed those of other halides for carbon [28,29], boron [30], all alkali metals [30], and even phosphorus (III) [30].

Table 1
Calculated atomic radii [27], and chemical hardness values [25] of some common oxidizing elements.

Element	Calculated atomic radius/Å	Chemical hardness/eV
Fluorine	0.42	7.01
Oxygen	0.48	6.08
Chlorine	0.79	4.68
Sulphur	0.88	4.14
Bromine	0.94	4.22
Iodine	1.15	3.69

In addition to thermodynamic considerations, formation rates are systematically different between fluorides and oxides. The rates of scaling metals by both fluorine and oxygen are limited by diffusion of the reactants through a growing layer of product forming at the fuel/oxidizer interface [31]. For example, it has been reported that the scaling of iron by fluorine gas has a low activation energy of 8.4 kJ/mol in the temperature range of 225–525 °C [31]. On the other hand, oxidation of iron in oxygen below 570 °C is reported to have a much higher activation energy ranging from 130 to 160 kJ/mol, depending on the crystal orientation [32]. The heterogeneous oxidation of iron remains slow even at much higher temperatures exceeding 900 °C [33].

For practical purposes, fluorine as an oxidizer is available from gaseous, polymeric and inorganic compounds. The dissociation energies of selected bonds in such compounds are presented in Table 2. Bond dissociation energies are expected to be a factor, influencing ignition in energetic composites. The bond dissociation energy of solid metal fluorides is slightly higher than the dissociation energies seen in fluorine-based gases like F_2 and SF_6, and comparable to that of C-C bonds in fluorocarbons. Compared to inorganic gases, the C-F bonds in fluorocarbons are more stable by about 100 kJ/mol, making the energetic cost of fluorine release slightly higher [32].

The bond energies given in Table 2 for metal fluorides and SF_6 are average values. Typically, fluorine atoms are dissociated one at a time, and the bond energies for the first and subsequent fluorine atoms removed from a fluoride are different. The bond energy of the first removed fluorine is likely to be the greatest, making it easier to cleave subsequently dissociated atoms. Thus, the values in Table 2 may serve only as a first approximation when assessing prospective ease of metal ignition using a metal fluoride as compared to fluorinated polymers as oxidizers. Note also that the bond energies in fluorocarbon polymers may be higher than shown in Table 2 due to the contribution of the other perfluorinated groups and chains attached to the carbon in question. However, in all fluorocarbons, the C-C bonds are comparatively easier to cleave compared to the C-F bonds, and therefore carbon-carbon dissociation often precedes release of fluorine during decomposition of carbon-based fluoropolymers.

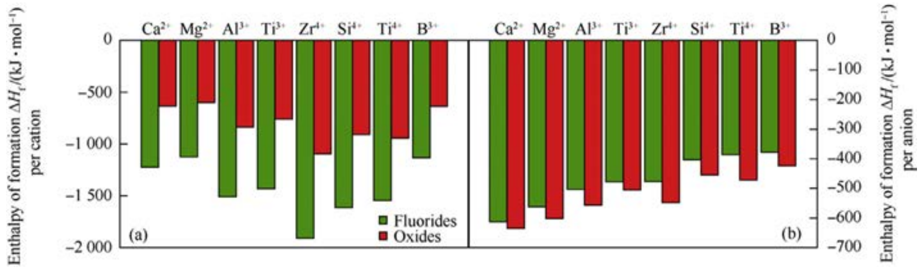

Fig. 1. Enthalpies of formation of selected fluorides and corresponding oxides of reactive metals.

Table 2
Bond energies of gas phase oxidizers, common fluorocarbon bonds and fluoride bonds of some prospective metal fluoride oxidizers.

Compound type	Compound	Bond	Dissociation energy/(kJ mol^{-1})	Reference
Inorganic gases	F_2	F-F	157	[34]
	SF_6	S-F	343	[34]
Fluorocarbons	CF_4	F_3C-F	547	[35]
	C_2F_6	F_5C_2-F	531	[35]
	C_2F_6	F_3C-CF_3	413	[36]
	C_3F_8	F_5C_2-CF_3	428	[35]
	C_2OF_6	F_3CO-CF_3	423	[35]
	CHF_3	F_3C-H	449	[37]
Solid metal fluorides	CuF_2	Cu-F	431	[34]
	CoF_2	Co-F	435	[34]
	BiF_3	Bi-F	259	[34]
	NiF_2	Ni-F	435	[34]

Potential advantages of fluorinated oxidizers with metal fuels may also be associated with the type of formed combustion products. The properties of metal fluorides formed as combustion products are quite different from their corresponding metal oxides. Table 3 contains relevant thermochemical and physical properties of some of the fluorides and oxides of metals used or considered for use in energetic formulations. The fluorides are usually more volatile than the corresponding oxides, and in some cases, the fluorides are gases at standard conditions. These cases include fluorides of practically important fuels boron and silicon: boron fluoride (BF_3) and silicon tetrafluoride (SiF_4). The semiconductor industry exploits this by using fluorine-rich gases, like SF_6, to etch silicon wafers removing the gaseous product SiF_4 [38]. Another important combustion product of aluminum, aluminum fluoride (AlF_3) sublimates readily [39] unlike the refractory Al_2O_3. In presence of oxygen, the oxy-fluorides (e.g., of boron and aluminum) may be formed, which are usually gaseous species as well. Thus, adding fluorine as an oxidizer for metal combustion makes it possible to generate more gaseous products and thus avoid or minimize the two-phase losses caused by the formation of condensed metal oxides [40,41]. At the same time, the effect on the energy released or flame temperature is relatively minor. For example, effects of using a fluorinated additive, polytetrafluoroethylene (PTFE or Teflon®), to an aluminized solid propellant with ammonium perchlorate (AP) as the main oxidizer and hydroxyl-terminated polybutadiene (HTBP) as a binder were considered in Ref. [42]. Calculated effect of pressure on the flame temperature for such a propellant is shown in Fig. 2. Sublimation temperatures for AlF_3 are shown for comparison. Adding PTFE reduces the adiabatic flame temperature slightly, however, the temperature remains much higher than the temperature of sublimation of AlF_3 for the entire range of pressures considered. Thus, gas products are generated,

Table 3
Physical and thermochemical properties [43] of metal fluorides and their oxide analogs.

Metal fuel	Fluoride combustion product				Oxide combustion product			
		Melting point/°C	Boiling point/°C	ΔH_f of metal/(kJ mol^{-1})		Melting point/°C	Boiling point/°C	ΔH_f of metal/(kJ mol^{-1})
Lithium	LiF	845	1717	−617	Li_2O	1438	2600	−299.5
Magnesium	MgF_2	1263	2262	−1124	MgO	2852	3600	−601.8
Calcium	CaF_2	1417	2484	−1229	CaO	2572	2850	−1207
Titanium	TiF_3	–	950	−1436	Ti_2O_3	2130	decomp.	−760.45
	TiF_4	283	–	−1649	TiO_2	1843	2972	−945
Zirconium	ZrF_3	1190	decomp.		–	–	–	
	ZrF_4	–	903	−1991	ZrO_2	2715	4300	−1097
Manganese	MnF_2	900	1820		MnO	1945	–	
Iron	FeF_3	–	926	−1039	Fe_2O_3	1565	decomp.	−411.1
	FeF_2	940	1800	−705.9	FeO	1377	3414	−272.04
Zinc	ZnF_2	872	1500		ZnO	1975	2360	
Boron	BF_3	−129	−100	−1137	B_2O_3	450	1860	−636
Aluminum	AlF_3	–	1275	−1510	Al_2O_3	2072	2977	−834.9
Silicon	SiF_4	−90.3	−86	−1615	SiO_2	1600	2230	−859.4
Tin	SnF_2	213	850		SnO	1080	–	
	SnF_4	–	701		SnO_2	1630	1800	

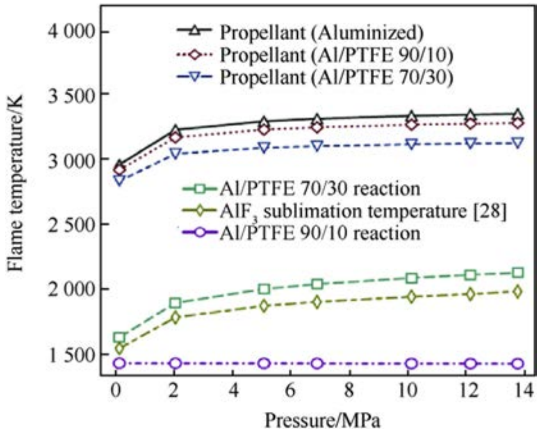

Fig. 2. Different formulation flame temperatures and aluminum fluoride sublimation [42].

whereas without PTFE, most of the products predicted to form in equilibrium are condensed oxides. Note that the AlF_3 sublimation temperatures are not attained for the 90% Al composition without the AP, while they are achieved if 70% Al react with 30% PTFE. Combining the AP with PTFE makes it relatively easy to custom tailor the flame temperature and reaction product makeup.

1.1. *Types of fluorinated oxidizers*

1.1.1. *Gases*

The use of elemental fluorine, as an oxidizer has long been considered in aerospace and aeronautic propulsion systems. Fluorine-liquid oxygen or FLOX, has been explored as an oxidizer in hybrid propellant engines for lithium based fuels [44]. It was also suggested to use FLOX with aluminized fuels in hybrid engines [45]. However, because of concerns over safety and handling procedures for extremely potent oxidizing agents like OF_2 and other active species, the studies on FLOX application were rather limited.

Fluorine based gases, which are relatively stable and even inert in most conditions, like SF_6, have been used as oxidizers for metallic fuels in laboratory experiments. Aluminum was found to burn in SF_6 environment without any condensed phase products [46]. In the same study, it was shown that for a broad range of ratios of oxygen to fluorine (present as SF_6), formation of oxy-fluorides, like AlF_2O, effectively reduces the formation of condensed Al_2O_3. Despite being non-toxic and easy to handle, SF_6 is not a practical oxidizer. It is not attractive thermodynamically because of its relatively high heat of formation. The presence of sulfur is also undesirable for most metal-based energetic systems.

Generally, gas oxidizers are less attractive for energetic formulations than condensed phases, capable of high packing densities, and, respectively, high energy densities.

1.1.2. *Fluorocarbons*

The discovery of polymers offered a stable, diverse, and safe group of fluorinated condensed phases, which could be incorporated into energetic formulations relatively readily. Although the mechanical properties of polymers were initially found to be conducive to use as binders, their utility as oxidizers was also appreciated early on. Table 4 has a list of commonly used fluoropolymers. Very few polymers in this list are perfluoropolymers, i.e., composed only of carbon and fluorine. The rest contain hydrogen or other halogens, like chlorine. The perfluoropolymers are of particular interest as they have greater fluorine content. Among different fluoropolymers, PTFE finds the widest range of applications in energetics. Table 5 lists some of the perfluorinated carboxylic acids used in energetics. These acids are large, linear molecules, which are completely fluorinated, save for the carboxylic groups at the chain ends.

In Fig. 3, the decomposition temperatures and fluorine content for materials introduced in Tables 4 and 5 are shown. The polymers are grouped into three quadrants; each quadrant could be associated with common applications. Polymers in quadrant (A) have higher carbon/low fluorine content combined with higher decomposition temperatures. As oxidizers, they typically react relatively slowly yielding carbonaceous products. This makes such polymers useful for applications in pyrotechnic formulations including various visible light and infrared obscurants and screens. Obscurants have been prepared with PCTFE [47], PVDF [48], and PMF [49]. Fluorocarbons in quadrant (C) have been used to coat reactive metallic powders to protect pyrophoric compositions [50] and to modify their combustion behavior [51,52]. They have been used as 'surface enhancers' due to high fluorine content and thus enhanced reactivity and hydrophobic properties; they are stable at room temperature but deteriorate as temperature rises [50,53]. These are primarily perfluorinated alkylcarboxylic acids. The polymers shown in quadrant (B) are most interesting for broader ranges of applications in energetics. They have both, high decomposition temperature and fluorine content. Due to higher fluorine content, reactivity is improved and gaseous products are readily produced. At the same time, the polymer oxidizers are robust, enabling longer storage and allowing for processing at elevated temperatures. In general, simple polymers in quadrant (B) may be tailored to perform as pyrolants [54], propellants [55] and explosives [56].

The fluorocarbons used thus far are conventional linear polymers/acids exploiting the exothermicity of the energy stored in C-F bonds. An extension of the energetic benefits, through strained structures was suggested by Koch [57]. Based on their enthalpy of combustion, as a measure of strained carbon skeletons and their hybridization and fractional electron transfer, as a measure of reactivity, several compounds were considered of interest. The reactivity is ranked highest for the most strained structures, fluorofulleranes. These fluorocarbons are prospective oxidizers that require further experimental focus.

1.1.3. *Metal fluorides*

Metal fluorides may be useful as stable solid fluorinated oxidizers although we were unable to find references to respective experimental or practical formulations aside from some very recent work [58,59]. The crystalline nature of these salts makes them stronger and more brittle compared to soft polymers. Thus, mixing metal fluorides and metals may be achieved more readily by mechanical milling, used recently for preparation of many metal-based reactive materials with attractive properties [59]. The properties of metal fluorides vary widely; some of the more stable and less hygroscopic materials, such as CuF_2, CoF_2, BiF_3 and NiF_2, may be more attractive as oxidizers than other fluorides, which are difficult to handle at ambient humidity.

Enthalpies of formation, normalized by mole of fluorine, for some potential metal fluoride oxidizers are shown in Fig. 4 along with that for PTFE. All metal fluorides carry a thermodynamic penalty as oxidizers, compared to PTFE. However, and as discussed above and shown in Table 3, the combustion products in thermite reactions with commonly used fuels, e.g., Al or B are fluorides and oxy-fluorides that are more volatile than oxides. This allows for large volume of gaseous products, comparable to that produced by the compositions with polymeric oxidizers. In addition, metal fluorides may be processed by powder methods, and the absence of carbon may account for different initiation reactions, and associated kinetics. Both issues will be discussed further below.

Table 4
Fluoropolymers common in energetic compositions.

Acronym	Full form	Formula
PCTFE	Polychlorotrifluoroethylene	-$(C_2ClF_3)_n$-
PVDF	Polyvinylidene fluoride	-$(C_2H_2F_2)_n$-
PVF	Polyvinyl fluoride	-$(C_2H_3F)_n$-
FEP	Fluorinated thylene-propylene polymer	-$(C_2F_4)_n$-$(C_3F_6)_m$-
ECTFE	Ethylene chlorotrofluoroethylene	-$(C_4H_4ClF_3)_n$
ETFE	Ethylene-tetrafluoroethylene	-$(C_4H_4F_4)_n$-
PFA	Perfluoroalkoxy polymer	-$(C_2F_4)_n$-$(C_2F_3OCF_3)_m$-
PTFE	Polytetrafluoroethylene	-$(C_2F_4)_n$
THV	Tetrafluoroethylene hexafluoropropylene vinylidene	-$(C_2F_4)_n$-$(C_3F_6)_m$-$(C_2H_2F_2)_o$-
PFPE	Perfluorinated polyether	-$(C_2F_4O)_n$-

Table 5
Perfluorinated alkylcarboxylic acids used in energetic compositions.

Acronym	Full form	Formula	Estimated chain length/Å
PFSA	Perfluoro sebacic acid	HOOC C_8F_{16}COOH	12
PFPA	Perfluoropentanoic acid	C_4F_9COOH	5
PFNA	Perfluorononanoic acid	C_8F_{19}COOH	10
PFUDA	Perfluoroundecanoic acid	$C_{10}F_{21}$COOH	12.6
PFTDA	Perfluorotetradecanoic acid	$C_{13}F_{27}$COOH	16.4

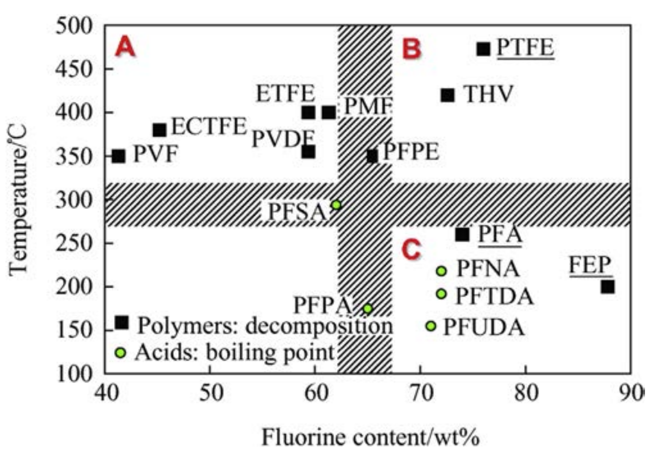

Fig. 3. Fluorinated polymers and acids based on their fluorine content and decomposition temperatures or boiling points.

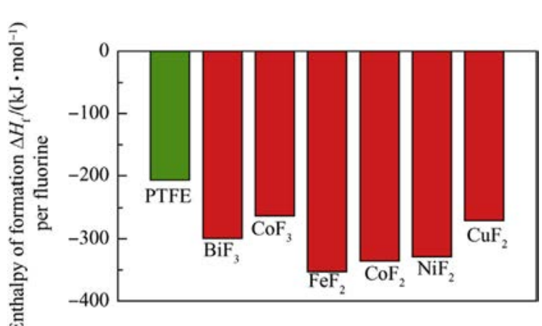

Fig. 4. Heats of formation of PTFE [60] and prospective metal fluoride oxidizers (from Ref. [43]).

1.2. Metal-based energetic systems with fluorine-containing oxidizers

1.2.1. Pyrotechnics

Pyrotechnic compositions are developed as igniters and initiators, including delay lines; they are also designed to generate specific gases, pressure profiles, high temperature, light, and sound effects. Energy density may be a less important metric for pyrotechnic formulations customized to generate optical emission with specific spectral range and duration, and achieve precise timing of the combustion event. Fluorinated compounds are used most extensively in this class of energetics. For example, magnesium-Teflon®-Viton (MTV) is a very common composition for various pyrotechnic formulations [61–63] as elaborately summarized by Koch in Ref. [64]. Metal-fluoropolymers constitute a very important type of pyrolants as reviewed in Ref. [65]. The majority of the compositions and materials contain PTFE polymer as the primary source of fluorine species. Table 6 provides a few examples of fluoropolymer based compositions used in pyrotechnics.

While metal-fluoropolymer compositions are used in practical pyrotechnics, current work remains active aimed at improving mixing and morphology of metal-fluoropolymer composites, characterizing their aging, as well as extending the range of time delays, temperatures, emission and pressure patterns achievable with such materials.

1.3. Propellants

Unlike pyrotechnics, most metalized propellant and explosive compositions including fluoropolymers remain experimental. For both propellants and explosives, energy density is of primary importance and thus additives, which might reduce the energy density, even if improving reaction rate and gas generation, may be unacceptable. For example, Mg/PTFE propellants have attractive burn rates, but their energy density is relatively low. Efforts are currently active to increase their energetic potential by adding boron-based thermites [77].

Materials for consideration as explosives and propellants include milled materials [78–81] and consolidated mixtures [82]. The constituent fuel powders are often micron sized spherical aluminum [56,78–82] and magnesium [79]. Additionally, nano-

Table 6
Examples of fluoropolymers used in pyrotechnic formulations.

Class of pyrolants	Materials
Agent defeat payloads	(High temperature incendiary) HTI-J-1000 (B/PTFE based) [66]
Countermeasure flares	Mg/PCTFE/Viton [67], MTV [68], RR81, RR-82 [69]
Incendiaries	Napalm substitute (Mg/Iron/PTFE based) [70], MTV [71]
Tracers	Pink tracer (MTV based) [72], small arms tracer (MTV based) [73]
Igniters	MTV [74]
Reactive fragments	RM4 (Al/PTFE based) [75], Ti/PTFE and Ta/THV [76]

metric aluminum (n-Al) powder has also found use in underwater explosive formulations [83,84]. The oxidizer of choice for such propellant/explosive systems has been micron sized Teflon® [56,78,80–82,85].

The milled materials have all been prepared using interrupted milling runs, to allow for intermittent cooling of the milled powders. Various compositions were tested; compositions with close to stoichiometric Al/PTFE ratio, approximately 30 wt% of Al, were found to have the highest flame velocities [85]. However, for propellants, very fuel-rich compositions with ca. 70 wt% of Al were favored [80,81] as the objective was to maximize the aluminum content enabling reaction with other oxidizers. Primary oxidizers, such as AP were added to employ PTFE as a secondary oxidizer/gas generator reacting with the aluminized fuel [80]. With added PTFE reduced agglomeration of condensed combustion products of an aluminized solid propellant was observed in Ref. [80]. This is illustrated in Fig. 5. Reference propellants used spherical Al, flake Al, and n-Al powders. For spherical Al, most of the particles become bright, and thus ignite, at a significant distance from the propellant surface. For flake Al, ignition appears to be closer to the propellant; however, most burning streaks are produced by very large, slowly burning droplets. In both cases, the thermal feedback from metal combustion to the propellant is negatively affected by the slow heat release at a distance from the propellant surface. Particles of n-Al ignite close to the propellant surface; however, the heat release is negatively affected by their relatively lower energy content, diminished because the surface oxide layer comprises a substantial volume fraction of the material [86]. With PTFE added, micron-sized aluminum particles are less agglomerated; they ignite and burn close to the propellant surface. This is expected to lead to an improved performance of the solid propellant. Several recent efforts considered solid propellants combining mechanically activated (ball milled) aluminum with PTFE [87,88], observing an increased burn rate without decrease in the propellant's energy density.

Boron is pursued as a replacement to aluminum in metal/fluoropolymer propellant compositions due to its high volumetric and gravimetric energy density. Primarily, PTFE was used as the polymer in almost all of these preparations. It was found by Young et al. [89], that the ignition and combustion behavior of boron was improved by addition of PTFE in experiments with heating rate of 10^5 K/s and various pressures to simulate propellant burning conditions. In a lab scale propellant experiment, linear regression rates of sintered and un-sintered powders in a diffusion flame were measured with gaseous oxygen as an added oxidizer. Sintered mixtures of B/PTFE, containing mass percentages greater than 25 wt% of boron were found to not combust at atmospheric pressures in the absence of pure oxygen. For un-sintered mixtures at the same condition, boron mass percentage could reach up to 30 wt% for combustion to occur [90]. The formation of gaseous BF_3 and hydrolyzed $B(OH)_3$ was confirmed through FTIR. At elevated pressures, the fuels tested could self-propagate without additional oxygen with up to 40 wt% boron loading due to improved porosity [91]. Several practical propellant experiments in hybrid rocket engines [92] and small-scale ramjet engines [93] were undertaken to test the B/PTFE fuel in solid propellants.

Boron has also been explored as an additive to fluorinated propellant formulations with another metal as a primary fuel. Studies with boron added to Mg/PTFE propellant compositions show the propellant ignited less readily though its overall combustion heat was improved [94]. A different study used pressed pellets of sonicated blends of milled Si and PTFE powders, in which silicon was doped with different amounts of boron. The apparent activation energy of the reaction decreased while the burn rates of the pellets increased with increase in the doped boron content [95].

1.3.1. Explosives

The PTFE-containing systems explored for potential use in explosives did not have any added oxidizers apart from PTFE but contained initiators like RDX and HMX representing less than 15 wt % of system [56,79,82]. The use of metal-fluoropolymer composites in explosives was also discussed in Refs. [96,97], where it was suggested that Al-PTFE mixtures subjected to high energy ball milling, consolidated, and initiated with a primer are capable of detonation. The observations of detonation-like propagation may be associated with release of gaseous combustion products in confined condition. In some experiments, the packing density was relatively low, in the range of 0.16–0.25 of the theoretical maximum density (TMD) [85], enabling gas evolution upon initiation. The materials with a higher aluminum content included over 50 wt% of aluminum and had very dense packing of around 85–95% of TMD [42,82]. Depending upon the composition and density of packing, very high flame velocities of 700–1300 m/s, referred to as detonation velocities for these experiments, have been observed

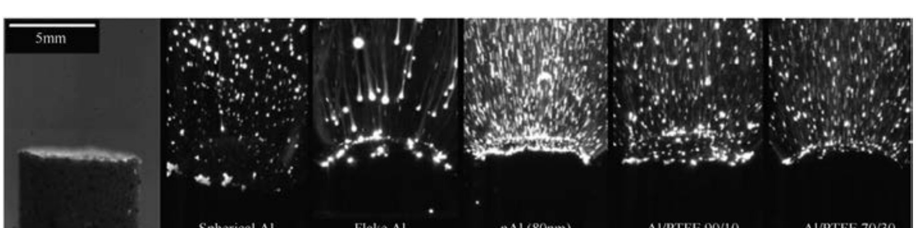

Fig. 5. Images solid propellant combustion with different aluminum-based additives, including PTFE [80].

[56]. Even in the case of powder mixtures, consolidated shapes were found to exhibit stable detonation when shock loaded. Mixtures of micron sized spherical powders, prepared to be slightly fuel-rich with compositions of 55/45 wt% of Teflon®/Al, were used in detonation experiments. These prepared powders were consolidated into shapes of various thickness and lengths. One such prepared shape is shown in Fig. 6 (marked "before") with a 30-mm cross section and about 30-mm length. These consolidated shapes had a high relative density with 93% TMD, and were initiated by a shock wave (caused by RDX or HMX boosters). For a densely packed sample initiated by a relatively small HMX booster (with HMX representing 10–15 wt% of the charge), the Teflon®/Al system displayed steady state detonation-like reactions with velocities of around 6.3 km/s [82]. The temperature was 2800 K for smaller cross-sections of 3 mm and smaller thickness of 6.4 mm, while it was reduced to 1800 K for greater cross-sections of 61 mm. The consolidated systems lose material as large chunks breaking off, as seen in the after detonation image in Fig. 6.

Despite the plausibility of detonation-like regimes observed for some compositions, further studies on combustion of bulk samples are desired. Detonation in a solid charge remains an elusive [98] but very interesting phenomenon, which, if confirmed, can find uses in a range of explosive systems.

1.3.2. Reactive structures

Some of the first materials designed to have dual use as structural components and sources of chemical energy, referred to as reactive materials or reactive structural materials, were prepared as Al-PTFE composites [99,100]. These compositions were developed further once n-Al became available [101], still relying extensively on fluoropolymers as binders capable of enhancing initiation and combustion of such materials. Prepared composites were initiated by impact, generating substantial pressure pulse, desired for applications. The density of such composites is relatively low, however. Respectively, more recent work focused on combining fluoropolymers with mixtures of metals, including titanium and high-density tungsten [102,103].

1.4. Current research: composites of metals with fluorinated oxidizers

The fuels used primarily in practical applications with fluorinated oxidizers are aluminum and magnesium. Boron and titanium are considered in related research. Silicon compositions have also been explored in some studies as surveyed in Ref. [104]. Different methods including sputtering, electro-spraying, sintering, mixing and ball milling are used to prepare composite materials combining metals with fluorinated oxidizers. Except for some very recent work [58,59], all such oxidizers have been polymers. Particle morphology and structures are studied using scanning and transmission electron microscopy methods (SEM and TEM). Depending on the preparation technique, the composite structures may contain varying interfaces between the metal fuel and fluoropolymer oxidizer. The nature and specific area of such interfaces is of critical importance for understanding both initiation and propagation reactions in such materials. The methods of composite preparation and the structures so prepared are briefly discussed below primarily for aluminum based systems due to the wide variety of research performed in aluminum-polymeric systems.

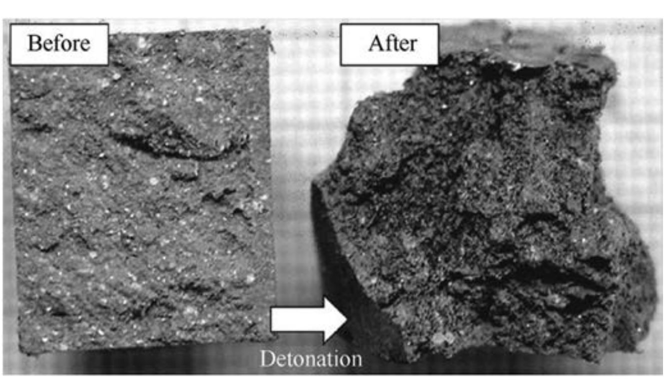

Fig. 6. Consolidated Al/PTFE mixture (45/55 wt%) with 30-mm cross section studied for their explosive behavior in shock loaded tubes, before and after experiment [82].

1.4.1. Mixed powders

The practical importance of mixed powders in energetic formulations is limited due to difficulty in processing which could cause the separation of components and therefore decrease mixing homogeneity. However, mixed powders are prepared easily for laboratory studies, aimed at initial understanding of more practical complex systems, such as laminates and consolidated composites.

The powders of aluminum preferred as fuel are usually spherical micron-sized or nanometric powders. Various sizes, e.g. 15 nm [105], 50 nm [105,106], 80 nm [105,107] as well as micrometric powders, 1–3 μm [105,106] have been employed. PTFE is the most explored fluorinated oxidizer and its powders typically have larger particle sizes, although a 200-nm powder is available from Dupont as Zonyl MP-1100 [105–107]. Compositions prepared were fuel rich, with 70 wt% [105,108] and 60 wt% [107] of aluminum.

The mixtures are prepared through sonication of powders in a liquid dispersant like hexane and then evaporating the dispersant [105,108]. Fluorine is separated from aluminum in these mixtures by two interfaces: aluminum-alumina and alumina-PTFE (or alumina-FeF_3 [58]). The amount of unoxidized or active aluminum maybe reduced noticeably for nanopowders, for which the particle size becomes comparable with the oxide layer thickness. The natural aluminum oxide thickness is hardly dependent on the particle size and varies around 2.7–3 nm [107]. This corresponds to about 90 wt% of active Al for 80-nm powder. Fig. 7(a) shows a characteristic image of n-Al powder mixed with nano-PTFE [108]. Fig. 7(b) shows SEM images of n-Al-PTFE prepared similarly but in a different study. The agglomeration in the n-Al powder is observable. The mixture seen in Fig. 7(b) shows PTFE particles compressed into non-spherical shapes. The two prepared powders, despite the same preparation technique have different structure, possibly due to difference in sonication intensity and duration, or simply due to difference in the PTFE powders used in the preparation.

The scale of mixing is relatively coarse and most of the surface of the fuel is only indirectly exposed to the oxidizer. This may be a concern, considering the gaseous nature of intermediates/decomposition products of PTFE, the primary oxidizing species [109], which may escape the system without interacting with the surface

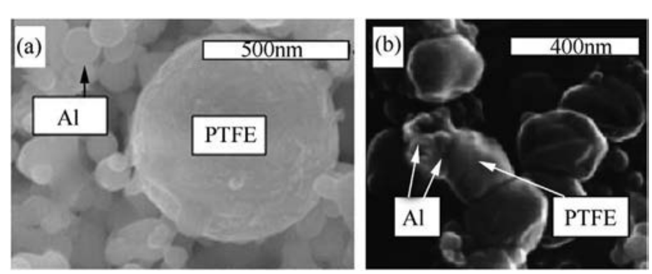

Fig. 7. (a) TEM image of a mixture of n-Al and micron-PTFE [108], (b) SEM image of a mixture of spherical 50-nm Al and 200-nm PTFE (Zonyl®) [106].

of the fuel powder at lower heating rates. In an experiment by Hobosyan et al. [109], where about 150 μm sized alumina powder was heated with PTFE (about 1- μm sized, by Sigma Aldrich) at various heating rates, an exothermic reaction was observed only at the rates above 150 K/min. However nanometric alumina powders (15–50 nm) with nanometric PTFE (200 nm) show exothermic reactions even at lower heating rates of 20 K/min [110]. Osborne et al. report that the nanometric powders show greater reactivity due to larger fuel surface area exposed to PTFE [105], which agrees with the difference in behavior between nanometric powders used by Pantoya and Dean [110] against the micron-sized powder studied by Hobosyan et al., [109].

Another popular method for preparing Si/Teflon/Viton, Al/Teflon/Viton mixtures is through shock-gel technique that involves the dissolution of the polymer in a low boiling ketone and mixing with Teflon and Si or Al and later precipitating the excess polymer through addition of non-polar solvent [111]. The blends are then extracted by drying the solvents. This method has long been used to prepare Mg/Teflon/Viton (MTV) powders as pointed out by Koch [65].

1.4.2. Core shell structures

Core-shell structures have been prepared with two major geometries: as coated spheres [50,51,112–115] and rods [116,117]. For both shapes, the core is commonly fuel and the shell is the polymeric oxidizer. The spherical core-shell structures are more common, however core-shell rods are used in the case of Mg-PTFE [116] and Si-PTFE [117] systems.

Table 7 summarizes different types of coated aluminized fuels prepared by various techniques. A broad range of polymers, such as PVDF [118] and PTFE [51], as well as perfluoroalkyl acids, such as PFTD (perfluorotetradecanoic acid) [113], PFS (perfluoro sebacic acid) [113], PFNA (perfluro-nonanoic acid) [50], PFTDA (perfluorotetradecanoic acid) [50], PFPA (perfluoropentanoic acid) [112], and PFUDA (perfluoroundecanoic acid) [50] have been used as coatings over aluminum powders.

The perfluroalkyl acid coats were intended to cover extremely pyrophoric freshly prepared oxide-less aluminum or n-Al powders, with a protective coating that does not hinder reactivity. These protective coatings could serve as an oxidizer due to fluorinated species that accelerate particle ignition unlike the naturally occurring aluminum oxide layer. Acids form coatings via surface reactions on freshly prepared n-Al particles in solutions; the obtained coatings are described as self-assembled monolayers [50]. The particles are in the size range of 20–150 nm and have a thin coat of the PFTD. The particles are spherical and seemingly agglomerated as seen in Fig. 8. It has been suggested that the acid's carboxyl group bonds to the aluminum particles as illustrated in Fig. 9. These bonding structures have been proposed by studying the spectral modes observed for (COO) groups in Attenuated Total Reflection-FTIR spectra for Al coated by PFTD contrasted against pure PFTD spectra. Unfortunately, due to control issues innate in

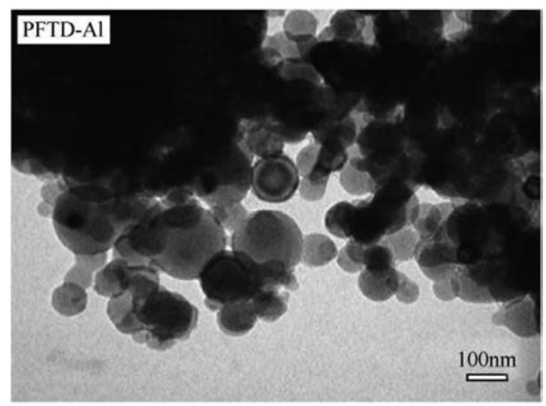

Fig. 8. TEM image of PFTD-coated bare aluminum particles [50].

Table 7
Coated powders prepared by various methods along with their compositions, interfaces and fuel sizes.

System	Fuel size/nm	Interfaces	Composition	Preparation	Ref
Polymer coats					
Al-PVDF (Kynar®)	50	Al/Al_2O_3/PVDF	5, 10 and 15 wt% PVDF	Electrospraying	[114]
Al-PTFE	100	Al/PTFE	Average Al size: 100 nm; average coating thickness: 10 nm calculated thickness for the exp. procedure: 34 nm.	In-situ radical-vapor deposition:freshly prepared aluminum (wire explosion), surface coated through cracking hexafluoropropylene oxide yielding CF_2 radicals that form a nano-film of PTFE	[51]
Al-PFPE (Fomblin®)	80	Al/Al_2O_3/PFPE	70 wt% PFPE	Wetting:described in Ref. [121]	[115]
Al-PFPE (Fomblin®)	80 100 120 5500	Al/Al_2O_3/PFPE	66.9 wt% PFPE 67.4 wt% PFPE 67.8 wt% PFPE 69.8 wt% PFPE	Co-solvent adsorption: Suspensions of Al with PFPE along with volatile dispersant PFS-2 mixed in planetary mixer for 3 min s.	[52]
Perfluoroalkyl-acid coats					
Al-PFTD	80	Al/Al_2O_3/perfluoroalkyl-acid	35 wt% PFTD	Co-solvent adsorption: prepared in a slurry of Al and respective fluoro-acids dispersed in diethyl ether and washed.	[113]
Al-PFS	80	Al/Al_2O_3/perfluoroalkyl-acid	35 wt% PFS		
Al-PFPA	~90	Al/PFPA	1–2 nm coat of PFPA	In-situ aerosol coating: aluminum prepared from precursor solution, aerosolized and coated by gaseous PFPA	[112]
Al-PFNA Al-PFTDA		Al/perfluoroalkyl-acid	N/A 15.4% aluminum left after in-situ chemisorption	In-situ chemi-sorption in solution phase: n-Al prepared by catalytic reaction is arrested and coated by respective acids added drop wise into solution.	[50]
Al-PFUDA			N/A		

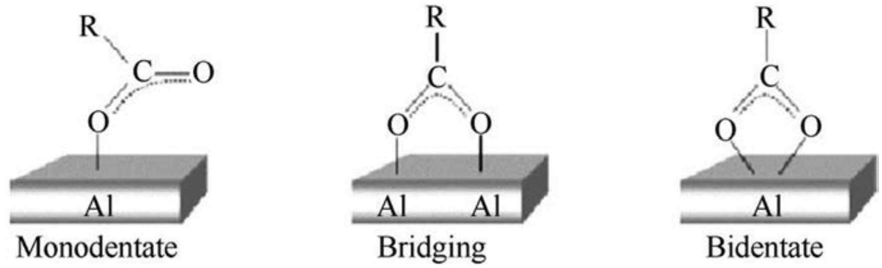

Fig. 9. Proposed carboxylate binding to aluminum surface in acid coating of aluminum powders [50].

solution state preparation involving acidic constituents in these samples, it was found that n-Al powders coated with PFTD had only 15 wt% of active aluminum, much lower than expected in the natural oxide coated n-Al. This issue is crippling and needs to be addressed by involving different control agents during synthesis. The powder properties, such as flowability were also impacted by choice of acid for the surface coating. The PFNA acid prepared composites were tar-like while the rest were better flowing powders.

The limitation of a solution-based synthesis has been addressed in a study relying on an aerosol synthesis technique [112]. This technique has largely been applied for polymeric coatings. A schematic diagram of the experimental setup is shown in Fig. 10. The aluminum precursor solution of tri-iso-butylaluminum is flushed by argon into a tubular furnace at 350 °C, which causes the pyrolysis of the precursor to yield an oxide-free n-Al. This particulate aerosol is then coated by PFPA vapors. This aerosol containing coated aluminum composites are cooled and collected. This synthesis method enables better control of coating through adjustable flow rates and use of inert media, argon, to transport and disperse constituents. The powders so obtained are interestingly of different shape; more polyhedral and less spherical. A TEM image in the inset in Fig. 10 shows a polyhedral structure of 100-nm particles, with a 1–2 nm thick coating of PFPA. This coating is thinner than the oxide coat growing naturally on n-Al prepared by the same method [112]. Thus, prepared PFPA-coated nanometric powders have a higher percentage of active aluminum. With 80% active aluminum for powders of about 90 nm diameter [112], the PFPA-coated powders outperform n-Al with 50 nm particle size and natural oxide layer, with ca. 70% active aluminum [119].

The combustion behavior of aluminum particles was affected differently by surface coats of different fluorinated substances. The co-solvent dispersed and perfluoroalkyl acid coated aluminum particles were found to burn with different velocities when used in thermite mixtures. Al-PFTD/MoO_3, Al-PFS/MoO_3, and control Al/MoO_3 thermite blends were tested in a custom built flame tube packed at 7% TMD. The experimental setup for the flame tube may be found elsewhere [120]. The Al-PFS/MoO_3 thermite blend had a low flame velocity, half of that for the control uncoated thermite, while Al-PFTD/MoO_3 demonstrated flame velocities twice as high as the control thermite. Note that as further discussed below, the flame velocity measured in such experiments optically is affected by multiple factors, including rate of gas release and flowability of the powders. In some cases, the measured velocities can be misleading because the visible flame front can be confused with the emission produced by advection of burning particles [22].

For thermite blends prepared with coated aluminum powders, the polymeric coats were reported to alter the combustion behavior depending on the major oxidizer used. In the case of CuO/Al (PFPE coated) blend, the coating impeded the reactivity while the reactivity was improved in the case of MoO_3/Al (PFPE coated) blend [52]. This was suggested to be due to thermodynamically costlier bond breakage of the polymer, to initiate reaction between CuO and aluminum fuel in the CuO/Al (PFPE coated) blend [52].

Polymeric materials, such as PTFE, have been coated onto the surface of aluminum through vapor deposition [51]. Freshly prepared n-Al was exposed to CF_2 radicals to achieve a continuous coating. The preparation setup involved n-Al prepared by the electro-exploded wire method in inert atmosphere. The powder was sieved in inert conditions, during preparation, to procure desirable sizes and then coated by CF_2 radicals, prepared by the decomposition of hexafluoropropylene (CF_3CFOCF_2). The radicals were deposited and not reacted with aluminum due to a careful temperature control of deposition surfaces maintaining aluminum at 25–30 °C. The progress of the coating process is illustrated in the field emission SEM images Fig. 11(a) and Fig. 11(b), which were taken with a 5 min interval between them. The surface has a localized deposition of the radicals, aggregations of which are

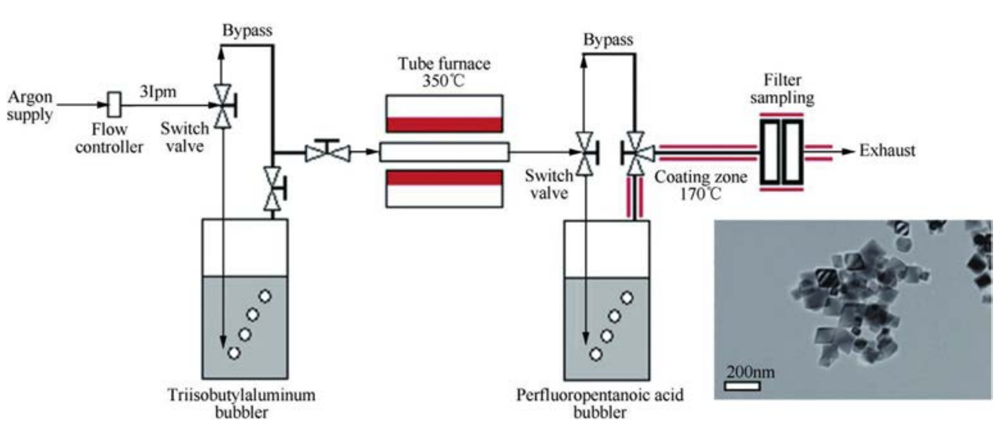

Fig. 10. Experimental setup for aerosol coating of perfluoropentanoic acid (PFPA) on freshly prepared Al; the inset shows a TEM image of coated aluminum particles [112].

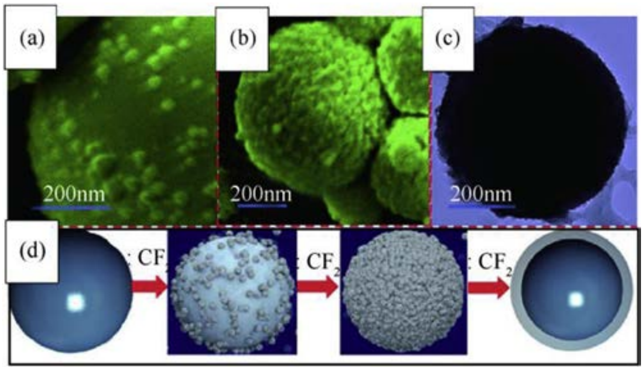

Fig. 11. FESEM images of Al coated by CF_2 by in-situ vapor deposition, (a) after 3 min and (b) after 8 min (c) TEM of sub-micron coated aluminum particle. (d) Schematic of core-shell formation model showing gradual development of nano-film of PTFE on aluminum surface [51].

visible as rough amorphous perturbations on the smooth aluminum surface. The completely coated particles were studied using TEM, as shown in Fig. 11(c). The coating on the surface was found to be homogenous and uniform with a thickness of about 10 nm. The deposited radical (CF_2) layer has an F/C ratio comparable to that of PTFE, and the CF_2 bonds are confirmed through FTIR studies [51]. The evolution of the deposited radicals into a thin film is schematically shown in Fig. 11(d).

In the vapor deposition technique, only a limited control of the coating thickness is possible using its dependence on the aluminum particle exposure time to radical vapor. Thickness of polymeric coats prepared through electro-spraying is controlled more readily [114]. However, it is unlikely that individual nanoparticles can be coated; instead, particle agglomerates, or mesoparticles can be assembled, which could have attractive combustion properties, as was shown with similar systems, although not involving fluorinated polymers as binders for n-Al [122].

A complex nano sized array of rod shaped Mg/PTFE core-shell structures has been prepared by Zhou et al., [116]. First, Mg nanorods were prepared by glancing angle deposition of magnesium vapors. The nanorod arrays were encased in fluoropolymer through magnetron sputtering deposition. These polymer shells were found to have shorter molecular chains than bulk PTFE. Similarly nano/micron sized rod arrays of Si were prepared by deep reactive ion etching method and coated with PTFE via sputtering by the same group to achieve Si/PTFE core-shell structures [117].

Coated systems may be explored to have larger relevance in energetics apart from mere atmospheric aging/oxidation protection and combustion/ignition modification. The established hydrophobic nature of perfluorinated polymers could be exploited for humidity/chemical shields and extending powder use in complex chemical environments without loss of potency. It is found, for example, that the coated aluminum powders were hydrophobic to a point where deionized water had a contact angle of 118° for a 30-nm thick coating [51]. Similar behavior of superhydrophobicity was observed for the core-shell rod shaped structures of Mg/PTFE and Si/PTFE systems [116,117]. For aluminum, it was shown that core-shell structures improve its stability to bases. Both regular aluminum powder and aluminum core-shell structures with PTFE coating thickness corresponding to stoichiometric composition, were exposed to NaOH solution. The dissolution of the core-shell structure occurred 8.6 times slower than for the uncoated powder [51]. Thus, by choice of a polymer and method of applying the coat, the powder properties may be tuned potentially to a wide variety of behaviors.

1.5. Laminated/layered systems

Layered or laminated systems are high reactive-interface systems that recently have attracted substantial interest. Based on their intended applications, these may be considered reactive structures (discussed separately below); however due to relatively small scales most such materials are prepared on, and because of their well-defined interface suitable for fundamental studies of reaction mechanisms, they are discussed in this separate section. Table 8 describes some of the prepared layered/laminated systems including their preparation techniques, structure and composition. All materials use aluminum as a fuel.

A polymeric-aluminum homogeneous system was prepared through electro-spraying [123]. The preparation involved using PVDF and nanometric-aluminum powder with very little ammonium perchlorate (AP) mixed in a DMF solution dispersant and electro-sprayed onto a negatively charged rotating drum substrate from positively charged nozzles to achieve the desired thickness of the film/laminate. An SEM image of a cross-section of the highest loaded, 50 wt% aluminum-PVDF film is shown in Fig. 12(a). PVDF forms a polymeric network and a matrix including spherical aluminum particles. The mechanical properties of such composites are a function of n-Al particle loading, where the loading of 17 wt% showed an increase in tensile strength from 18 MPa for an unloaded polymer sheet to 24 MPa. The toughness increases likewise [123] as observed for other particulate additions in a polymeric matrix [124]. An increased particle loading caused formation of agglomerates, leading to an increased porosity and some deterioration of mechanical properties; nevertheless 50 wt% Al-PVDF films were non-brittle and could be deformed and flexed [125]. The films had a uniform thickness of about 175 μm.

Similar laminates with a sandwich-like structural arrangement with alternating PVDF spacers and layers of nano-thermite comprising n-Al-CuO particles in PVDF matrix have also been prepared through electro-spraying method [126]. Based on flame front propagation velocity in these systems, it was found that the systems with PVDF spacers and a layer of nano-Al-CuO particles in PVDF matrix were superior to those with n-Al-CuO particles uniformly distributed in PVDF [126]. Having several such alternating layers improved the rate further [126]. An SEM image of a cross-section of such a multi-layer system shown in Fig. 12(b) reveals a homogenous n-Al-CuO mixture in PVDF and amorphous PVDF layers with thickness varied around 5 μm.

These spray coated strips/films have burn times of the same order of magnitude as those of a loose powder blend of n-Al − n-PTFE in an open air configuration observed in Ref. [106]. Higher flame propagation rates exceeding 1200 m/s were observed in Al-PTFE pressed nanocomposite layers [111]. However, the 6-layer sandwich laminate structure prepared in Ref. [126] having thickness of 111 μm, width of 0.5 cm, and length of 2.5 cm exhibited a maximum burn rate of 9.5 cm/s. The strips were placed in argon and resistively heated at an end by Ni-Cr wire. It was found that for multi-layered thermite-like laminates, the burn rates fall with laminate thickness and for a given thickness, increase with number of layers present. For the purely polymeric and aluminized laminates such as n-Al − PVDF-AP [123], the burn rates increase with nano-aluminum particle loading. At 50 wt% aluminum loading, the burn rate was 23 cm/s when ignited in atmospheric conditions.

The super-laminate or super-lattice structures prepared using magnetron-sputtering technique [123] are dimensionally different from other laminates. Vacuum-deposited layers with a stoichiometric Al/PTFE ratio with 10-nm layers of Al alternating with 15-nm layers of PTFE were prepared as shown in Fig. 13. The entire structure consisted of a sub-micron sized Al-PTFE super-laminate upon Au/Pt/Cr films (800/120/20 nm) fabricated on a silicon

Table 8
Different laminated reactive material systems, their preparation techniques, interfaces and compositions.

System	Fuel size/nm	Interface	Structure (composition)	Preparation	Ref
nAl-PVDF-AP	50	Al/Al_2O_3/PVDF or Al/Al_2O_3/AP	Strips 50, 70 and 83.3 wt% PVDF and 2 wt% AP	Electrospray: Intimately mixed/sonicated mixture in DMF, electrosprayed onto rotating drum substrate.	[123]
(nAl-nCuO-PVDF)-PVDF-AP	50	Al/Al_2O_3/PVDF or Al/Al_2O_3/CuO	Sandwich laminate structure alternating nAl-nCuO-PVDF and PVDF,32 wt% CuO and 42 wt% PVDF, 1 wt% AP		[126]
(nAl)-PTFE	~10	Al/PTFE	Laminate structure with alternating Al layer of 10 nm thickness and PTFE layer of 15 nm thickness, about 70 wt% PTFE	Magnetron sputtering: alternating layers of PTFE and Al onto 500-μm thick Si substrate	[123]

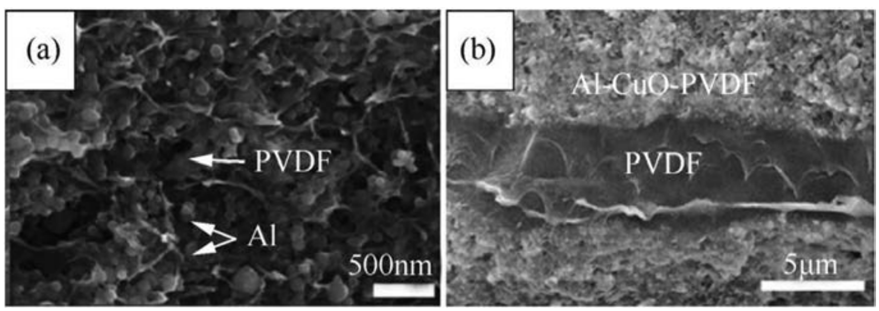

Fig. 12. SEM image of (a) the cross-section 50 wt% Al-PVDF electro-sprayed film [123] and (b) the cross-section of a multilayered, n-Al-CuO-PVDF with PVDF spacers [126].

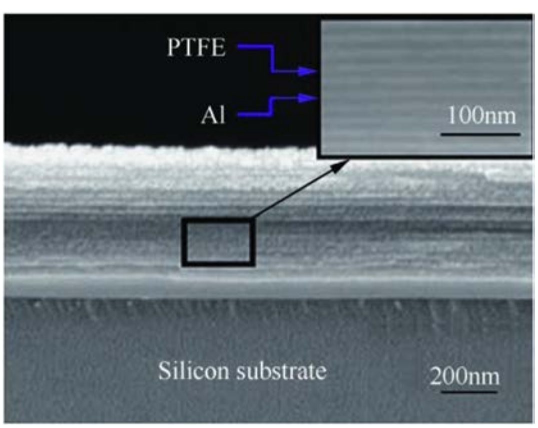

Fig. 13. SEM image of cross section of a n-Al/PTFE super-laminate (super-lattice) system with 10 and 15-nm thick layers of alternating Al and PTFE respectively [123].

substrate of 500 μm. The structure was 5 μm wide and could sustain a self-propagated reaction. When a 10-μm thick 30-mm long strip was ignited by a match in atmospheric conditions, it burned at a rate of 1.5 m/s [123].

Like coated powders, the laminated structures have interesting hydrophobic properties. It was found that laminate structures with polymeric layers of PTFE along with Mg and CuO layers retained 82% of their chemical energy after 240 h of exposure at a temperature of 35 °C in 95% relative humidity accelerated aging test [127]. The same laminate sandwich structure was found to have retained over 50% of its chemical energy after 6-hour underwater storage [127]. The water contact angles were found to be very high, 153° + indicative of a super-hydrophobic surface [127]. These properties may be useful for storage and diverse applications.

1.5.1. Ball milled composites

Milled composites provide one of the most practical and widely utilized powder systems that can achieve homogenous dispersion in each particle's bulk. They also have superior combustion properties compared with mixtures of similar sizes and show improvement in burn rates and ignition kinetics without employing nanometric powders that have processing difficulties. Powder morphology can be tuned through varying parameters, such as milling time, milling media utilized, (glass, steel, etc) and the ball-to-powder mass ratio. The effect of different milling parameters can be summarily described through a single term, the milling dose D_m, which is defined as the energy transferred from the milling tools to the powder [128–131].

Al-PTFE composites have been prepared by milling at both room and cryogenic temperatures [81,132]. Room temperature-milled, fuel-rich Al-PTFE milled composites are close in morphology to conventional aluminum flakes as seen in Fig. 14(a). The material shown was prepared in a shaker mill with a milling time of 1 h. For longer milling times, e.g., 2 h in a shaker mill, the flakes begin to agglomerate forming more equiaxial powder. For both 1 and 2- h milled powders, the particles contain a nearly fully-dense homogenous mix of the polymer and metal fuel, as established by SEM and energy-dispersive x-ray spectroscopy. One major point of difference between regular aluminum flakes and milled material is that the commercial flakes are covered with a natural oxide layer;

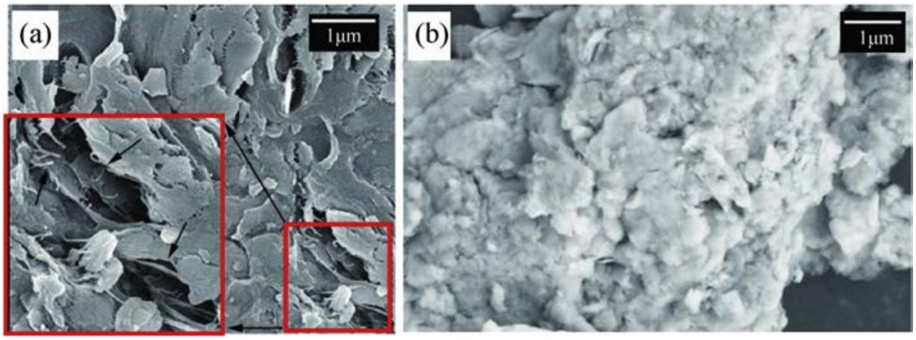

Fig. 14. SEM images of structural features observed in Al-PTFE system; (a) Composites with 70 wt% Al prepared by room-temperature milling [81] and (b) Composites with 90 wt% Al prepared by cryo-milling [132].

they also often have an additional organic coating. In the milled particles, the oxide-coated surface of the starting aluminum particles is sheared off and dispersed in the bulk of the milled material, while the newly formed flake surfaces are coated with PTFE before they oxidize.

A closer look at room-temperature milled metal-polymer composite shows fibrous strands in lower micron scale infused into the aluminum matrix (black arrows in the inset in Fig. 14(a)). The polymer losing its structural and bulk properties; its crystallinity drastically reduces to a point where it is not observable clearly by XRD.

Al-PTFE powders with similar morphologies were prepared using different milling equipment [132]. The milling dose was maintained approximately the same for both shaker and attritor mills, although the milling period and ball to powder mass ratio were different. Milling for 2 h in the shaker mill corresponded to a 6- h milling in the attritor mill. Samples prepared by milling at liquid nitrogen temperature (cryomilled) in the attritor had smaller particle sizes and similar surface morphology, as shown in Fig. 14(b) [132]. The cryogenically milled material was found to have better dispersion of PTFE in the bulk of the particles compared to that prepared by room-temperature milling. It is interesting that although longer periods of cryomilling produced equiaxial particles, those particles were found to be partially reacted during milling and, thus, unattractive as reactive material components.

A unique 2 stage milling procedure involving both room temperature milling and cryomilling was used to prepare various fuel rich compositions of Si/PTFE composites by Terry et al., [133]. The mixed powders were cryomilled for 6 cycles of 1 min milling spaced by a minute of rest and once the sample was restored to room temperature, it was milled again in a shaker mill for 20 cycles (1 min on and 1 min off). These composites were found to have improved combustion enthalpies compared to mixed powders with relatively moderate surface area and improved dispersion of constituent powders.

1.5.2. Consolidated shapes for explosives and reactive structural materials

Different methods have been used to prepare consolidated shapes of metal-fluoropolymer composites. These techniques commonly involve mixtures or composites, which are then molded, cured or sintered into consolidated forms.

Consolidated materials discussed in the literature, including samples prepared using in-situ polymerization [134], sintering [90,135], pressing [91,95,111,136] and curing in a mold [42], are summarized in Table 9 along with details on their respective interfaces, structures and particle sizes.

The techniques involving a mold are some of the best established, and typically are used to make propellants. The typical preparation has mixture of components: a binder, like HTPB (hydroxyl-terminated poly butadiene), AP oxidizer, and a fuel, a metal-rich Al-PTFE milled composite [42]. This molded composite strand is allowed to cure at a slightly elevated temperature for an extended period to dry into a well-packed integrated structure. Consolidated sticks prepared in this way are shown in Figs. 5 and 6. In such consolidated structures, the actual concentration of fluorinated oxidizer is quite low; it is added as a secondary oxidizer/gas generator that helps reducing aluminum agglomeration as illustrated in Fig. 5. It was found that the agglomerates were reduced by 66% by diameter and 96% by volume [42].

Milled composites of Al-PTFE with 70 wt% of aluminum were compacted in a $25 \times 25\ mm^2$ die by a hydraulic press at 34.5 MPa [136]. Al-PTFE system with a similar composition (74 wt% Al and micron sized powders) but consisting of a mixed powder, was consolidated at higher pressures of 72–182 MPa [137]. These 'foils' or pressed structures with milled composites were intended as ignitor fibers to tailor burn rates of solid propellants [136]. The inclusion of a fluoropolymer as an oxidizer contributed efficiently to the overall gas generation and energetic output of the ignitor-propellant system as compared to other systems consisting of similar fibers made of Al-Ni and Pyrofuse® [136].

The sintering technique employed to make pellets used thermal treatment after pressing. In one study, consolidated pellets were prepared with micron sized, mixed powders of Al-PTFE and then sintered at elevated temperatures and cooled at controlled rates [137]. SEM images of the cross-sections of pellets prepared with different sintering temperatures and cooling rates are shown in Fig. 15 (a) and (b). PTFE was more crystalline at a lower cooling rate (Fig. 15(a)), while the sample cooled more rapidly contained amorphous PTFE, see Fig. 15(b).

Another technique preparing aluminized fluorinated acrylic (AlFA) composites involves in-situ polymerization and surface activation of 80-nm aluminum powder yielding composites that have mechanical integrity while retaining their reactive properties [134]. This technique yielded composites with a high metal loading exceeding 60 wt% of Al. The process develops the per-fluorinated material which is attached to aluminum powders through surface modifications [134]. Fig. 16 schematically shows the initially treated aluminum powders with surfaces activated with phosphate groups, which are then involved in polymerization process to make the aluminized fluorinated acrylic composites. Even for the high aluminum loading of 70 wt%, the melting temperature of the composites remains low at about 85 °C. These composites can be processed and cast into various shapes due to the pliability and malleability of the materials. They retain their thermoplastic behavior (due to low melting temperature) and machinability (due

Table 9
Consolidated aluminum-fluoropolymer structures and respective details of fuel size, interface and structure along with preparation technique.

System	Fuel size/ μm	Interface	Structure (composition)	Preparation technique	Ref.
nAl-fluorinated acrylic composite	0.08	Al/Al_2O_3/ fluorinated acrylic polymer	Strands 90, 70, 50 and 40 wt% of polymer	In-situ polymerization: Al particles surface functionalized and added into polymerization process	[134]
Al-PTFE	6–7	Al/Al_2O_3/PTFE	Cylindrical pellets 74 wt% PTFE	Sintering: powders were mixed in ethanol dispersant, cold pressed into shape at 60 MPa and sintered at temperatures of around 350 °C and cooled at controlled rates of about 70° C/h	[135]
Al-PTFE-HTPB-AP	35	Al-PTFE or Al/AP	Cylindrical sticks-6 cm long and 5.8 mm diameter 71 wt% AP 14 wt% HTPB 15 wt% Al-PTFE composite (composite has 90 or 70 wt% Al)	Curing in mold: composites prepared by milling are added with binder and oxidizer in mentioned ratios into a mold and cured at 60 °C for approximately 7 days.	[42]
Al-PTFE	6–7	Al/Al_2O_3/PTFE	Cylindrical pellets 26 wt% PTFE	Press: the mixture was blended and sonicated in ethanol and sieved. If composite was used, it was prepared by milling and sieved.	[137]
Al-PTFE	50	Al/PTFE	Foil/laminate structure of Al-PTFE composite 30 wt% PTFE	The sieved powder was then pressed in a mold with pressures ranging from 30 – 182 MPa	[136]

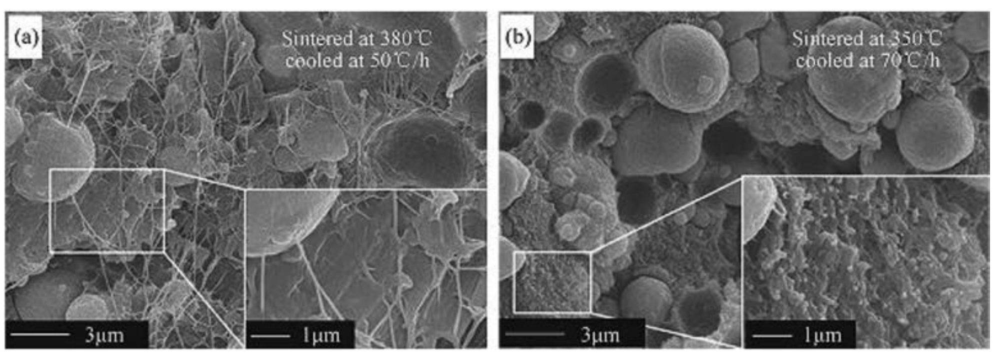

Fig. 15. SEM cross sections of Al-PTFE sintered pellets prepared by (a) sintering at 380 °C and cooled at 50 °C/h rate and (b) sintering at 350 °C and cooled at 70 °C/h [137].

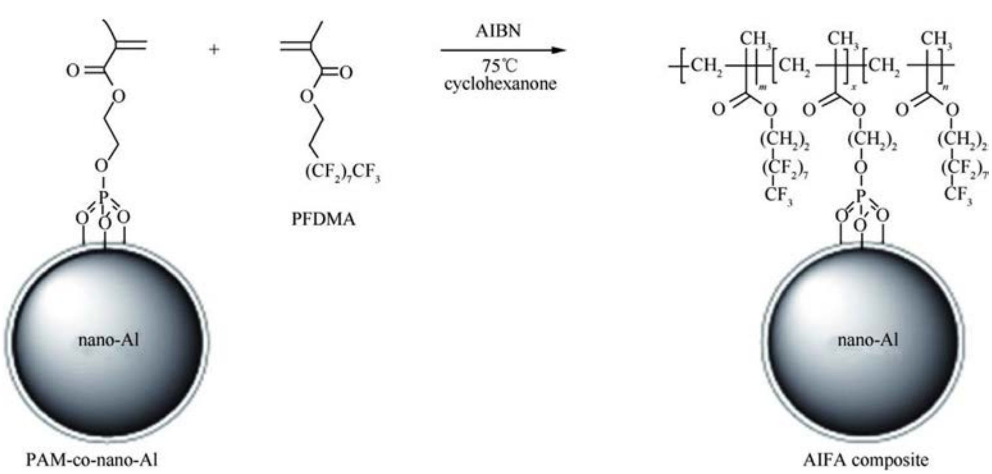

Fig. 16. Schematic structure of aluminized fluorinated acrylic (AlFA) composites [134].

to structural integrity) without compromising the reactivity. Composite pellets with aluminum weight percentage of 30 or higher develop a self-sustained reaction upon ignition. The presence of large amounts of polymeric material (30–70 wt%), the composite yields copious amounts of smoke and charred residue, aluminum fluoride and minor amounts of carbide and oxide of aluminum. It was found that the most energetic composition contained 50 wt% of aluminum.

1.6. Reactions in metal-based reactive materials with fluorinated oxidizers

1.6.1. Thermo-analytical measurements

Thermal analysis has been widely employed across all preparations to characterize material performance, reactivity and, sometimes, even composition. DSC and DTA plots for several Al-PTFE systems prepared by different methods are combined in Fig. 17. All materials show substantial exothermic reactions occurring prior to the aluminum melting; for most composites these exothermic reactions are clearly separated into at least two steps. The magnitudes of the individual exothermic peaks and the temperatures at which they occur are both affected by the preparation and thus material structure.

Results for a sonicated blend prepared using nano-sized powders [105] are shown in Fig. 17(a). The weak endothermic peak slightly above 600 K is due to melting of PTFE. Two exothermic peaks are clearly visible and well separated. The first peak occurs between 675 and 725 K, and the second, stronger exotherm is observed around 825 K. Blending the micron-sized powders makes the peaks much weaker or even undetectable [88]. However, at higher heating rates, the exothermic peaks are clearly observed even for micron-size powder blends [109]. A qualitatively similar two-peak exothermic pattern is observed for different Al-PTFE composites prepared by mechanical milling, Fig. 17(b) [88,132]. For the materials prepared by room-temperature milling, the first peak shifts to higher temperatures; however, for the material prepared by cryomilling, it occurs at a lower temperature, before 670 K. The position and strength of the second peak are also affected, while the difference between the heat effects in the first and second peaks is generally smaller for the milled materials compared to the sonicated nanopowders.

Results for the vapor coated powders [51] and a super-laminate structure [123] are given in Fig. 17(c) and (d), respectively. In both cases, the traces are remarkably similar to each other. The two exothermic events are nearly overlapping; the first exotherms begin at about the same temperature as for blended composites.

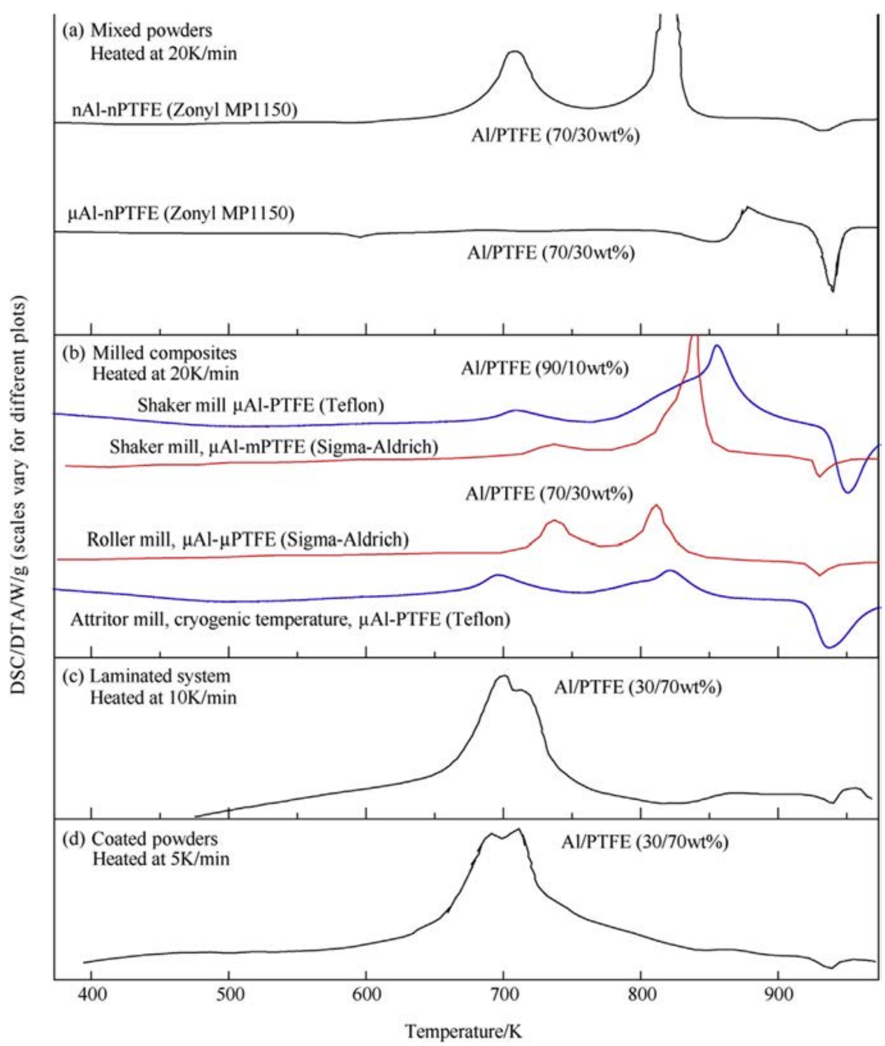

Fig. 17. Al-PTFE preparations thermally analyzed in argon (a) DSC plots (20 K/min) of different Al-PTFE (70/30) sonicated blend preparations [81,105]. (b) DSC plot (20 K/min) of milled Al-PTFE (70/30) [81] and DTA plot cryo-milled (90/10) composites [132]. (c) DSC plot (5 K/min) of 30 nm PTFE coated n-Al powder [51]. (d) DSC plot (10 K/min) of Al-PTFE (30/70) super-laminate structure [123].

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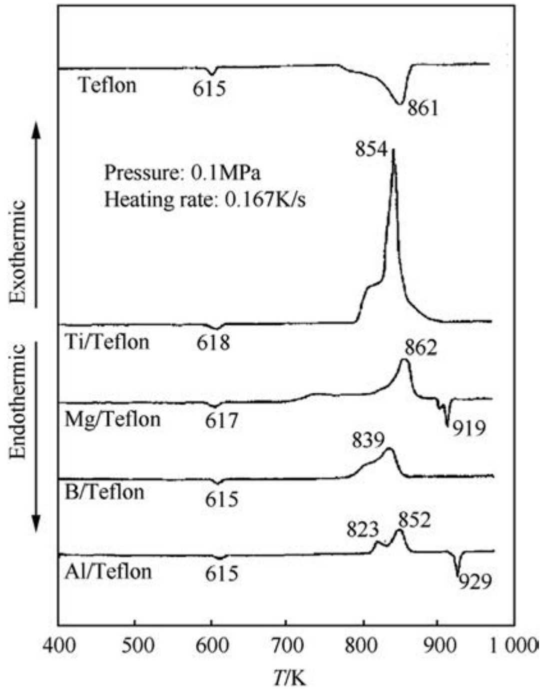

Fig. 18. DSC plots for various metal/Teflon based reactive systems in helium atmosphere at heating of 0.167 K/s and 0.1 MPa pressure [138].

However, the first exotherm is only slightly ahead of the second one; the heat effect is similar for both exotherms for these materials. It is hypothesized that the overlap between the exothermic events is associated with a very fine scale of mixing between Al and PTFE for these materials. A very thin layer of PTFE compromised by the low-temperature reaction decomposes more readily, releasing fluorine and shifting the second exothermic step to lower temperatures.

Fig. 18 summarizes the different metal-PTFE systems thermally studied by Kuwahara et al. [138]. The different reactive systems all exhibit common features, the initial melting of Teflon around 615 K and two exothermic peaks. The stronger second exotherm is observed for all systems in a relatively narrow temperature range, 830–860 K, which coincides with the complete melting of the Teflon in the system. The melting is also observed for magnesium and aluminum suggesting that not all the metal fuel reacted upon heating to their respective melting points. All the different systems have a smaller initial exotherm followed by a stronger second exotherm. The initial smaller exotherm however occurs at slightly different temperatures for different systems. The similarity in DSC plots suggests that decomposition of PTFE, a common denominator for all composites, plays a critical role in enabling the exothermic metal fluorination.

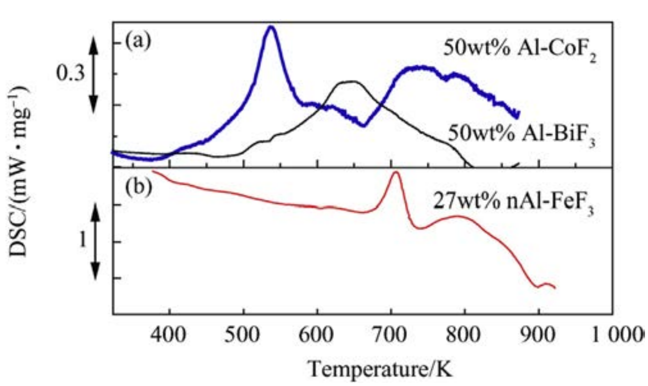

Fig. 19. DSC plots for various metal/metal fluoride based reactive systems in argon atmosphere.(a) 50 wt% Al·CoF_2 and 50 wt% Al·BiF_3 milled composites, heated at 5 K/min [59]. (b) 27 wt% nanoAl-FeF_3 mixed powders heated at 10 K/min [58].

The thermal analysis of aluminum-metal fluoride systems provide an initial understanding of metallic composites that containing inorganic fluorine based oxidizers. Fig. 19 collates thermal behavior of three such aluminum-metal fluoride systems in inert gas; Fig. 19(a) containing Al·CoF_2 and Al·BiF_3 [59], and Fig. 19(b) containing Al·FeF_3 [58]. Despite different methods of preparation (milling and blending) and differences in compositions, all metal fluoride systems exhibit a common feature. Unlike the polymeric oxidizer based systems, the metal fluoride oxidizer systems exhibit a strong first (or even single) exotherm. The diffused secondary exothermic hump, as seen in Al·CoF_2 and Al·FeF_3, may be attributed to the phase transformation of the aluminum fluoride as understood through XRD studies of systematically quenched samples, detailed in Ref. [132].

1.7. Ignition and combustion experiments

1.7.1. Ignition through quasi-static compression

Al-PTFE composites may be of interest as reactive structures and reactive fragments. Their initiation through compression has thus been studied in both dynamic and quasi-static compression experiments [137]. Correlations between ignition characteristics and mechanical properties, including yield stress, elastic modulus and density as a function of pressure at which the pellets were pressed/molded were studied for consolidated composites prepared by sintering and cold pressing Al-PTFE (26 wt% Al). Ignition occurring upon quasi-static compression is illustrated in Fig. 20. The development of the crystallinity during sintering and molding was determined to be pivotal in ignition due to mechanical properties that dictate shear and crack propagation [137]. Lower crystallinity was favored, as high crystallinity allowed the formation of fibrils that bridge cracks and dissipate the energy preventing initiation [137].

The more traditional dynamic compressive test involves a drop-weight experiment, where a fixed weight is dropped on a pellet from various heights. The initiation during these tests was confirmed through visual emission observed [139,140].

Both the dynamic and quasi-static experiments was discussed qualitatively based on a plot shown in Fig. 21 [137]. It shows energy absorbed during the tests as a function of the pressure used to press/mold the sample. The energy levels required for initiation between both static (88–103 J) and dynamic (77–91 J) compressive experiments were found to be comparable to each other. A relatively minor discrepancy was assigned to an unquantified effect of dissipation of heat into surroundings during a slow quasi-static compression. It was, therefore, suggested that the initiation of Al-PTFE under compression may be insensitive to the rate of imported energy. The energy absorption was proposed to be a better metric for the initiation than stress or impact speed.

Fig. 20. Violent exothermic reaction of Al-PTFE during quasi-static compression [137].

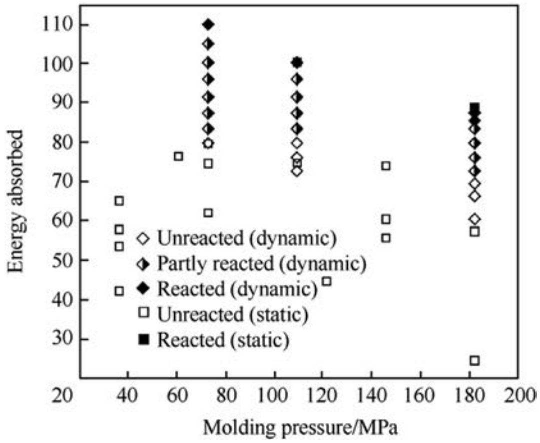

Fig. 21. Energy absorption for Al/PTFE under quasi-static and dynamic compression [137].

1.7.2. Shock initiation/compression

Short time-scale experiments exploring a compression-initiated Al-Teflon® reaction were performed in a series of studies [141,142]. A pulsed laser was used to punch a 25-μm thick flyer from a Cu foil. The flyer traveled 375 μm in vacuum onto a 3-μm thick, Al-Teflon® film spin-coated on a 6.35-mm thick sapphire substrate. Beneath the substrate, emission of the ignited sample is monitored by a 32-channel fiber-optics spectrometer built using 32 photomultiplier tubes and digitizers. The speed of the flyer is assessed by a photon Doppler velocimeter. The Al-Teflon® film is prepared using 40-nm spherical Al powder with varying Al/Teflon® ratios.

When initiated by the laser pulse, the flyer accelerated to 0.7–1.7 km/s. Upon impact with the Al-Teflon® film, a steady shock was produced for several ns, during which the material was initiated. It was found that 0.6 km/s was the threshold speed required to initiate the reaction, irrespective of Al wt% in the system. The compressive stress thus subjected onto the film results in a heat release and optical emission. A two-burst optical emission pulse was repeatedly measured. The first burst appeared with a time delay slightly reduced from ca. 40 to 25 ns at increased impact energies. The later burst, occurring after the shock unloads, represented ambient pressure combustion. The intensity of both bursts increased at greater Al load and at greater flyer energies. The first burst was of primary interest, as indicating the shock-initiated ignition.

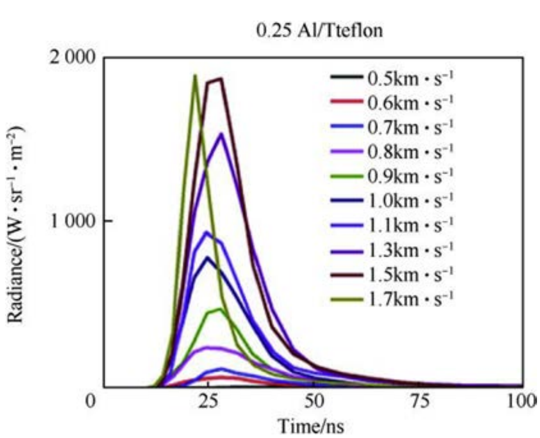

Fig. 22. The first bust radiance transients on an expanded time scale for various flyer speeds with 0.25 Al/Teflon® ratio [141].

Fig. 22 shows the optical emission for different flyer speeds for a fuel-lean composition with Al/Teflon® equivalence ratio of 0.25. The emission occurs at about the same time for different flyer speeds, suggesting that the timing is governed by reaction between Al and Teflon®. The experiments showed that thermal decomposition of Teflon® occurred in the same fashion with or without Al present, and thus was shock driven. The exothermic reaction with Al leading to temperatures in the range of 3800–4600 K followed the Teflon® decomposition. The proposed reaction mechanisms differs from that discussed elsewhere [143] which suggests that thermal decomposition of PTFE is facilitated by interactions between alumina surface and fluorine atoms from PTFE.

1.7.3. Ignition through flash-heating

Processes occurring during very short, nano-second time scales leading to ignition were probed in experiments where a 3–4 μm thick Teflon® film doped with 30-nm aluminum (2-nm oxide layer) was subjected to near-IR laser pulses [144]. The Al-Teflon® composite was fuel lean with only 18 wt% Al. The film was prepared by spin-coating well-dispersed Al-Teflon® system onto a CaF_2 substrate. The reaction was detected using IR transient absorption spectroscopy and ultrafast microscopy. Fig. 23 shows the ultrafast microscopy images during the flash heating of the samples at various times. A 100-ns laser pulse assisted by the subsequent chemical reactions between the heated Al and surrounding polymer matrix, generates a blast wave. The flash heating delivers a dose of energy making a weakly ionic aluminum plasma at a temperature of 4000–8000 K. The 200-μm crater formed due to the local ignition and combustion of aluminum particles with immediately surrounding Teflon® matrix. The propagation of the wave front (seen as a hemisphere) is slightly faster than the reaction of bulk/plasma (darker splatter). The time scale of the ignition delay is in about 10 ns (time difference between first frame and second frame where the wave front and reaction are observed), comparable to that observed in shock initiation experiments.

Considering Teflon® as a copolymer of tetrafluoroethylene (TFE) and 2,2-bis(trifluoromethyl)-4,5-difluoro-1,3-di-oxole (dioxole), changes in spectral features attributed to TFE and dioxole were of particular interest. The system was assumed to behave as having two oxidizers interacting with n-Al as fuel. It was found that aluminum reacted with CFO species (from dioxole) 10 times faster than with CF_2/CF_3. Since the system is fuel-lean, aluminum preferentially reacts with dioxole as a sole oxidizer almost entirely till the fuel/oxidizer ratio is at stoichiometry and then begins to consume the TFE oxidizer [144].

1.8. Performance characterization in practical applications

In a typical test, cylindrical pellets of 10 mm diameter and 7.8 g/cm^3 density were pressed and sintered with 11.3 wt% of PTFE, 7.5 wt % of Al and 81.2 wt% of W [145]. These pellets were used as reactive projectiles and shot into aluminum plates kept 8 m away from the gun. The effect of projectile weight, velocity and thickness of target plate on penetration behavior was considered. The impact velocity was measured by probes and a high-speed camera observed the flight of the projectile through the aluminum plate. Semi-empirical equations were developed to predict the velocity required for the projectile to penetrate reliably a given piece of aluminum target sheet or its ballistic limit velocity. For a given set of conditions, the ballistic limit velocity of the reactive material, (W/Al/PTFE) projectiles, was found to be higher than for steel projectiles. It was also found that when the projectile impacts the aluminum plate at approximately ballistic limit velocity, the chemical energy released during penetration slows the projectile down by the deflagration pressure increased in the penetration direction [145].

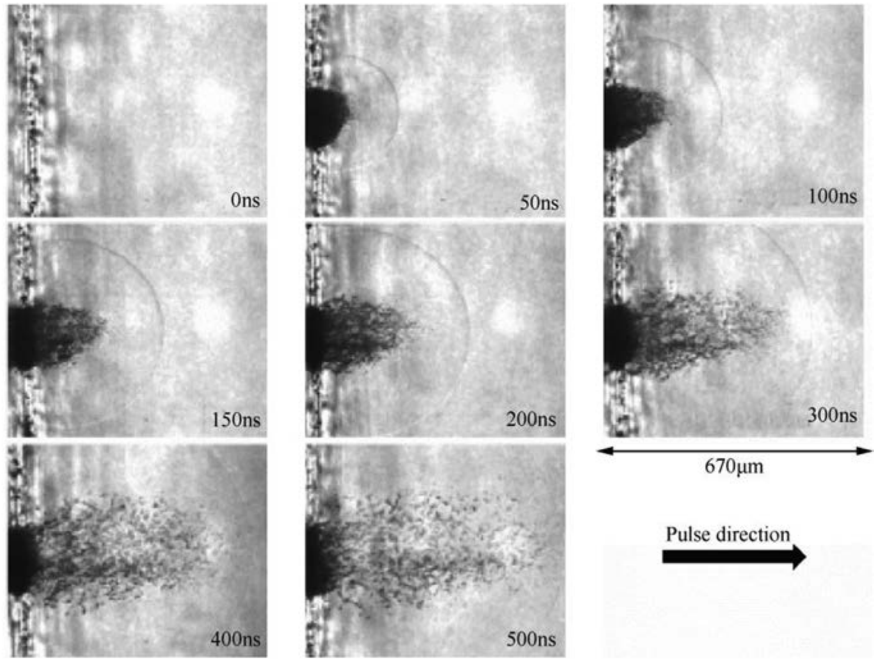

Fig. 23. Time series of ultrafast microscopy images obtained by flash-heating an Al/TeflonAF thin film using a 100 ns duration 1064 nm laser pulse. The beam diameter is 50 μm and the pulse energy is 40 μJ. The images show the Al/Teflon® surface at far left. The images show the explosive ablation of material from the surface at the indicated times. The hemisphere is a blast wave [144].

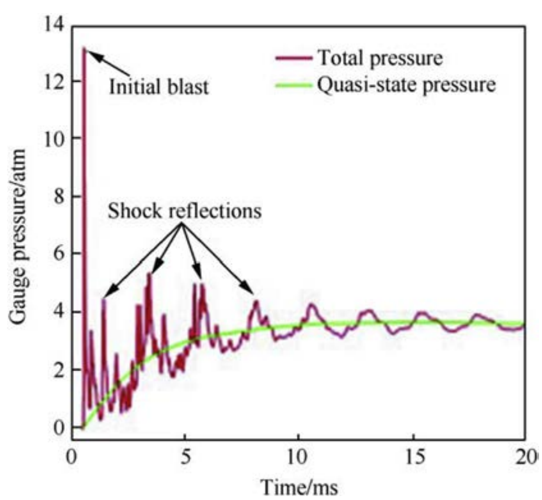

Fig. 24. The quasi-state and blast pressures as a function of time [146].

A related experiment was performed for Al-PTFE (26.35 wt% Al), Zr/THV (52 wt% Zr), Ta-THV (74 wt% Ta) and Hf-THV (69 wt% Hf) where THV is a mixture of tetrafluoroethylene, hexafluoropropylene and vinylidene fluoride. These materials were pressed and sintered to form spherical projectiles with densities greater than 96% TMD [146]. The projectiles were fired at 1.2–2.4 km/s onto a closed drum-like chamber covered with a 1.5-mm thick steel plate. The projectile pierced through the plate and disintegrated. Pulverized fragments, some of which ignited, continued moving towards the center of the chamber hitting an anvil, which caused their secondary initiation [146]. The pressure inside the chamber was measured as a function of time as presented in Fig. 24. The reaction between metal and the fluorinated polymer was found to be very fast and occurred within about 10 μs or faster. It produced a pressure wave, which reflected around the drum until a quasi-static pressure was attained, as seen in Fig. 24. Extended combustion continued in the chamber for 1–10 ms following the impact with the anvil. The reaction efficiency for various systems used depends on reaction mechanisms and speed of projectile. At lower speeds of 1.2 km/s, Al-PTFE and Zr-THV outperformed other systems. With increase in the speed of projectiles, the increase in efficiency was observed in all samples. All materials had efficiencies of 70%–80% while Ta-THV underperformed substantially. The effect of binders/oxidizer used was also observable. The compression yield strength and melt temperature difference between the two versions of THV resulted in different efficiencies for Hf-THV and Ta-THV systems. The effects of density and mass loading of a projectile are important, for mass loadings over 19.6 g, the reaction efficiency is a direct function of loading.

1.9. Proposed reaction mechanisms

Regardless of other details, a two-stage reaction sequence is observed for many systems involving a metal and fluorocarbon, as illustrated in Figs. 17 and 18. A two-stage sequence is also noted in the shock-initiated samples, as evidenced by the two-burst

Table 10
Temperatures at which smaller first and second exotherms are observed for fuel/oxidizers for samples heated in inert atmosphere as compiled by Koch [147].

Fuel/oxidizer	Temperature of the exotherm/°C		Reference
	First	Second	
Mg/PTFE	377	489	[138]
Mg/PMF	420	500	[148]
Al/PTFE	450	480	[138]
Zn/PTFE	170	320	[147]
Ti/PTFE	464	480	[138]
Zr/PTFE	410	470	[149]

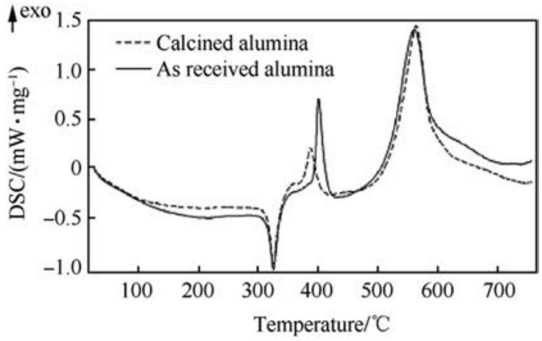

Fig. 25. DSC of 15-nm Al_2O_3-200nmZonyl (PTFE source) and calcined Al_2O_3-200 nm Zonyl [105].

structure of the recorded optical emission [141,142]. The temperatures of the first and second exothermic peaks observed for different metals by different investigators are summarized in Table 10.

A similar two-stage sequence, although with a reduced heat effect was observed for reaction between PTFE and alumina, as shown in Fig. 25. This latter case is relevant because the reaction in the blended aluminum and fluorocarbon powders begins at the interface of fluorocarbon and surface alumina layer covering particles of aluminum. Because alumina is a common catalyst substrate, fluorination of alumina reacting with different fluorocarbons has been studied extensively, [150–152]. It is generally understood that the reaction occurs in two main steps. The first step is commonly catalyzed by oxygen present as hydroxyl groups on the surface of transition alumina and/or as an impurity, or a dioxole copolymer in PTFE. Hydroxyl groups, for example, serve to attract carbocations of fluorinated species chemisorbed to the alumina surface [151]. The initial reaction results in formation of selected Al-F bonds, while the species formed are transient in nature. The role of surface hydroxyl groups in the initial stage reaction is illustrated in Fig. 25, where the DSC traces are shown for as received and calcined alumina interacting with PTFE. For the calcined alumina, hydroxyl groups are removed; respectively, the first exothermic peak corresponding to the initial reaction is weakened, but it does not disappear, suggesting that hydroxyl groups are helpful but not necessary for the reaction to begin. The copolymer used as source of PTFE may contain oxygen in the form of ether links between units, thus resulting in the minor initial exotherm albeit with reduced intensity as compared to the alumina surface with hydroxyl groups.

The second reaction step occurs when all unsaturated sites of surface aluminum are fluorinated, which often requires additional source of fluorine, and thus can coincide with decomposition of the fluorocarbon. As the fluorination progresses, the terminal groups condense to slowly form α- AlF_3 rather than the unstable β- AlF_3.

While the two-step mechanism outlined above is generic, the rates of individual steps are affected by the type of fluorocarbon used, available reactive interface area, and alumina structure. For example, an effect of solvent used to sonicate and disperse blends of nano-aluminum and micron-sized Zonyl (PTFE source) on the rate of exothermic reactions was observed in Ref. [107]. Both heat release and temperature for the second exothermic reaction step were affected, with the blend prepared using a polar solvent, 2-propanol, being most reactive. It was proposed that alumina treated with a polar solvent retains mobile hydroxyl groups increasing the probability of attracting fluorinated species. The suggested mechanism has been extended for other polymeric oxidizers such as PVDF, where Delisio et al. [153], show the same mechanism at work for films consisting of nano-sized aluminum particles with natural oxide layer, embedded in PVDF polymer for a range of aluminum/polymer ratios. The decomposition of polymers yields different fluorinated species depending on the polymer used [150,151,153] and the conditions in which decomposition occurs [154]. The presence of excess alumina, however, does not increase the reaction extensively as the reaction is a function of the surface sites on the alumina, inviting fluorinated species attack and not with alumina itself [105,153].

In addition to reactions of fluoropolymers with alumina, their reactions with magnesium have been discussed in detail because of a widespread use of magnesium-fluoropolymer systems in pyrotechnics. The first exotherm occurring in the reaction of magnesium with PTFE is interpreted based on the Grignard reagent forming nature of magnesium. The initial Mg-F bonds formed in the reaction produce a C-Mg-F complex considered to be the Grignard type intermediate [155]. This Grignard intermediate breaks down to give magnesium fluoride and broken polymeric chains in the second exothermic step, as shown in the following reactions [65]:

Step 1: $\mathrm{Mg} + (\mathrm{C_2F_4})_n \rightarrow (\mathrm{CF_2\text{-}CF\text{-}Mg\text{-}F})_n + \mathrm{heat}$
Step 2: $(\mathrm{CF_2\text{-}CF\text{-}Mg\text{-}F})_n \rightarrow (\mathrm{CF{=}CF})_m\text{-}(\mathrm{C_2F_4})_n + m\mathrm{MgF_2}\,(\mathrm{s}) + \mathrm{heat}$

Overall equation: $\mathrm{Mg} + 2\mathrm{R^FC\text{-}F} \rightarrow \mathrm{MgF_2}\,(\mathrm{s}) + 2\mathrm{R^FC} + \mathrm{heat}$

The proposed reaction mechanism was tested through FTIR analysis. It can be seen from Fig. 26, where the FTIR of two samples of Mg/PTFE collected at 600 and 700 °C, that the formation of C-Mg-F bond intermediate is confirmed experimentally [156] as predicted through ab-initio calculations [155]. The Grignard intermediate C-Mg-F bond frequencies formed at 600 °C give way to MgF_2 bonds at higher temperature, as the reaction is completed.

While most common pyrotechnic magnesium-fluorocarbon systems are prepared by mixing components, a similar two-stage reaction sequence is observed in the ball-milled composite, in which the interfaces between magnesium and polymer may form differently [157]. Steletskii et al. [157], offer an explanation for the presence of two exotherms considering sequence of decomposition of fluorine atoms from the polymer in presence of magnesium. It is suggested that the exothermic effect observed at lower temperatures, 300–420 °C, is relatively weak due to the removal of the first fluorine atom being energy intensive and dampening the exothermicity of magnesium fluorination. The subsequent loss of fluorine atom is relatively easy and the exotherm is stronger [157]. Conceptually, this explanation is consistent with the previously discussed mechanism. Indeed, formation of the initial Grignard reagents, involving removal of the first fluorine atom from the

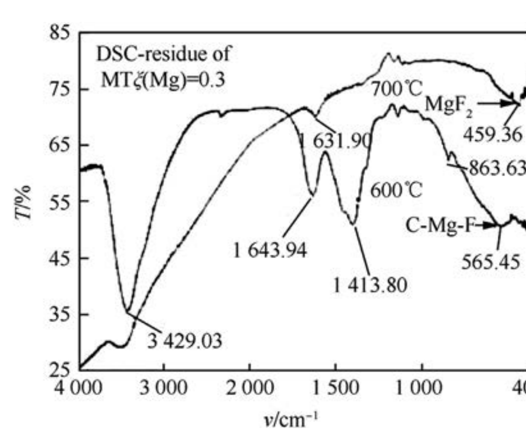

Fig. 26. FTIR Absorption spectra of MTV samples collected at two temperatures 600 °C and 700 °C [156].

polymer, is not as exothermic as formation of the fully fluorinated magnesium. For the systems prepared by milling, it is proposed that the stacking faults in the material grains gradually move towards the surface as the temperature increases and the reactions occur at the dislocations. This leads to an improved dispersion of decomposition products which fluorinate the magnesium crystallites more effectively through defects induced by milling.

It is apparent that the reaction sequences for metals like aluminum and magnesium reacting with fluorocarbons are similar despite their reactivity difference. In each case, a metastable complex with a metal-fluorine bond is formed in the first step, while the complete fluorination occurs in the second step. Depending upon the reactivity of the metal involved, the structure of the intermediate species formed is dependent. In case of the more reactive magnesium, the C-F bond is cleaved by the metal in the first step while for the less reactivity aluminum, the difference in polarity between species involved allows for bond formation that subsequently weakens the C-F bond. Thus, apparently, a similar reaction mechanism, in which formation of intermediate partially fluorinated compounds precedes the complete fluorination is valid for reactions with other metals (Fig. 18, Table 10). This points to the larger applicability of the mechanisms offered for other metal-fluoropolymer systems. Depending upon the reactivity of the metal involved, and the availability of non-fluorine species in the polymer upon metal surface, the appropriate specific reaction mechanism needs to be refined based on the outlined above generic two-step process.

2. Conclusions and future work

Metal-fluoropolymer composites are widely used in pyrotechnic applications. Their advantageous features range from metal surfaces protected by fluoropolymers during storage to reduced agglomeration due to volatile combustion products. Potential benefits of such composites are underutilized, however, for other energetic formulations, specifically, propellants and explosives. Among different fluoropolymers, PTFE is the most widely used, although use of PVDF, PMF and perfluorinated carboxylic acids is increasing. Often, it is difficult to achieve homogeneous and fine scale mixing of a fluorinated oxidizer, commonly, a polymer, with metal fuel, which prevents the use of such composites in many advanced energetic formulations. New processing techniques, including aerosol synthesis, cryomilling, and others are being explored. The milled, coated and laminate composite systems are generally more attractive than composites using mixed or blended powders because of the improved metal-oxidizer interfaces and high energy density. A systematic study of physical and chemical properties of fluoropolymers may be of interest to make informed choice of the synthetic approach using specific oxidizers based on the application requirements.

In all metal-fluoropolymer composites, the polymers largely decompose releasing gaseous fluorocarbons that act as primary fluorinating agents. To predict or interpret kinetics of chemical reactions in such composites, the composition and structure of polymers obtained from commercial sources needs to be considered carefully, owing to presence of secondary active species that may function as competing oxidizers. Differences have been reported in ignition and combustion behaviors of energetic formulations depending on the choice of specific type or brand of the fluoropolymer, even when the main fluorinated molecules are the same.

Despite multiple laboratory studies, no generalized framework for the reaction mechanism across different preparations currently exists. The reactions are not described in detail even for Al/PTFE and Mg/PTFE, the most studied compositions. There may be, however, an approach for developing a generic reaction mechanism for a broad range of metal-PTFE composites because upon heating, all such composites exhibit a qualitatively similar, two-stage reaction irrespective of the method of preparation. The two-stage mechanism may also be extended for other systems with polymeric oxidizers as shown for Al/PVDF. Experimental studies have provided plausible mechanistic explanations for mixed systems where shared interfaces are limited. For most metal fuels, including aluminum, oxygen plays a role in initiating fluorination, and intermediate fluorinated compounds formed in the first reaction stage convert to final fluoride products during the more high-temperature second stage reaction. Oxygen can be available from metal oxide, hydroxyl groups attached to the surfaces, from additives to polymer, or from oxygenated environment. For some particularly reactive metals, such as magnesium, the metal can directly embed into and then cleave C-F bond in fluorocarbons, thus initiating fluorination. For magnesium, this is described by the Grignard mechanism. This mechanism needs to be explored for a broader range of compositions to establish its applicability for reactive fuels other than magnesium.

The correlation between structural defects, coordination and chemical pathways may provide detailed understanding of reaction mechanism in metal-fluoropolymer systems. Experiments with simple configurations, such as planar laminates or core-shell spherical particles are desired, which are readily interpreted mechanistically. Systematic efforts in selecting and comparing composites with fuels with varied properties are needed to elucidate direct fuel/polymer and fuel/oxide layer/polymer interactions, enabling us to extend the known mechanisms to describe the presently poorly understood staged fluorination reactions initiated by different stimuli.

Connecting thermal initiation with shock-driven initiation and with reactions in detonation-like regimes is also of interest. These are complex systems with multiple simultaneous processes. Planning reproducible simple combustion experiments building up on the recent progress, e.g., with flash and shock-ignited samples would be desirable. Exposing the same composites to different reaction stimuli would be of particular interest.

Use of fluorinated oxidizers other than polymers, such as metal fluorides, has just started attracting attention of researchers dealing with reactive and energetic materials. Reactions in these cases may be qualitatively different due to the absence of the carbon backbones of the polymers and elimination of gaseous active fluorocarbon generation step. A semi-empirical mechanism suggested based on early experiments for metal-fluoride composites focuses on a low-temperature reaction, releasing substantial heat, unlike a relatively weak first step reaction involving polymeric systems. The low-temperature exothermic reaction leads to very low ignition temperatures and improved reaction kinetics. A reduced ESD ignition sensitivity in these materials is also very attractive. A detailed study into prospective inorganic oxidizers like metal fluorides and the combustion mechanisms for respective formulations would be of value for future studies.

Acknowledgement

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December 22, 2021 *via email: president@whitehouse.gov*

U.S. President Biden
1600 Pennsylvania Ave. NW
Washington, DC 20460

Dear President Biden,

We the undersigned are elevating a matter in urgent need of your immediate attention and action. Many of us live in the Gulf coast region directly impacted by the 2010 BP Deepwater Horizon oil disaster and use of toxic chemical dispersants during spill response. Hazardous dispersants were staged in our public marinas and neighborhoods; they were sprayed in coastal seas where we swim and fish; they were used long after the "official" end date in July 2010. Hazardous oil spill waste was disposed of in several of our municipal landfills. Now our families, friends, and neighbors suffer with long-term diseases, disabling chemical illnesses, cancers, and early deaths linked with toxic exposures from this oil spill, and childhood cancer rates have soared in our coastal communities. Many of us have participated in the long-term studies that found that dispersants make oil more toxic. Local wildlife is also experiencing long-term health harm from this oil disaster. Yet dispersant use continues even now. We are joined by others who share our concerns, having experienced oil spills and dispersant use in other areas. We all pray for relief from these toxic chemicals.

We have sought relief from both the EPA and the courts, and we have played by the rules only to be frustrated by industry pressures. Some of us filed a rulemaking petition with EPA on November 14, 2012, urging an NCP update to rules governing dispersant use. On January 22, 2015, EPA issued a Notice of Proposed Rulemaking to revise the NCP's Subpart J, the rules governing dispersant use. EPA stated the proposed rule was "anticipated to encourage the development of safer and more effective spill mitigating products, and would better target the use of these products to reduce the risks to human health and the environment." Sounded good, but for years, EPA took no action to finalize its proposed rule.

After waiting through two federal administrations for a final rule, a group of environmental justice and tribal advocates and individuals, including some of us, sued EPA under the Clean Water Act (CWA) and the Administrative Procedures Act (APA) (*Earth Island Institute/ALERT Project et al. v. Andrew Wheeler et al.* 3:20-cv-00670-WHO).

We were elated when, on August 9, 2021, the federal district court ruled in our favor on both claims! Indeed, the court clarified an important part of the CWA, finding that "EPA has a nondiscretionary duty to revise or amend the NCP *when there is new information that shows that the current standards for efficient, coordinated, and effective action to minimize damage from oil and hazardous substance pollution are insufficient to safely provide for mitigation of any pollution*." See Order at 8 (p. 6, lines 9–12), interpreting 33 U.S.C. §1321(d)(2). And the court agreed that waiting through now three administrations and eight years "was unreasonable and compelling agency action." (p. 16, lines 12–13). The court imposed a deadline of May 31, 2023, for EPA to issue a final rule, under court supervision, in its 2015 proposed rulemaking.

Despite our victory, we have several reasons to remain concerned that EPA will continue to ignore critical new information in this rulemaking. On July 27, 2021, just prior to the court decision in August, the EPA signaled its intent to allow dispersant use in future spills when it issued a final rule, based on the 2015 Proposed Rule, to require monitoring of dispersant use in certain situations. Yes, that's right, it did. It's almost like EPA didn't really hear the court since this final rule does not include the current science from May 2015 to present. Given this action, we now also fear that EPA will ignore this science in its final rule in May 2023. This latest science contains the bulk of a growing number of independent studies that show deadly harm to human health and the environment from dispersant use during oil spill response under the NCP. *This is a solid block of new information that clearly meets the federal court's recent interpretation of the CWA and compels EPA to update the NCP accordingly.*

Mr. President, we have done everything we can to hold EPA accountable to the spirit and intent of the laws that are designed to protect the waters of the U.S. and our health and wellbeing. But we feel a higher hand is now needed to compel EPA to do the right thing. This is why we now ask *you*, the President of the United States, to take two specific and immediate actions: 1) order EPA to withdraw its final rule issued on July 27, 2021, before it goes into effect on January 24, 2022; and 2) order EPA to issue one comprehensive rule in its 2015 proposed rulemaking, on or before May 31, 2023, based on the latest science, i.e., to present. (More support for each of these requests is provided in Appendix A and B.)

Mr. President, we have paid the ultimate price for short-sighted, industry-driven public policy. Yet we are willing to wait until May 31, 2023, for one comprehensive rule that is revised to reflect the emerging proof of deadly harm to people and sealife from dispersant use. It is time to recognize that oil dispersants are a net environmental *loss*, not benefit. We believe that only you can make this happen. Please hear us.

Sincerely,
Gulf Coast advocates and allies

Eastern Shore Community Health Partners, Inc.
Lesley Pacey, Founder & Director
http://easternshorechp.org/

Healthy Gulf
Cynthia Sarthou, Executive Director
www.healthygulf.org

Gulf Coast Creation Care
Lella B. Lowe, Co-President
https://gulfcoastcreationcare.org

ReThink Energy Florida
Kim Ross, Executive Director
www.rethinkenergyflorida.org

Surfrider Foundation
Nicole de Venoge, Florida Policy Manager
https://www.surfrider.org/

Texas Environmental Justice Advocacy Services
Juan Parras, Executive Director
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Turtle Island Restoration Network
Joanie Steinhaus, Gulf Program Director
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Dr. Yolanda Whyte Pediatrics
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Kindra Arnesen (Plaintiff)
Plaquemines Parish, Louisiana

ALLIES

Dr. Rosemary Ahtuangaruak (Plaintiff)
Nuiqsit, Alaska

Alaska Community Action on Toxics (Plaintiff)
Pamela Miller, Executive Director
www.akaction.org

ALERT, a project of Earth Island Institute (Plaintiff)
Riki Ott, PhD, Founder & Executive Director
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Cook Inletkeeper (Plaintiff)
Sue Mauger, Executive Director
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Friends of the Earth
Hallie Templeton, Legal Director & Senior Campaigner
https://foe.org/

Friends of the San Juans
R. Brent Lyles, Executive Director
https://sanjuans.org/

Government Accountability Project
Tom Devine, Legal Director
www.whistleblower.org

cc:

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ENVIRONMENTAL JUSTICE ADVISORY COUNCIL

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Senior Director for Environmental Justice

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Beverly Wright, PhD (SE)
Founder & ED, Deep South Center for Environmental Justice

Susana Almanza (SW)
Director, People Organized in Defense of Earth and Her Resources

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NATIONAL ENVIRONMENTAL JUSTICE ADVISORY COUNCIL

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U.S. ENVIRONMENTAL PROTECTION AGENCY

Administrator Michael Regan

Office of the Inspector General
Inspector General Sean O'Donnell

Office of Land & Emergency Management
Carlton Waterhouse, Deputy Assistant Administrator

Appendix A

A brief history to support our first request

The BP Deepwater Horizon (DWH) disaster raised serious concerns about dispersant use that EPA had failed to consider or address in its rules that were then (and still are) in effect. For example, should dispersants be used deep underwater, when oil spill response plans only contemplated and authorized surface use? How much dispersant can spill responders apply before toxic effects overwhelm local ecosystems and pose health hazards to first responders, contract workers, and the exposed public? EPA could not answer these questions, yet it allowed the federal response team and BP to proceed with unprecedented "atypical" dispersant use in large volumes through deep sea injection and surface spraying of long durations – and monitor effectiveness during operations – in effect, to make it up as it goes. This proved devastating for people living in coastal communities, oil spill responders, and wildlife.

During May through December **2010**, BP collected and analyzed literally tens of thousands of water samples between the ocean floor and sea surface, likely to test the efficacy of an experimental technology, subsea dispersant injection (SSDI), for response to offshore oil spills. BP reported that SSDI reduced levels of benzene and other volatiles at the sea surface, which BP claimed would (theoretically) lower health risks for oil spill workers at the surface.[1]

Based on BP's preliminary results, the federal government acted in November **2010**, requiring lessees and operators of oil and gas leases on the outer continental shelf to demonstrate capability to access and deploy subsea dispersant injection equipment.[2]

Encouraged by BP's preliminary analysis and the Dept. of Interior's actions, the National Response Team and American Petroleum Institute developed environmental monitoring requirements for atypical dispersant use by **2013.**[3]

In **2015**, the EPA issued its Proposed Rule, which addressed 1) the outdated testing protocols with updated authorization of use procedures and methods, and 2) the need for less toxic products with new data and information requirements, and promoted 3) the

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[1] This industry-sponsored paper ignores studies that disprove this claim (see footnotes 20, 24, and 2). Lin Zhao, D.A. Mitchell, R. Prince, et al. **2021**. [BP DWH] 2010: Subsea dispersants protect responders from VOC exposures. *Marine Pollution Bulletin,* 173(20):113034. DOI: 10.1016/j.marpolbull.2021.113034.

[2] US Interior Department, Bureau of Ocean Energy Management, Regulation, and Enforcement, 2010, National Notice to Lessees and Operators (NTL) of Federal and Gas Leases, Outer Continental Shelf, Statement of Compliance with Applicable Regulations and Evaluation of Information Demonstrating Adequate Spill Response and Well Containment Resources, NTL No. 2010-N10, effective Nov. 8, 2010 to Nov. 8, 2015.

[3] NRT **2013**. Environmental Monitoring for Atypical Dispersant Operations: Including Guidance for Subsea Application and Prolonged Surface Application. May.

API, American Petroleum Institute. **2013**. Industry Recommended Subsea Dispersant Monitoring Plan. Technical Report 1152, Sept.

industry-government monitoring protocol for atypical dispersant use in response to an oil discharge to waters of the United States.[4]

In March **2019**, environmental justice and Tribal advocates sued EPA over its failure to maintain and update the NCP based on current science.

On July 27, **2021**, EPA issued a new rule for monitoring atypical dispersant use, based on science through the close of the April 2015 public comment period.[5] This monitoring rule suggests that atypical dispersant use will ultimately be authorized in the final rule – even though it remains to be seen whether such use will be authorized in May **2023**, given that the post April 2015 science has consistently found that chemically-dispersed oil is more toxic than mechanically- or naturally-dispersed oil.[6]

Mr. President, we have a national contingency plan precisely to prevent the oil industry and our government from conducting such wide-scale experiments in our ocean. Yet this is exactly what is happening. The U.S. GAO even said so in its December **2021** report: "While epidemiological study of human health effects of dispersant use during the [BP] Deepwater Horizon oil spill is ongoing, additional epidemiological study into human health effects cannot occur until the next oil spill in which dispersants are used."[7]

Surely, it doesn't take much imagination to realize that deepsea oil drilling carries risk of deepsea oil spilling. Surely, this should drive scientific inquiry as to how to safely mitigate that risk *before any leasing and harm occur*, and it should result in contingency plans to mitigate that risk and harm, plans which engage the public in decision-making, as required by the law.

<u>The rule for atypical dispersant use makes a mockery of public process. Allowing it to go into effect is a win for the oil industry and only encourages them to put even more pressure on policymakers to bend to the industry's will.</u>

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[4] EPA **2015**. National Oil and Hazardous Substances Pollution Contingency Plan; Proposed Rule. published January 22, 2015. EPA-HQ-OPA-2006-0090; FRL-9689-9-OSWER

[5] EPA **2021**. National Oil and Hazardous Substances Pollution Contingency Plan; Subpart J Product Schedule Listing Requirements. Final rules published July 27, 2021. EPA-HQ-OPA-2006-0090

[6] Baurick T. Oil dispersants used in BP disaster must undergo EPA health, safety review, judge rules. *New Orleans Times-Picayune*, Aug. 10, **2021**

[7] U.S. GAO. **2021**. Offshore oil spills. Additional information is needed to better understand the environmental tradeoffs of using chemical dispersants. GAO-22-104153. Dec., 59 pp.

Appendix B

A brief summary of science to support our second request

There is literally a scientific sea change in thinking about the utility and safety of dispersants in oil spill response. After the BP DWH oil disaster, it became plainly evident to the public and scientists that the scientific theory of the past forty years didn't square up with the reality of long-term harm to people and marine wildlife. An unprecedented amount of dispersant was used at the surface and subsea, yet an unprecedented amount of oil still came ashore, despite industry assurances to the contrary. It's also when more funding for dispersant research became available for independent studies – and these more recent studies (in particular, post April 2015) often counter the industry rhetoric.

For example, dispersants are very effective at sinking surface oil to the bottom of the ocean, something industry still maintains they do not do. Independent studies now find that up to 20% of the BP DWH oil disaster may have sunk through interaction with marine snow (derived from plant or bacteria interactions with oil droplets) and then sedimented on the ocean floor (through interactions with mineral particles). Sinking agents are prohibited under the NCP (40 CFR 300.910(e)(1)).

- "… sinking marine oil snow and oil-sediment aggregations during the [BP] DWH contributed appreciably to the unexpected, and exceptional, accumulation of oil on the seafloor…" (Francis and Passow 2020).[8]

- "Formation of marine snow, and resulting sedimentation, is greatly enhanced by dispersants, which increase the formation of microdroplets in the water column … with efficiency by up to 80%–100%..." (Chiu et al. 2019).[9]

Oil-dispersant mixtures also increase the toxicity of oil to sealife by increasing the concentrations and persistence of hazardous oil components (VOCs and PAHs) in the water column, and by increasing the photo-toxicity of oil.

- "Toxicity studies in the period of 2017-2021 (current period of this report) involved more than 27 individual studies conducted by more than 25 separate study groups… All of the studies found that chemically-dispersed oil was more toxic than mechanically-dispersed oil." Further, "[in] most studies, it was found that [the chemically-dispersed oil-water mixture] was from slightly to 1.5 to 100 to as much

[8] Francis S., Passow U. **2020**. Transport of dispersed oil compounds to the seafloor by sinking phytoplankton aggregates: A modeling study, *Deep-Sea Research Part I: Oceanographic Research Papers*, 156, 103192.

[9] Chiu, M.-H., Vazquez, C.I., Shiu, R.-F., et al. **2019**. Impact of exposure of crude oil and dispersant (Corexit) on aggregation of extracellular polymeric substances, *Science of the Total Environment*, 657, 1535-1542.

Ziervogal K., Joye S.B., Kleindienst S., Malkin S.Y., Passow U., et al. **2019.** Polysaccharide hydrolysis in the presence of oil and dispersants: Insights into potential degradation pathways of exopolymeric substances (EPS) from oil-degrading bacteria, *Elementa*, 7, 1, 31.

as 500 times more toxic than the [mechanically-dispersed oil-water mixture], depending on the variables" (Fingas 2021).[10]

- "Dispersants increase the amount of benzene, toluene, ethylbenzene, and xylenes (BTEX) into the water column, as is already known." Further, "Addition of dispersant to a [mechanically-dispersed oil-water mixture] always enhances the [PAH] content of the oil [which] may be a factor in the increased toxicity of the [chemically-dispersed oil-water mixture] (Fingas 2021).[11]

- Dispersants increase the photo-toxicity of chemically-dispersed oil as much as 48 times more than that of physically-dispersed oil, a finding that is extremely relevant for the overall health of the marine foodweb as many species spend all or part of their lives near or on the sea surface (Fingas 2017; Finch et al. 2017, 2018).[12]

Oil-dispersant mixtures cause rapid shifts in microbial community structure, which, ultimately, are much more likely to suppress populations of oil-degrading bacteria than enhance them, contrary to industry claims, while increasing populations of oil-tolerant pathogenic microorganisms that eat human flesh (*Vibrio*) or cause harmful red tides.

- "[T]he presence of dispersants alters both the numbers and succession of hydrocarbon degrading organisms. This appears to be the result of selective toxicity of dispersants to some species while other species are tolerant of dispersants. This effect is different for different dispersants and different dispersant constituents. The end result of this number and succession shift is generally a reduction in biodegradation compared to a situation where dispersants are not used. Further, "[m]ost authors conclude that dispersants suppress biodegradation… consistent with past reviews" (Fingas 2021). Exceptions tend to be studies sponsored by the oil industry as shown in Table 1 (from Fingas 2017).[13]

- "[O]il contamination has been associated with potential for increases in harmful algal blooms and numbers of pathogenic *Vibrio* bacteria in oil-impacted waters."[14]

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[10] Fingas M, **2021**. A summary of dispersants research: 2017–2021, on behalf of Prince William Sound Regional Citizens' Advisory Council, Anchorage, Alaska. PWSRCAC Contract Number 955.18.01.

[11] Fingas, **2021**, Summary of dispersant research, pp. 10 and 12, respectively.

[12] Fingas, M. **2017**. A review of dispersant literature.
Finch, B.E., Marzooghi, S., Di Toro, D.M., et al. **2017**. Phototoxic potential of undispersed and dispersed fresh and weathered Macondo crude oils to Gulf of Mexico Marine Organisms, *Environmental Toxicology and Chemistry*, 36,10, 2640-2650.
Finch, B.E., Stefansson, E.S., Langdon, et al. **2018**. Photo-enhanced toxicity of undispersed and dispersed weathered Macondo crude oil to Pacific (*Crassostrea gigas*) and eastern oyster (*Crassostrea virginica*) larvae, *Marine Pollution Bulletin*, 133, 828-834.

[13] Fingas, **2021**, Summary of dispersant research, p. 9 and 17 (Table 1).
Fingas, M., **2017**. A review of literature related to oil spill dispersants, on behalf of Prince William Sound Regional Citizens' Advisory Council, Anchorage, Alaska. PWSRCAC.

[14] Eklund R.L., Knapp L.C., Sandifer P.A., et al. **2019**. Oil Spills and Human Health: Contributions of the Gulf of Mexico Research Initiative, *GeoHealth*, 3, 12, pp. 391-406. doi.org/10.1029/2019GH000217
Almeda, R., Cosgrove, S., Buskey, E.J. **2018**. Oil Spills and Dispersants Can Cause the Initiation of Potentially Harmful Dinoflagellate Blooms ("Red Tides"), *Environmental Science and Technology*, 52,10, 5718-5724. doi: 10.1021/acs.est.8b00335

- The removal of key grazers due to oil and dispersant disrupts the predator-prey controls ("top-down controls") that normally function in plankton food webs. This disruption of grazing pressure opens a "loophole" that allows certain dinoflagellates with higher tolerance to oil and dispersants than their grazers to grow and form blooms when there are no growth limiting factors (e.g., nutrients). Therefore, oil spills and dispersants can act as disrupters of predator-prey controls in plankton food webs and as indirect inducers of potentially harmful dinoflagellate blooms" (Almeda et al. 2018).

Dispersants are also very effective at turning oil spills into toxic oily mists, composed of ultrafine particles that drastically increase the toxicity of oil to humans, in contrast to industry claims. This also conflicts with the number one priority in oil spill response: protection of human life and safety (40 CFR 300.910(d)).

- "The total number of concentrations of airborne particulates originating from oil-dispersant mixtures are 1–2 orders of magnitude [10 to 100 times higher] than those of crude oil across the entire nano-scale range… Conversely, the differences in concentration are small" (Afshar-Mohajer et al. 2018).

- "Inhalation of airborne particles emitted from the slick containing dispersant increased the total mass of deposited particles in upper respiratory regions compared to the slick of crude oil only… [T]he application of dispersant onto the pollution slick increased the total mass burden to the human respiratory system about 10 times…" (Afshar-Mohajer et al. 2019).[15]

- Results from the Coast Guard cohort study suggested strong relationships between oil and oil-dispersant exposures and acute respiratory symptoms – coughing (19.4%), shortness of breath (5.5%), and wheezing (3.6%) – among disaster responders. The combination of both oil and oil-dispersants presented associations that were much greater in magnitude than oil alone for these three symptoms. Further, prevalence ratios for all three acute respiratory symptoms were higher among responders who did not report any use of Personal Protective Equipment (PPE) compared to those who did report any use of PPEs. A similar pattern was found for responders reporting use of a respirator; i.e., those who did not report use had higher prevalence for shortness of breath and wheezing (Rusiecki et al. 2018). Statistically significant statistically significant associations for crude oil were also found with neurological symptoms of headaches and light-headedness/dizziness, dermal symptoms of skin rash/itching, gastrointestinal symptoms of diarrhea and stomach pain (associated with exposure response relationships) and nausea/vomiting, genitourinary symptoms of burning or painful urination.[16]

[15] Afshar-Mohajer, N., Li, C., Rule, A.M., et al. **2018**. A laboratory study of particulate and gaseous emissions from crude oil and crude oil-dispersant contaminated seawater due to breaking waves, *Atmospheric Environment*, 179, 177-186.
Afshar-Mohajer, N., Fox, M.A., Koehler, K. **2019**. The human health risk estimation of inhaled oil spill emissions with and without adding dispersant, *Science of the Total Environment*, 654, pp. 924-932.
[16] Rusiecki J, Alexander M, Schwartz EG, et al. **2018**. The [BP] Deepwater Horizon oil spill Coast Guard cohort study. *Occup Environ Med.* **2018** Mar, 75(3):165-175. doi: 10.1136/oemed-2017-104343.

- Further, another Coast Guard cohort study found "evidence of positive associations between oil spill clean-up exposures and both acute and longer-term cardiovascular symptoms/conditions", including chest pain associated with increasing levels of crude oil exposure via inhalation and direct skin contact, and sudden heartbeat changes associated with being in the vicinity of burning oil exposure. "In prospective analyses, responders (vs. non-responders) had an elevated risk for mitral valve disorders during 2013–2015… Responders reporting ever (vs. never) crude oil inhalation exposure were at increased risk for essential hypertension, particularly benign essential hypertension during 2010–2012… Responders with crude oil inhalation exposure also had an elevated risk for palpitations during 2013–2015… Cardiovascular symptoms/conditions were generally stronger among responders reporting exposure to both crude oil and oil dispersants than among those reporting neither' (Alexander et al. 2018).[17]

- Unlike the Coast Guard study cohort, the Gulf Long-term Follow up (GuLF) study cohort represented a unique population of culturally, ethnically, and linguistically diverse peoples, and included areas with some of the highest rates of poverty and unemployment and the lowest rates of access to health care in the United States. Of the full cohort, 82.3% lived in Gulf Coast states. The GuLF study found that potential exposure to either of the Corexit dispersants used during the BP DHOS response was significantly associated with health symptoms from oil spill exposure, including cough wheezing, shortness of breath, skin irritation, burning in nose/throat/lungs, tightness of chest, and burning eyes. The last three had the strongest associations. Also, weaker, but still significant, associations were found between dispersant exposure and all outcomes except cough and itching eyes at the time of study enrollment (March 2011).[18]

- The Women and Their Children's Health (WaTCH) study involved women and their children who lived in southeast Louisiana, including a small number of responders, during the 8-months immediately following the BP DWH. Statistically significant associations between health and spill exposure were found for all thirteen physical health symptoms with the strongest associations for burning in the nose, throat, or lungs; sore throat; dizziness; and wheezing. Women who were spill responders or

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Alexander M, Engel LS, Olaiya N, et al. **2018**. The BP DHOS Coast Guard cohort study: A cross-sectional study of acute respiratory health symptoms. Environ Res. Apr, 162:196-202. doi: 10.1016/j.envres.2017.11.044.

[17] Hristina Denic-Roberts, N. Rowley, M. C. Haigney, et al. **2022**. Acute and longer-term cardiovascular conditions in the [BP DWH] oil spill Coast Guard cohort, *Environment International* 158: doi.org/10.1016/j.envint.2021.106937

[18] Resnik DB, Miller AK, Kwok RK, et al. **2015**. Ethical issues in environmental health research related to public health emergencies: Reflection on the GuLF Study. Environ. Health Propect. **Sep**, 123(9): A227-31. doi: 10.1289/ehp.1509889.

Stewart PA, Stenzel MR, Ramachandran G, et al. **2018**. Development of a total hydrocarbon ordinal job exposure matrix for workers responding to the BP DHOS: The GuLF STUDY. *J. Expo Sci Environ Epidemiol.* May, 28(3):223–230. doi: 10.1038/jes.2017.16.

McGowan CJ, Kwok RK, Engel LS, et al. **2017**. Respiratory, dermal, and eye irritation symptoms associated with Corexit™ EC9527A/EC9500A following the BP DHOS: Findings from the GuLF STUDY. *Environ Health Perspect.* Sep, 125(9): 097015. doi: 10.1289/EHP1677 commercial fishers were significantly more likely to report wheezing; headaches; watery, burning, itchy eyes; and stuffy, itchy, runny nose.[19]

During this same timeframe, a study on air quality in southeast Louisiana used all available data and health-protective standards and concluded that the ambient air concentrations of benzene and PAHs were likely a threat to public health and should have been a cause for concern *and preventative action* as the "geographic exposure disparities in air quality were measurable in real time and therefore could have been used to issue region-specific preventive health announcements and precautions."[20]

- "… the [BP DWH] oil spill of 2010 increased concentrations of PM2.5, NO2, SO2, and CO in affected coastal counties [and] increased incidence of low birth weight (<2500 g) and premature born infants (<37 weeks of gestation). Heterogeneity effects reveal more pronounced adverse infant health outcomes for black, Hispanic, less educated, unmarried, and younger mothers" (Beland and Oloomi 2019).[21]

Since this disaster, new and rare cancer clusters now dot the maps in several coastal counties from Florida to Louisiana, where childhood cancer rates have soared.[22] Yet none of this is surprising, given that this is a logical extension of the early science on the BP disaster that documented bioindicators predictive of long-term harm in people and wildlife[23] – and that it matches our lived experiences with the BP DWH oil spill disaster and others.[24]

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[19] Peters ES, Rung AL, Bronson MH, et al. **2017**. The women and their children's Health (WaTCH) study: Methods and design of a prospective cohort study in Louisiana to examine the health effects from the BP oil spill. *BMJ Open*. Jul 10, 7(7):e014887. doi: 10.1136/bmjopen-2016-014887
Peres LC, Trapido E, Rung AL, et al. **2016.** The [BP] Deepwater Horizon oil spill and physical health among adult women in southern Louisiana: The Women and Their Children's Health (WaTCH) Study. *Environ Health Perspect.* Aug, 124(8):1208–13. doi: 10.1289/ehp.1510348

[20] Nance E, King D, Wright B, Bullard RD. **2016**. Ambient air concentrations exceeded health-based standards for fine particulate matter and benzene during the BP DHOS. *J. Air Waste Manag. Assoc. Feb, 66(2):224-36. doi: 10.1080/10962247.2015.1114044.*

[21] Beland, L.-P., Oloomi, S. **2019**. Environmental disaster, pollution and infant health: Evidence from the [BP] Deepwater Horizon oil spill, *Journal of Environmental Economics and Management*, 98, 102265.

[22] Eastern Shore Community Health Project, using National Cancer Institute statistics for 2013–2017. http://easternshorechp.org/cluster-maps/

[23] D'Andrea MA, Reddy GK. The development of long-term adverse health effects in oil spill cleanup workers of the BP DHOS offshore drilling rig disaster. Front Public Health. **2018** Apr 26; 6:117. doi: 10.3389/fpubh.2018.00117

[24] Government Accountability Project, Shanna Devine and Tom Devine. **2013**. *Deadly dispersants in the Gulf: Are public health and environmental tragedies the new norm for oil spill cleanups?*
Government Accountability Project, S. Devine and T. Devine, **2015**, Addendum Report.
Government Accountability Project, T. Devine and A. Arnold, **2020**, Ten Years After [BP] Deepwater Horizon: Whistleblowers continue to suffer an unending medical nightmare triggered by Corexit.
For a synopsis of science on human health harm from oil spills and dispersant use up through April 2015, *see* Riki Ott, **2015**, Expert testimony on behalf of North Shore No Pipeline Expansion, intervenor in National Energy Board hearing on consideration of the Trans Mountain Expansion Project, British Columbia, Canada. OH-001-2014.
For a synopsis of science on human health harm from oil spills and dispersant use from May 2015 through 2018, *see* Riki Ott, **2018**. Expert testimony on behalf of North Shore No Pipeline Expansion, intervenor in National Energy Board hearing on reconsideration of the Trans Mountain Expansion Project, British Columbia, Canada. P.C. 2018-01177, MH-052-2018

And finally, relevant to the July 27 rule, when independent scientists re-analyzed BP's massive subsea dataset, they disagreed with BP's preliminary findings, concluding instead that oil distribution at depth was controlled by temperature and pressure, not SSDI.[25] The latest review by the National Academy of Sciences, sponsored in part by the American Petroleum Institute, even stated that SSDI was not effective as the available evidence from the BP DWH indicates that insignificant amounts (less than 5%) of the liquid oil was trapped as suspended microdroplets in the deep intrusion layers with or without SSDI: "Evidence… compiled by Gros et al. (2017)… indicates that ~5% or less of the liquid oil was trapped in the deep intrusion layers. *Evidence preceding the onset of SSDI is similarly consistent with low percentages of liquid oil in the deep intrusion layers* (emphasis added, p. 51).[26]

In light of the post April 2015 oil dispersant research, the rule on monitoring atypical dispersant use is clearly insufficient to safely mitigate harm from oil spills. So, too, will be the rest of the 2015 Proposed Rule, if not revised and reissued, based on current science. Our nation's oil spill emergency response plan is currently operating under science and rules governing dispersant use that are twenty-seven years old. These rules will be nearly thirty years old before they are updated in 2023. Every time dispersants are used during oil spill response, we sacrifice the health and wellbeing of ourselves, our children, our communities, and our marine life.

Fingas M., **2018**. A review of literature related to human health and oil spill dispersants, 2014–2018, on behalf of Prince William Sound Regional Citizens' Advisory Council, Anchorage, Alaska. PWSRCAC.

[25] Paris CB, Berenshtein I, Trillo ML, et al. **2018.** BP Gulf Science Data reveals ineffectual subsea dispersant injection for the Macondo blowout. Front. Mar. Sci., 30 October 2018. doi.org/10.3389/fmars.2018.00389

[26] National Academy of Sciences, Committee on the evaluation of the use of chemical dispersants in oil spill response, **2019**. *The Use of Dispersants in Marine Oil Spill Response*, Washington, D.C.

Gross et al. **2017.** Petroleum dynamics in the sea and influence of subsea dispersant injection during [BP] Deepwater Horizon. PNAS September 19, 114 (38) 10065–10070. doi.org/10.1073/pnas.1612518114

Feb 9, 2022

WHEJAC Members
White House Environmental Justice Advisory Council

Sent via electronic mail to whejac@epa.gov

Subject: Written comments as follow up to Jan. 26, 2022 WHEJAC Meeting

Dear members of the White House Environmental Justice Advisory Council,

Thank you for the opportunity to provide these comments in regard to the administration's environmental justice priorities and policies. These written comments are offered as a follow up to oral comments the Grand Canyon Trust provided at the WHEJAC's January 26th meeting.

The Grand Canyon Trust is headquartered in Flagstaff, Arizona, with satellite offices across the Colorado Plateau. Our mission is to safeguard the wonders of the Grand Canyon and the Colorado Plateau, while supporting the rights of its Native peoples. At the Trust, we envision a Grand Canyon and Colorado Plateau where wildness, the diversity of native plants and animals, clean air, and flowing rivers abound, sovereign tribal nations thrive, a livable climate endures, and people passionately work to protect the region they love for future generations. Earthjustice is the premier nonprofit public interest environmental law organization. We wield the power of law and the strength of partnership to protect people's health, to preserve magnificent places and wildlife, to advance clean energy, and to combat climate change. Earthworks is a nonprofit organization dedicated to supporting communities on the front lines of extraction and promoting sustainable alternatives to new extraction. We have staff all over the world working in solidarity with peoples fighting new and existing oil, gas, and hardrock mining projects. At all levels of government, we advocate for a circular minerals economy in which new extraction for critical minerals is a last resort and the communities that have borne the brunt of hardrock mining—namely Indigenous and poor and rural communities—are no longer considered acceptable sacrifices. NRDC is a national non-profit environmental organization with over one million combined members and activists. NRDC's activities include maintaining and enhancing environmental quality and monitoring federal agency actions to ensure that federal statutes enacted to protect human health and the environment are fully and properly implemented. Since 1970, NRDC has sought to improve the environmental, health, and safety conditions at both defense and civil nuclear facilities, including uranium recovery operations.

The undersigned groups write not as members of frontline communities themselves, but as groups who work shoulder-to-shoulder in support of communities, Indigenous communities in particular, that are and have been on the frontlines of mining-related environmental injustices. What we share in the letter below stems from our own experiences and observations in our advocacy work and should not be construed as coming from frontline communities themselves. We ask that the WHEJAC urge the Biden administration to do the necessary work to actively seek the input of impacted tribes and tribal communities. The Biden administration needs to hear directly from the people whose health and cultures are impacted by current and past hardrock mining and mineral processing, and the toxic and radioactive waste left behind.

We are grateful that the Biden administration has committed to take action against climate change and to support environmental justice communities. But we fear that the devil is in the details and the Biden administration either does not see or is not giving sufficient attention to the ways in which its own actions–even if intended to reduce carbon emissions–either are negatively impacting or could negatively impact Indigenous communities. Climate action should be approached carefully and in close consultation with those who are or would be impacted. Without a more careful approach, the administration risks worsening environmental injustices in tribal communities, not curbing them.

According to one estimate, more than 600,000 Native Americans live within 10km of abandoned mines.[1] Mineral extraction, processing, and waste is a major perpetrator of environmental injustice in America, and particularly in the West where our groups work. Mineral supply chains and waste disposal is only growing more important with the shift away from fossil fuels as the nation works to curb carbon emissions and mitigate climate change. But with the administration's focus on securing these supply chains must come a sincere effort to acknowledge, address, and avoid worsening the injustices faced by American citizens as a result of the mineral industry today.

Below are a few environmental justice-related observations and concerns we would like the Biden administration to be aware of and address. We appreciate the assistance of the WHEJAC in elevating these issues before the administration if it so chooses.

EPA Environmental Justice Screening Tool

The EPA's Environmental Justice Screening Tool is surely well-intended, but as organizations that work with communities on the frontlines of mining and milling operations, the tool misses what to us are obvious layers of impact–the proximity of a community or ancestral lands to mining or mineral processing and disposal sites. The tool allows one to add Superfund sites or sites reporting to the EPA in some fashion. But not all mine operations report to the EPA, rather they report to state environmental agencies. The Pinyon Plain uranium mine on the Kaibab National Forest, within the Havasupai Tribe's Red Butte Traditional Cultural Property, for instance, does not show up in the tool despite its decades-long impact on the tribe. Neither does the White Mesa uranium mill show up in the tool. The mill is just a few miles north and upgradient of the Ute Mountain Ute Tribe's White Mesa community and holds hundreds of acres of toxic and radioactive waste that will remain in perpetuity. The Ute Mountain Ute Tribe has long contested the mill's operations, and recently called for its closure.[2] Further, while

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[1] Lewis, Johnnye, Joseph Hoover & Debra MacKenzie. "Mining and Environmental Health Disparities in Native American Communities." Curr Envir Health Rpt (2017) 4:130–141. Springer. 26 April 2017. Page 131. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5429369/pdf/40572_2017_Article_140.pdf. Accessed 25 January, 2022.

[2] On August 24, 2021, the Ute Mountain Ute Tribal Council passed Resolution No. 2021-135 in opposition to the proposed federal "strategic uranium reserve" stating "the White Mesa community sits three miles

Indigenous nations with larger populations like the Navajo Nation show up as Communities of Color in the EJ Screening Tool, neither the Havasupai Reservation nor the Ute Mountain Ute Tribe's White Mesa community appear on the tool. These are only some examples of where the tool, in our view, falls woefully short of providing a true picture of communities at risk. We urge the Biden administration to work with impacted communities directly to continue to improve the tool, and also urge officials to not rely on the tool exclusively to identify issues of environmental justice.

A Pro-Nuclear, Pro-Mining Agenda Endangers Indigenous Communities

The world still lacks a reasonable long-term solution to nuclear waste disposal. And mining and processing uranium has already created a toxic and radioactive legacy in America, one that heavily impacts Indigenous communities today. On the Colorado Plateau, an area that comprises much of what many think of as the American Southwest, Indigenous communities are still living with uranium-contaminated land, air, and groundwater. Prioritization of nuclear power, uranium, and other hardrock mining and milling carries with it the threat of further damage from current and prospective uranium operations. The Biden administration must proceed with caution and careful attention to the needs and concerns of Indigenous communities who have already carried enough of America's pollution burden, and it must do so without transferring injustices from one impacted Indigenous community to another. The WHEJAC itself put forth recommendations to the Biden Administration in 2021 that state "100% of [Justice40] investments must do no harm to Environmental Justice communities. We want 100% Justice; it would be unreasonable to have any climate investment working against historically harmed communities."[3] A set of too-hasty, nuclear and extraction-focused climate policies risks creating this very problem.

The lasting impact of uranium mining on Indigenous peoples and cultures is extensive and cannot be ignored by the administration as it sets out to address climate change. On the Navajo Nation, for instance, which outlawed uranium mining and milling on its lands in 2005,[4] more than 500 abandoned uranium mines still litter the landscape from the last time the federal government incentivized domestic uranium mining in the 1950s, 60s, and 70s.[5] While these sites await proper cleanup, they are contaminating people's bodies, homes, and drinking water.[6] One ongoing study[7] shows that more than a quarter of over 700 study participants on the

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south of the White Mesa Mill owned and managed by Energy Fuels Resources (USA) which process radioactive waste materials, such as uranium, and has not only far exceeded the period of time for its operations, but has accepted and processed materials that were not a part of its original design and has gone beyond the purposes for which the Mill operates;" and "...the operations of the White Mesa Mill has had severe health impacts on the residents of White Mesa and should cease entirely;" https://www.grandcanyontrust.org/sites/default/files/resources/UMUT_Resolution2021-135_Opposition_Strategic_Uranium_Reserve.pdf Accessed 7 Feb. 2022

[3] https://www.epa.gov/sites/default/files/2021-05/documents/whiteh2.pdf page 57

[4] Diné Natural Resources Protection Act of 2005. https://www.nrc.gov/docs/ML0723/ML072340482.pdf. Accessed 25 January, 2022.

[5] https://www.epa.gov/navajo-nation-uranium-cleanup. Accessed 7 Feb. 2022.

[6] http://sric.org/nbcs/docs/NBCS%20update%20for%20TCRHCC%2010%2014%202021.pdf

[7] Southwest Research and Information Center. Navajo Birth Cohort Study. http://sric.org/nbcs/index.php. Accessed 25 January 2022.

Navajo Nation have elevated levels of uranium in their urine compared to 5 percent of the U.S. population as a whole.[8] Children are among those impacted: according to one update from the same study, exposures start early for children on the Navajo Nation, "by age 4, children are reaching adult concentrations [of uranium and arsenic in their bodies]."[9] Prioritizing and investing in further nuclear–and consequently–uranium development without properly addressing this problem is unacceptable.

In southeastern Utah, the White Mesa uranium mill sits just a few miles north and upgradient of the Ute Mountain Ute Tribe's White Mesa community. The Ute Mountain Ute Tribe opposes the operation of the mill[10] and worries about the mill's long-term consequences for community members, including impacts to their drinking water. The mill was originally built in 1980 for the sole purpose of operating as a conventional uranium mill. The planning documents asserted that the mill would run for 15 years. The toxic and radioactive waste from the mill's process is meant to remain on site in perpetuity, even after the mill's closure. The current closure plan for the mill's waste ponds is one that the Grand Canyon Trust argues is inadequate, and litigation is ongoing. Today, the mill's operator, Energy Fuels, continues to reinvent itself and its business plan, now advertising itself to the Department of Energy and the rest of the world as a rare earth minerals processing facility. A 2021 open house at the mill even earned a message of support from a Biden administration official at DOE.[11] In reality, the mill has seized upon regulatory loopholes that enable it to process radioactive waste streams (that are called "alternate feed") for fees amounting to "$5-$15 million each year,"[12] so long as it is primarily uranium that the mill extracts from the waste, and not some other substance. And the Department of Energy has granted the mill funding to explore production of rare earth element products.[13] This has allowed the mill to transform itself into a long-term disposal facility for toxic and radioactive waste from around the country and the world in the name of "recycling," while increasing the amount of toxic and radioactive waste that will be the forever-neighbor to the Ute Mountain Ute Tribe's White

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[8] Morales, Laurel. "For the Navajo Nation, Uranium Mining's Deadly Legacy Lingers." National Public Radio. April 10, 2016. https://www.npr.org/sections/healthshots/2016/04/10/473547227/for-the-navajo-nation-uranium-minings-deadly-legacylingers. Accessed 25 Jan 2022.

[9] "Navajo Birth Cohort Study initiated in 2010 to address the impacts of uranium exposure on child health outcomes." Oct. 14, 2021
http://sric.org/nbcs/docs/NBCS%20update%20for%20TCRHCC%2010%2014%202021.pdf Slide 7. Accessed 7 Feb. 2022. (Enclosed as Attachment).

[10] On August 24, 2021, the Ute Mountain Ute Tribal Council passed Resolution No. 2021-135 stating: "...the operations of the White Mesa Mill has had severe health impacts on the residents of White Mesa and should cease entirely…;"
https://www.grandcanyontrust.org/sites/default/files/resources/UMUT_Resolution2021-135_Opposition_Strategic_Uranium_Reserve.pdf Accessed 7 Feb. 2022. (Enclosed as attachment).

[11] Podmore, Zak. "Is Utah's uranium industry key to a green energy future or a radioactive threat? High uranium prices and new rare earth production capabilities at the White Mesa Mill could revive the aging facility in San Juan County." Salt Lake Tribune. Sept 26, 2021.
https://www.sltrib.com/news/2021/09/22/uranium-company-wants/ Accessed 7 Feb. 2022.

[12] "Energy Fuels Inc. (UUUU) CEO Mark Chalmers On Q2 2020 Results - Earnings Call Transcript." SeekingAlpha.com. August 29, 2020. https://seekingalpha.com/article/4366563-energy-fuels-inc-uuuu-ceo-mark-chalmers-on-q2-2020-results-earnings-call-transcript. Accessed 7 Feb. 2022..

[13] "Energy Fuels wins $1.75m award from US Department of Energy." https://www.mining.com/energy-fuels-wins-1-75m-award-from-department-of-energy/. Accessed 7 Feb. 2022.

Mesa community. The mill has also sought contracts with the EPA to take higher grade wastes from abandoned mines on the Navajo Nation.[14]

Not far away, near the Grand Canyon, a region sacred to at least eleven Indigenous tribes and nations, uranium mining companies have not only left contamination from past operations,[15] but seek to continue and expand operations today that threaten the well being of tribes–including the Havasupai Tribe which still resides within the Canyon's walls–and the critical water systems that make up the greater Grand Canyon watershed. After uranium prices briefly spiked to an all-time high in 2007, thousands of mining claims were staked near the Grand Canyon.[16] Since 2008, many tribes have worked with elected leaders, community members, and non profit organizations in pursuit of a permanent ban on new uranium mines on federally managed lands adjacent and hydrologically connected to the Grand Canyon. Such a ban would protect critical water sources and the region as a whole, which is culturally and spiritually significant to, and a homeland for the Havasupai, Hopi, Hualapai, Kaibab Band of Paiute, Las Vegas Band of Paiute, Moapa Band of Paiute, Navajo Nation, Paiute Indian Tribe of Utah, San Juan Southern Paiute, The Pueblo of Zuni, and the Yavapai-Apache Nation. To-date, the effort to ban mining permanently in the region has not yet succeeded and federal actions–including some currently proposed–only increase the pressure on the region.

In particular, the proposed "strategic uranium reserve," a holdover concept from the Trump administration for which the Department of Energy in 2021 put out a request for information,[17] is of concern. While the RFI states the DOE does not intend for a uranium reserve program to initiate production on tribal lands, the RFI states "[t]he Department expects the acquisition of natural uranium to result in new uranium production at existing domestic sites." The RFI also states "uranium newly-produced from 'alternate feed' materials are eligible to be bid for sale."[18] Indigenous communities are not just impacted by operations on reservation lands. Indigenous peoples have existing connections to lands far beyond, and far older than the boundaries that the U.S. government has drawn. For example, both of the aforementioned production types deemed acceptable for government purchase in the DOE's uranium reserve RFI incentivize and support operations at the White Mesa Mill and an existing uranium mine in the Grand Canyon Region, the Pinyon Plain Mine (formerly called Canyon Mine). Both of these operations have

[14] Crux Investor. "Uranium: Energy Fuels - Owning Mill Creates Value. Here is Why." At 00:27:50. https://www.youtube.com/watch?v=cVL4jomk8QI. Accessed 7 Feb. 2021.
[15] Reimondo, Amber. "Uranium Mining in the Grand Canyon Region." Grand Canyon Trust. Report. Feb 2019. https://www.grandcanyontrust.org/uranium-mining-grand-canyon-region Accessed 8 Feb. 2022.
[16] Smith, Stephanie. "Map of Mining Claims Around Grand Canyon Prior to 2012." Grand Canyon Trust. https://www.grandcanyontrust.org/mining-claims-grand-canyon-before-2012-mineral-withdrawal Accessed 8 Feb. 2022.
[17] US Department of Energy. "Request for Information Regarding Establishment of the Department of Energy Uranium Reserve Program" 11 Aug. 2021. https://www.federalregister.gov/documents/2021/08/11/2021-17145/request-for-information-regarding-establishment-of-the-department-of-energy-uranium-reserve-program. Accessed 7 Feb. 2022.
[18] US Department of Energy. "Request for Information Regarding Establishment of the Department of Energy Uranium Reserve Program" 11 Aug. 2021. https://www.federalregister.gov/documents/2021/08/11/2021-17145/request-for-information-regarding-establishment-of-the-department-of-energy-uranium-reserve-program. Accessed 7 Feb. 2022.

direct and longstanding implications for Indigenous communities despite not being within reservation boundaries. The White Mesa Mill is on mostly fee land directly adjacent to the northern boundary of Ute Mountain Ute Tribal lands[19] and the Pinyon Plain mine sits within the Red Butte Traditional Cultural Property–a site on the Kaibab National Forest that is sacred to the Havasupai Tribe.[20] The Pinyon Plain mine is less than ten miles from the South Rim of the Grand Canyon and sits atop a regional aquifer that is the source of Havasu Creek, which flows through the Havasupai Tribe's remote village of Supai. The Havasupai Tribe has long opposed the Pinyon Plain Mine for the environmental and cultural threats it poses to the Havasupai People.[21]

Despite some improvements to the regulatory framework that governs mineral extraction in America, it is far from adequately protective of frontline communities. One reason for this is that there remains no reasonable way for communities to balance cultural and environmental protection with hardrock mining. That's because the 1872 Mining Law declares hardrock mining–including uranium mining–to be the "highest and best use of the land." Unlike for leasable minerals, uranium or other hardrock mines can only be prevented in places with important non-mining values by an act of Congress (e.g. a permanent mineral withdrawal or National Park designation) or use of Presidential authority under the Antiquities Act. All of these options require extraordinary time and resources from the communities that wish to protect certain landscapes and communities often must do so in the face of the lobbying power of the mining industry, which spent over $18 million in 2021 alone to lobby U.S. officials.[22]

Reform the 1872 Mining Law

Hardrock mining, including uranium mining, has left a lasting and tragic impact across the West. Indigenous tribes and nations continue to live with the consequences of mining operations that were and still are given precedence over other land uses because of the grossly antiquated 150-year-old Mining Law of 1872.[23] Tribal communities today live adjacent to radioactive and toxic waste that comes from mining and processing everything from uranium ore to rare earth metals. And the needed work of transitioning away from fossil fuels to more renewable sources of energy comes with additional mining and processing risks for Indigenous communities across the country.

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[19] Denison Mines (USA) Corp., "White Mesa Uranium Mill, Environmental Report In Support of the License Renewal Application," License Renewal Application, State of Utah Radioactive Materials License No. UT1900479, Feb 28, 2007. Pages 13-15. (Enclosed as Attachment)

[20] Ross, Jamie. "Grand Canyon Tribe Fights to Protect Sacred Mountain From Mining." *Courthouse News Service*. Dec. 15, 2016. https://www.courthousenews.com/grand-canyon-tribe-fights-to-protect-sacred-mountain/ Accessed 9 Feb. 2022.

[21] Havasupai Tribe. "The Havasupai Tribe Comments Regarding the Pinyon Plain Mine Individual Aquifer Protection Permit." Letter. 6 Aug. 2021. https://www.grandcanyontrust.org/havasupai-tribe-comments-canyon-mine-permit Accessed 8 Feb. 2022.

[22] "Industry Profile: Mining." Open Secrets: Following the Money in Politics. https://www.opensecrets.org/federal-lobbying/industries/summary?cycle=2021&id=E04 Accessed 7 Feb. 2022.

[23] Earthworks. Mining 101. https://earthworks.org/issues/mining/. Accessed 25 January 2022.

Almost all hardrock mining in the United States has been governed by the same law since 1872, the age of manifest destiny, before women and people of color were able to vote, and has had only minimal adjustments and no comprehensive reform. Legislative reform has always been of interest to representatives of mining-dependent states, but thanks to the lobbying of the mining industry, their objectives have failed to materialize.

However, the Biden administration has an opportunity to fundamentally change hardrock mining through rules in the Bureau of Land Management (BLM) and the US Forest Service within the Departments of Interior and Agriculture, respectively. In 2021, nine Indigenous communities and organizations and thirty-one conservation groups filed a petition with the BLM asking them to redo the rules governing hardrock mining on federal lands.[24] We strongly encourage the administration to accept our petition and update the rules accordingly. Proposed revisions include:

- Clarifying that the BLM must use its authority to protect tribal and cultural resources and values, wildlife, and water quality and quantity;
- Requiring the BLM to verify mining rights; and
- Closing loopholes that allow the mining industry to escape public review and consultation with local tribes and governments.

With the inevitable transition away from fossil fuel energy upon us, it is extremely urgent that policymakers and other people in power recognize the deeply racist past and present of hardrock mining and radioactive waste in order to stop the cycle of violence before it harms more communities and their environments. If federal and state governments do not actively change the policies and rules that govern hardrock mining, and look at the full impact of the nuclear fuel cycle, more communities will face health impacts like cancer, asthma, and renal disease;[25] more Indigenous people will face continued genocide;[26] more Indigenous women, girls, and Two-Spirit and LGBTQ+ peoples will face sexual violence;[27] and more biodiversity will be destroyed. These are just a few examples of the violence hardrock mining and the nuclear fuel cycle has inflicted upon mining-affected communities, especially Indigenous communities,[28] and it will continue to do so unless the federal government gets serious about prioritizing people over the demands of the extractive industry. More extraction and short-sighted policies aren't the only way forward: intentional, frontline community-informed recycling, reusing, substitution,

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[24] Earthworks. Tribes, Indigenous Groups, Conservation Organizations File Petition to Strengthen Federal Mining Rules. https://earthworks.org/media-releases/tribes-indigenous-groups-conservation-organizations-file-petition-to-strengthen-federal-mining-rules/. Accessed 25 January 2022.
[25] Congress.gov. H.R.5338 - Radiation Exposure Compensation Act Amendments of 2021. https://www.congress.gov/bill/117th-congress/house-bill/5338/text?q=%7B%22search%22%3A%5B%22HR+5338%22%2C%22HR%22%2C%225338%22%5D%7D&r=5&s=2. Accessed 25 January 2022.
[26] Penteah, Val R. "Pueblo of Zuni Letter to President Biden." Censored News. 2021.
[27] CCSG Association. Never Until Now: Indigenous and Racialized Women's Experiences Working in Yukon and Northern British Columbia Mine Camps. http://www.liardaboriginalwomen.ca/index.php/never-until-now-laws-mining-report/file. Accessed 25 January 2022.
[28] Lorenzo, June. "Gendered Impacts of Jackpile Uranium Mining on Laguna Pueblo." International Journal of Human Rights Education. 2019.

and efficiency improvements are legitimate alternatives, but need government support focused on benefitting all communities, not just those that are predominantly white and well-to-do.

Close Clean Water Act Loopholes

One of hardrock mining's most devastating impacts is water pollution: according to the EPA hardrock mining has contaminated 40 percent of all western watersheds' headwaters.[29] This is in part because of two loopholes in EPA and Army Corps of Engineers regulations: in the 2002 revision of the Clean Water Act "fill material" was rewritten to include mine waste, and water-defining regulations allow mine developers to designate natural water sources as "waste treatment systems" exempt from the Clean Water Act.[30], The Biden administration is in the process of revising the regulations implementing the Clean Water Act,[31] and closing these two loopholes is crucial to ensure mining-affected communities, and everyone who depends on freshwater (which is all of us), are prioritized above mining companies' profits.

Mining Does Not Belong in FAST-41

Approximately a month before President Biden was sworn into office, the Trump administration added hardrock mining to the Fixing America's Surface Transportation (FAST-41) program,[32] which was established by legislation passed in 2015 to fund and expedite permitting for infrastructure projects. Hardrock mining does not belong in FAST-41, not least of which because it does not pertain to either the surface or transportation. Its addition was a ploy to further minimize community input and consultation to benefit mining operators. Thus far, the Biden administration has not announced any interest in removing hardrock mining from FAST-41, which exacerbates environmental injustice and racism every day. The removal of mining from FAST-41 does not require Congressional approval, and the positive impact would be significant. We strongly encourage the Biden administration to act immediately.

Need for EPA ISL Uranium Rulemaking

Finally, it is long past time for EPA to act on the much-delayed In Situ Leach (ISL) uranium rulemaking (Health and Environmental Protection Standards for Uranium and Thorium Mill Tailings (40 CFR Part 192)). The rulemaking was abandoned by former President Trump's EPA in 2018. If EPA does not act now by re-proposing and then finalizing the ISL uranium rulemaking, frontline communities across the West and billions more gallons of scarce groundwater could be permanently contaminated by domestic ISL uranium mining across the arid American West, from Texas to Wyoming.

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[29] Earthworks. Mining 101. https://earthworks.org/issues/mining/. Accessed 25 January 2022.

[30] Earthworks. Loopholes in the Clean Water Act. https://earthworks.org/issues/loopholes_in_the_clean_water_act/. Accessed 25 January 2022.

[31] US Environmental Protection Agency. EPA and Army Take Action to Provide Certainty for the Definition of WOTUS. https://www.epa.gov/newsreleases/epa-and-army-take-action-provide-certainty-definition-wotus. Accessed 25 January 2022.

[32] Earthworks. STATEMENT: Earthworks Opposes Adding Mining to FAST-41. https://earthworks.org/media-releases/statement-earthworks-opposes-adding-mining-to-fast-41/. Accessed 25 January 2022.

Conclusion

Climate action is urgent and important, but without appropriate actions by the Biden administration, the shift away from fossil fuels could lead to more significant impacts on frontline, particularly Indigenous communities across the country. The nuclear fuel cycle–from mining to processing and disposal–and hardrock mining in general, have already left a lasting toxic and radioactive legacy for Indigenous communities. It is incumbent upon this administration to lead the way toward a livable future for every American and to take every precaution in order to advance climate action, without further burdening Indigenous tribes and nations with the consequences of America's energy and mineral demands. Thank you to the WHEJAC for the work that you do and for considering the issues we have raised in this letter as you advise the policies and priorities of the administration.

Sincerely,

Amber Reimondo

Amber Reimondo
Energy Director
Grand Canyon Trust

/s/
Blaine Miller-McFeeley
Senior Legislative Representative
EarthJustice

/s/
Aaron Mintzes
Senior Policy Counsel
Earthworks

/s/
Geoffrey Fettus
Senior Attorney, Nuclear Climate and Clean Energy Program
Natural Resources Defense Council

3 Enclosures

- Denison Mines (USA) Corp., "White Mesa Uranium Mill, Environmental Report In Support of the License Renewal Application," License Renewal Application, State of Utah Radioactive Materials License No. UT1900479, Feb 28, 2007.
- Southwest Research and Information Center, Navajo Birth Cohort Study Update, Oct 14, 2021
- Ute Mountain Tribal Council Resolution No. 2021-135, "Opposition to the Proposal by United States National Nuclear Security Administration, Department of Energy to

Establish a Strategic Uranium Reserve and Authorization to Submit Comments in Opposition." August 24, 2021

Using Moss as a Biomonitor to Detect Atmospheric Sources of Mercury

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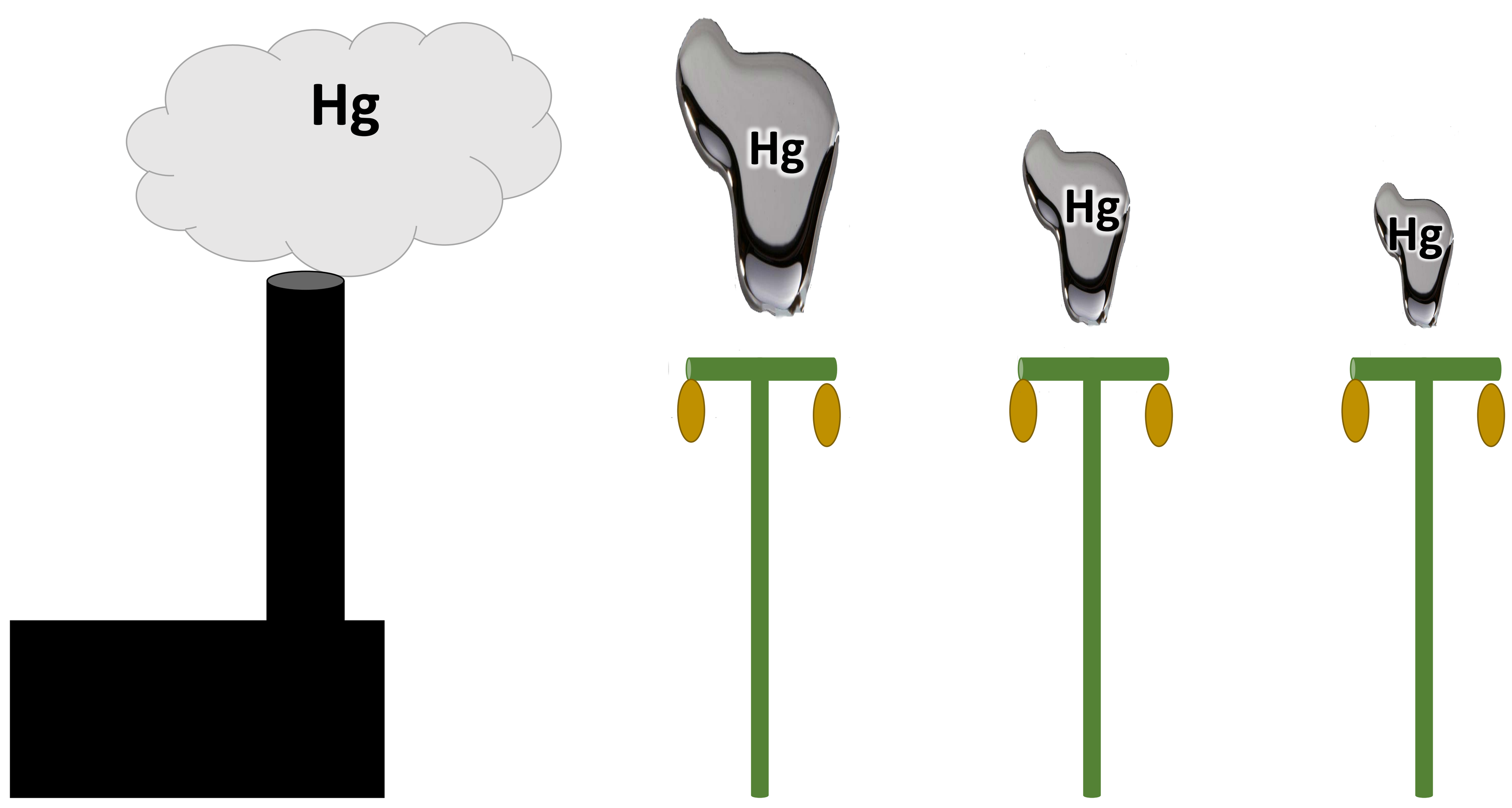
Introduction

- Waste incinerators are anthropogenic pollution sources, which emit elemental (Hg^0) and inorganic mercury (Hg^{2+}) to the atmosphere.
- Airborne deposition is a primary pathway for mercury movement to surface waters, but atmospheric mercury (Hg) monitoring can be technically challenging and expensive.
- Moss as a biomonitor is an inexpensive alternative to traditional monitoring methods which allows for qualitative comparison of atmospheric Hg emissions at different locations.
- Moss has a high number of proton binding sites, and Hg binds to its oxygen-lacking functional groups.
- Moss has been used as biomonitor for atmospheric Hg pollution in rural environments, where background Hg concentrations are low.
- Moss' sensitivity to Hg in a non-pristine environment has yet to be tested.
- If moss is sensitive enough to accumulate mercury from a small point source in an urban environment, mercury concentration in moss samples would decrease as distance from the incinerator increased.



Methods

- In the Summer of 2015, 21 samples of moss were distributed within 5 km of a municipal sewage sludge incinerator.
- In the Summer of 2016, 19 samples of moss were placed in three transects within 5 km around the municipal sludge incinerator in New Haven, Connecticut from June 30 – July 21, 2016.
- The samples were collected, homogenized using a Thomas Wiley Mini Mill, and then stored in the dark at -15 °C until analysis.
- For Hg analysis, 8 – 12 mg aliquots were microwave acid digested in Teflon sleeves using 10% HNO_3 and NIST 1547 peach leaf standard reference material was run coincident with samples to test the performance of the method.
- Digested samples were analyzed using a Tekran Model 2600 CVAFS Mercury Analysis System.

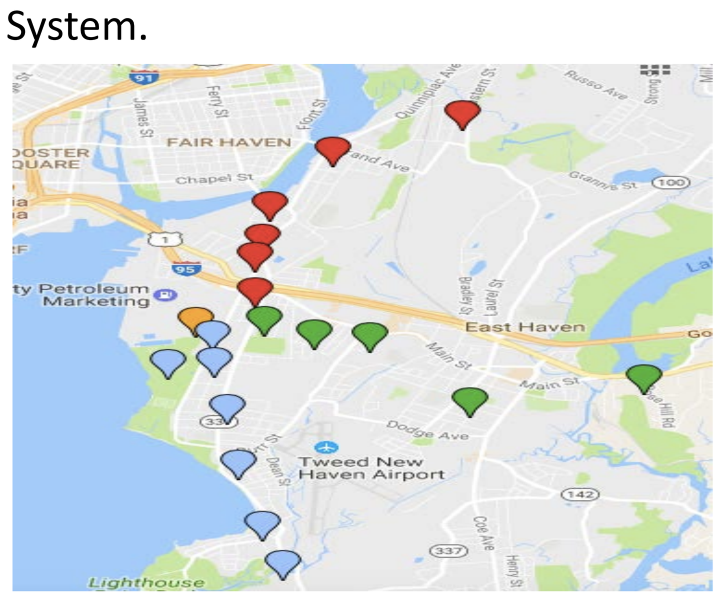


Figure 1. Map illustrating the 19 sites where moss bags were exposed in 2016. Sample sites were separated into north transect (red), east transect (green), and south transect (blue).

Figure 2. Moss samples after microwave assisted acid digestion in preparation for Hg quantification with Tekran 2600.

Conclusions

- **Moss is an effective biomonitor** for mercury from a small point source in an urban environment.
- Our data suggests that the **sewage sludge incinerator in New Haven, CT is a source** of atmospheric mercury pollution to the area.

Results

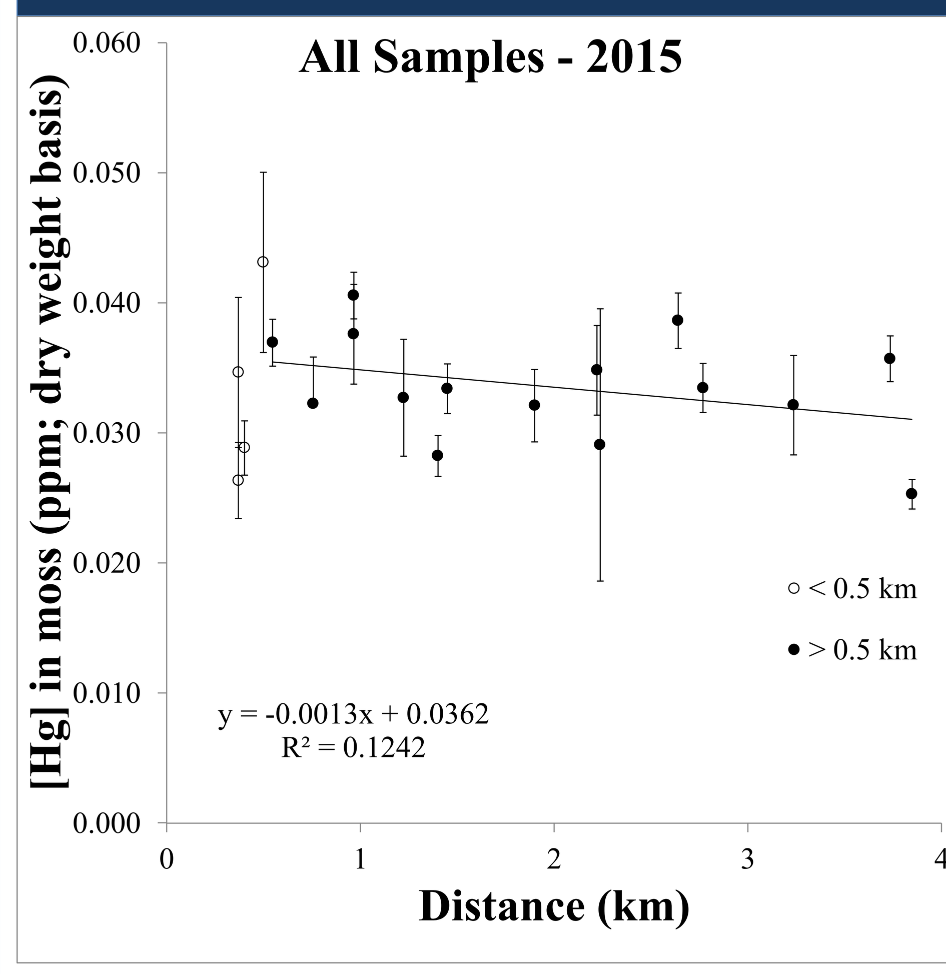


Figure 3. Data from pilot study performed in the summer of 2015 shows that as distance from the incinerator increased, mercury concentrations in the moss decreased. Error bars represent the 95% confidence intervals for analytical replicates run for moss at each sample location. Note that the rings represent sites that were < 0.5 km from the incinerator, and these are not included in the linear regression.

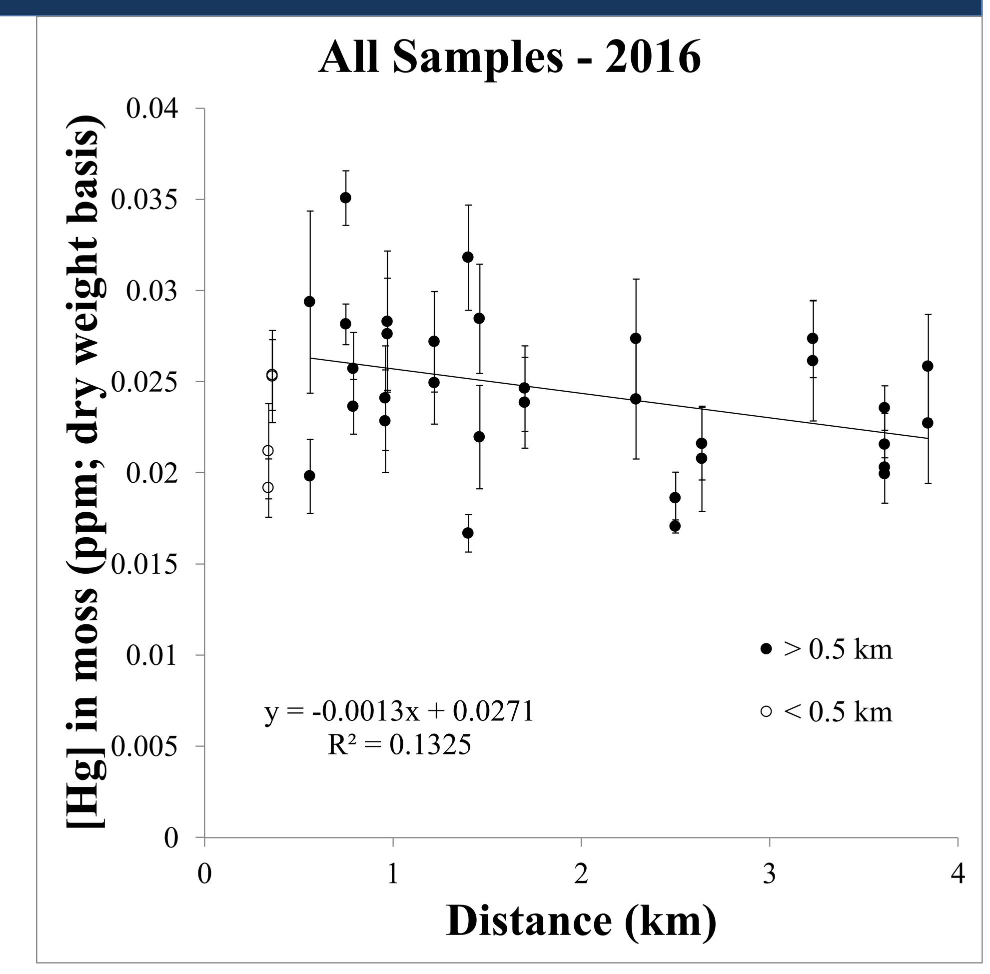


Figure 4. Mercury concentrations in all moss samples for the summer 2016 campaign showed decreasing concentrations as distance from the incinerator increased. Each site had duplicate moss sample placement, and error bars for each datum represent 95% confidence intervals for the analytical replicates run for each moss sample. Sites were separated into near (< 0.5 km) and far (> 0.5 km), where the near sites are indicated as rings and these were not included in the linear regression.

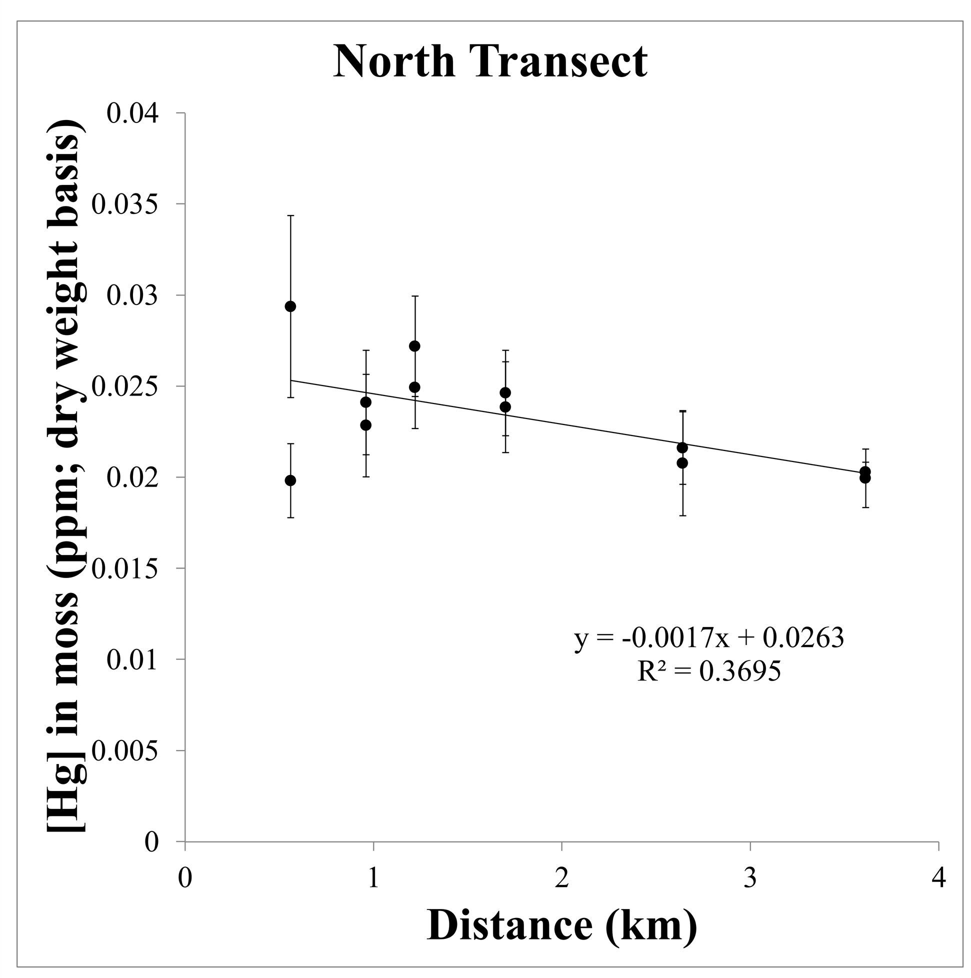


Figure 5. Mercury concentrations in moss samples from the north transect showed a strong, negative linear relationship with increasing distance from the incinerator. Error bars represent the 95% confidence intervals for the average of analytical replicates run for each moss sample.

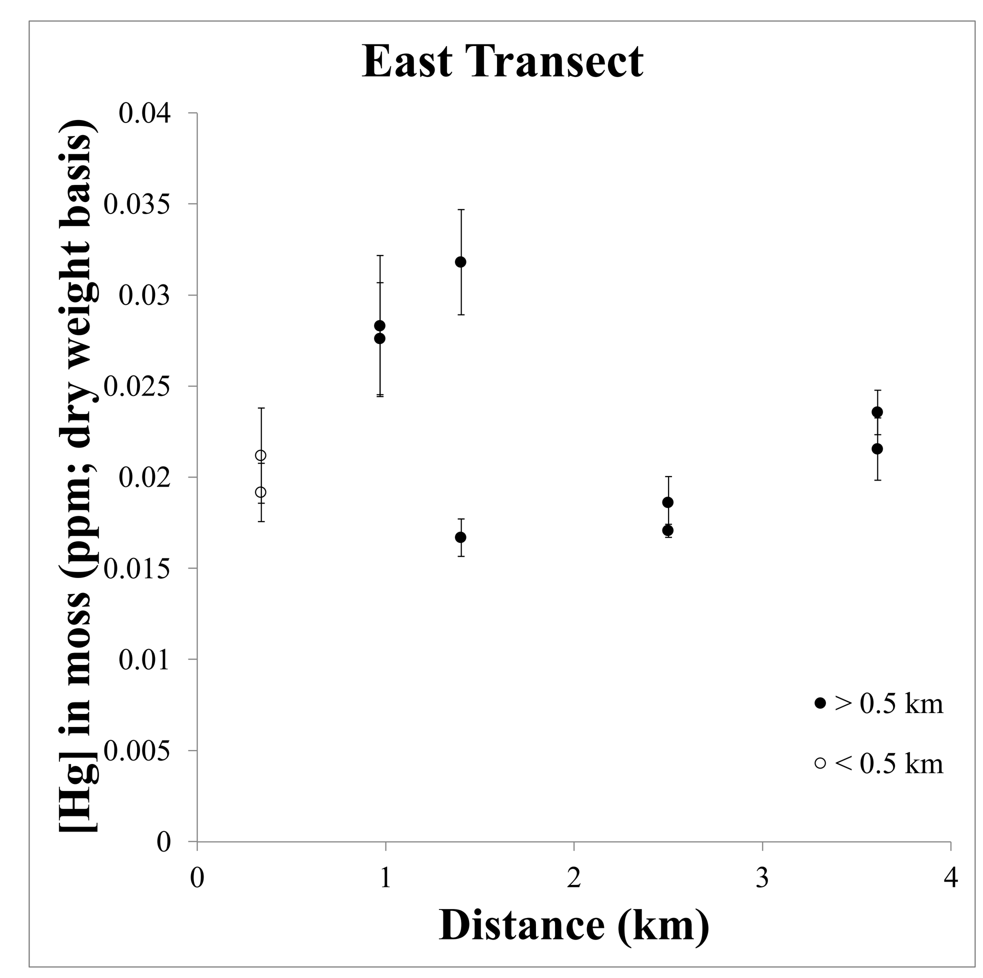


Figure 6. There was no significant trend observed for the moss samples placed in the east transect. Error bars represent the 95% confidence intervals for the average of analytical replicates run for each moss sample.

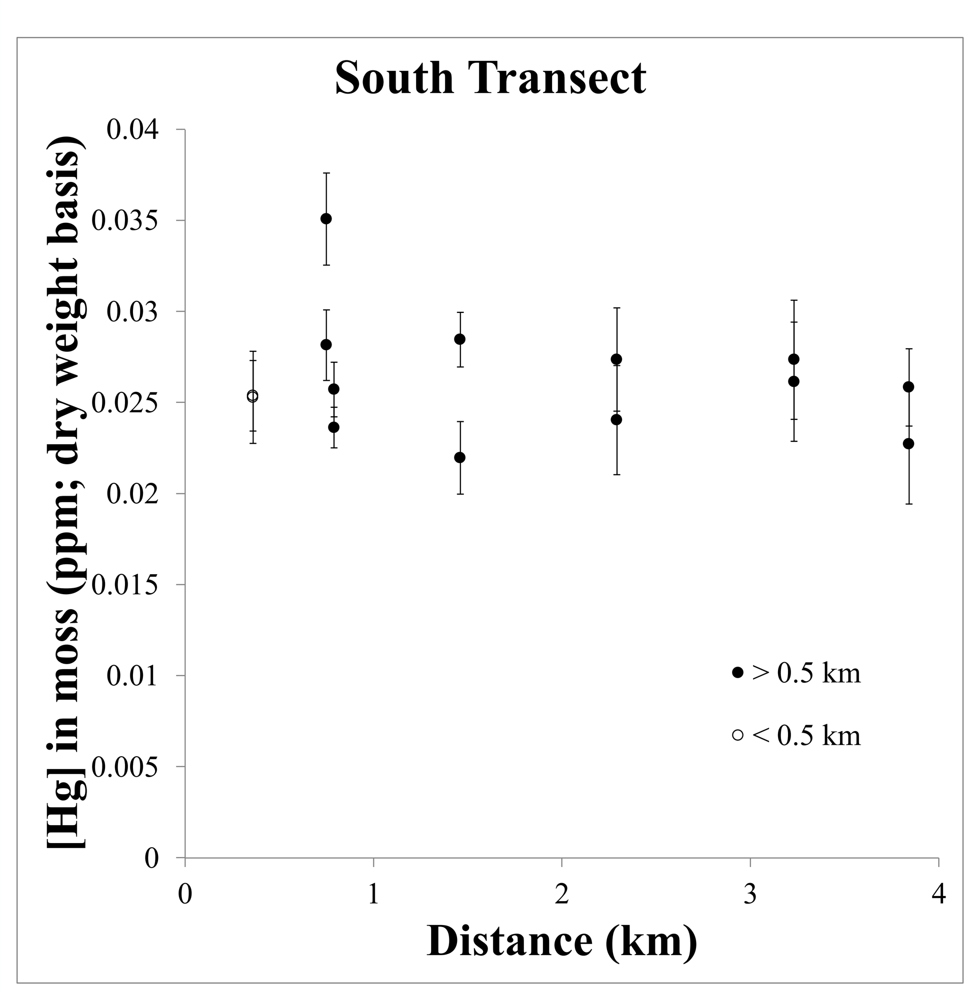


Figure 7. Mercury concentrations in moss samples from the south transect displayed no relationship. Error bars represent the 95% confidence intervals for the average of analytical replicates run for each moss sample.

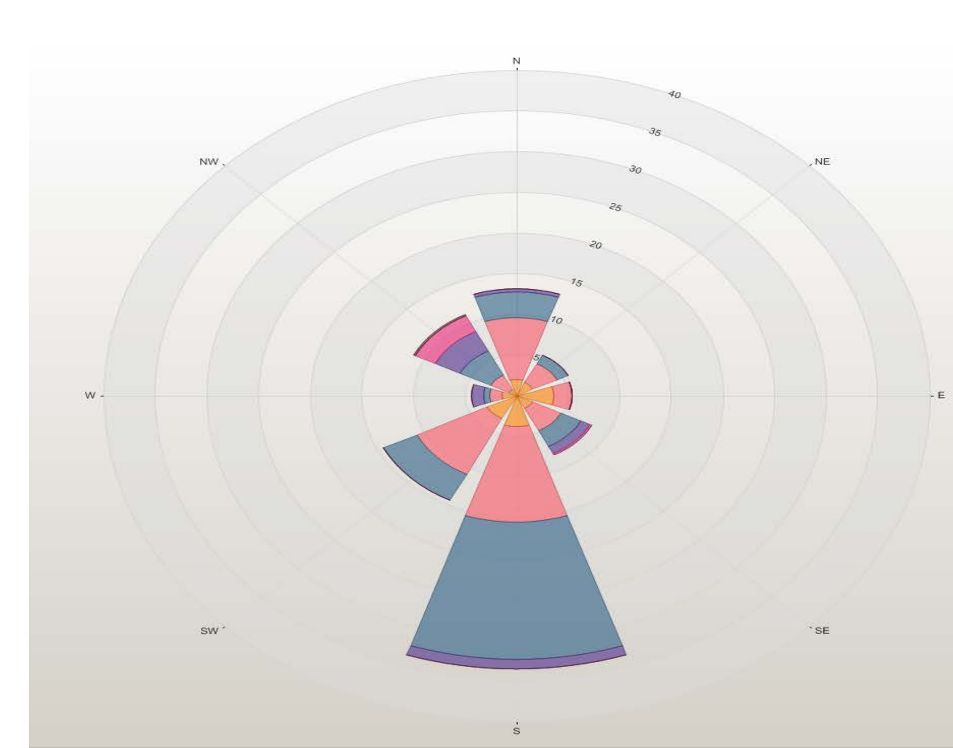


Figure 8. Wind rose data from a nearby local meteorological station (Criscuolo Park) shows that during the sampling period, the dominant wind direction was from the south to north. These winds would also push mercury emitted from the incinerator, which may contribute to the strength of the relationship observed in Fig. 3, relative to the weakness/lack of a relationship observed in Figs. 6 and 7.

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Rural Power Coalition White House Environmental Justice Advisory Council (WHEJAC) Public Comment

Docket ID: EPA-HQ-OA-2021-0683

Rural Electric Cooperatives (RECs) serve 42 million Americans and power 56% of the nation's landmass. RECs cover 90% of counties federally-recognized for persistent poverty, yet the member-owners of those utilities face some of the highest energy burdens—percentage of income spent on energy bills—in the country. These counties include broad swaths of the Black Belt region, Appalachia, Tribal Lands, and the US border, where many economically-disadvantaged Americans live. For this reason, it is essential that the path to a lower carbon future does not drive electric cooperatives, or the communities that own them, further into debt.

RECs directly own over 300 fossil fuel power plants in the United States, emitting over 130 million metric tons (mmt) of carbon pollution annually. Though they serve just over 12% of the U.S. population, RECs account for over 20% of all electrically-generated carbon emissions, with nine of the most carbon-intensive electric utilities in the US energy sector being owned by RECs.

To meet the necessary goal of creating a carbon pollution-free power sector by 2035, the REC sector must retire these plants and replace them with carbon-free energy sources—and they must do so in just 14 years.

Without federal relief, the only way that RECs can finance clean energy investments is via new debt. Unfortunately, the REC sector already holds roughly $100 billion in debt, nearly half of which is financed directly by the federal government. To put this into context: East KY Power Cooperative owes the Rural Utility Service (RUS) more than $2.1 billion for energy infrastructure that needs to be retired. They carry daily debt service of $563,000. That's an average of $29/month in debt service for every meter served.

Electric cooperatives and member-owners have been at a historic disadvantage. Even in the face of insufficient energy infrastructure and devastating environmental impacts (like mountaintop removal coal mines and air and water pollution from coal plants) rural communities were last to get electricity to their homes. RECs have also been historically disadvantaged by federal incentives for clean energy, while their investor-owned counterparts have been able to leverage robust state and federal tax credits for proactive investments.

Without federal funding, these communities will be the last to transition to clean energy, or will be unable to decide their own clean energy futures, while outside companies buy up rural land for large scale solar projects, lining the pockets of out of state developers. Rural communities require an equitable and just energy transition that promotes local ownership and local wealth building.

Federal investments in Rural America have been proven to be effective and necessary. The Rural Electrification Act of 1936 brought electrification to the countryside. As a result of this renowned New Deal program, electrification of rural areas (including homes and businesses) increased from 10% to 80% between 1936 and 1950. Unfortunately, the economics and social conditions have changed relatively little since the first power lines went up—some have even gotten worse.

Today, faced with the economic and climate imperative to transition to a new energy system, rural communities will once again require major federal investment.

The Rural Power Coalition (RPC) represents the national movement for electric cooperative reform that is being led by member-owners and advocacy groups across the country. Specifically, the RPC is asking the White House Environmental Justice Advisory Council to recommend the Biden Administration:

- Ensure any debt relief to Rural Electric Cooperatives is conditional on new investment in local energy upgrades in rural communities on terms that are broadly inclusive. (It's imperative that the benefits of federal support reach rural communities!)
- Ensure that Justice 40 recognizes and includes in its definition the counties that are already federally recognized for persistent poverty. These are counties that have had more than 20% of the population living below the federal poverty line for at least three census reports, or 30 years.
- Ensure that President Biden fulfills his pledge as a candidate to implement the 10-20-30 rule. This is a policy championed by Rep. Clyburn and Sen. Booker to assure that at least 10% of federally funded programs be directed toward persistent poverty communities.

- Ensure the Department of Energy actively participates in a whole-of-government response. The DOE has the capacity to work with every single Rural Electric Cooperative in the country to chart a technology transition path, but right now, it is helping very few.
- Ensure meaningful engagement at a community level for every new application requesting federal financing from the Rural Utilities Service—and for every requested issuance of debt for the Tennessee Valley Authority.
- Treat electric cooperatives fairly in reform of renewable energy tax credits so that they receive the same vital option for direct payment that is afforded to most other utilities.

Sincerely,
Rural Power Coalition 2/3/22
https://www.ruralpower.us/
info@ruralpower.us

Appalachian Voices (NC)
CURE (MN)
Kentuckians for the Commonwealth (KY)
Mountain Association (KY)
Partnership for Southern Equity (GA)
RENEW Missouri (MO)
Western Organization of Resource Councils (MT)

Complaint Details

Status	SENT Submission Log
Date/Time	Feb 2, 2022, 12:20 am
Type	Commercial
Operation	Arrival
Dist / Alt	0.57 mi / 2500 ft
Operator	PrivatAir Saudi Arabia
A/C Type	B738 - Boeing 737NG 8DR/W BBJ2
Callsign	---
Registration	VP-COH
From - To	
Route of Flight	
FlightAware Track	
Weather	
Traffic Source	ADSB

‹ Back

From: **Karen Spencer** KFSpencer@comcast.net
Subject: Public Comment: SAB review for Draft CCL 5 per SDWA
Date: February 4, 2022 at 12:40 PM
To: kilgore.carolyn@epa.gov

Since first enacted in 1974, the Safe Drinking Water Act has stated that "[n]o national primary drinking water regulation may require the addition of any substance for preventive health care purposes unrelated to contamination of drinking water." - *SDWA (P.L. 93-523)*

"New evidence questions existing policies about the safety of fluoride for babies' developing brains. Given that safe alternatives are available and that there is no benefit of fluoride to babies' teeth before they erupt or appear, it is time to protect those who are most vulnerable." *- Bruce Lanphear MD, PhD; Christine Till PhD; & Linda S. Birnbaum PhD in "It is time to protect kids' developing brains from fluoride." Environmental Health News (October 7, 2020)*

Re: https://sab.epa.gov/ords/sab/f?p=100:18:2011705921474:::RP,18:P18_ID:2600

Dear Ms. Kilgore and SAB,

Please accept the attached public comment recommending a review of modern science relevant to establishing an updated MCLG and MCL for fluoride in the drinking water that is protective of susceptible sub-populations. This applies to fluoride in water generally and HFS, FSA, HFSA, $Na_2[SiF_6]$ & NaF supplementation specifically.

Artificial fluoridation directly impacts approximately 75% of the US population living in communities with fluoridation schemes and any who consume food or drink prepared with that water, i.e. those subjected to the 'halo effect.'

SABreSDWA2022.02.pdf

Karen Spencer
Gloucester, MA
978.283.4606
Subscribe on YouTube
See the Call to Action

More power to you if fluoridation doesn't bother you, but not the power to assume it's safe for your neighbor with kidney disease, his pregnant wife or their diabetic daughter!

About Karen: Currently a semi-retired consultant working with software development teams, Karen Spencer is a former analyst and project leader. She is adept at conducting research and analyzing trends. Her special interests include critical thinking, data-driven decision making, and organizational theory. She and others in her family are among the 15% of Americans with chemical sensitivities triggered by exposure to fluoridated food and drink. Karen's most recent publications were featured in:
Medical Hypotheses (2018): https://www.sciencedirect.com/science/article/pii/S0306987718308600
GreenMed (2019): https://www.greenmedinfo.com/blog/wetoo-medical-assault-and-battery

Karen Spencer

67 Langsford Street, Gloucester MA 01930
h: 978.283.4606 ✧ KFSpencer@Comcast.net

February 4, 2022

To: Science Advisory Board (SAB)

Re: Contaminant Candidate List (CCL) under the Safe Drinking Water Act (SDWA).

Attn: kilgore.carolyn@epa.gov

Dear Board,

As you know, water concentrations of substances like fluoride are measured in parts per million (ppm) and doses consumed by people are measured as milligrams per liter (mg/L). These measurements are considered the equivalent of each other, so when we assume a fluoride dose for a 'typical' adult, we are assuming a consumption of one liter per day at a one ppm concentration of fluoride in that water.

However, because our assumption does not take into consideration susceptible populations like diabetics or kidney patients who can consume six liters of water per day, or the fetuses of pregnant women, bottle-fed babies, young children and the elderly for whom lower doses are advisable, the EPA has a rule of thumb that a safety factor (SF) of at least ten should apply to water contaminants based on the lowest observable adverse effect level (LOAEL) in order to protect these groups. Fluoride, despite dental claims of mineral status, is not a nutrient. It is an enzyme poison and water contaminant to which that EPA standard applies.

In the late 20th century, the assumptions promoted by fluoridation advocacy groups were that:

1) Fluoride needed to be consumed in childhood in order to effectively protect teeth
2) Consumption up to 10 milligrams (mg) per day was safe for adults
3) Consequently, the EPA considered a 1ppm fluoride concentration in water as safe for all consumers as it was one-tenth of 10 mg/day.

Those 20th century assumptions have been recognized as untrue in the 21st century.

For the moment, let's ignore the fact that the CDC admitted in 1999 and 2001 that any dental benefit from fluoride is due to topical exposure in high concentrations such as those found in toothpaste, not systemic low exposure. The CDC qualified their statements with the word "predominantly" in order to not dismantle the fluoridation program which they had recently declared one of their top ten achievements in the 20th century. Since fluoride is readily and cheaply available for either topical use or consumption, any dental benefits are a moot point. The matter at hand is the safe dose.

In 2006, the National Research Council (NRC) advised the EPA that its 4 ppm maximum contaminant level goal (MCLG) for fluoride was not protective of human health and that the NRC could not find any evidence of safety for susceptible populations at any dosage. The EPA failed to take action to change either the 4 ppm MCLG and MCL or 2 ppm SMCL, even when a team of EPA scientists determined that fluoride is one of fewer than two dozen 'gold standard developmental neurotoxicants' with substantial evidence of harm. (Mundy et al. 2009 & 2015)

In 2019, the National Toxicology Program (NTP) published the following hazard assessment:

> "NTP's conclusion that fluoride is ***presumed to be a cognitive neurodevelopmental hazard to humans*** is based on consistent evidence from 26 lower risk-of-bias studies that evaluated fluoride exposure and effects on children's IQ and other cognitive effects.… A few studies also support the possibility of heightened sensitivities to the detrimental cognitive effects of fluoride exposures in individuals…" - *Draft Monograph from National Toxicology Program, "Systemic Review of Fluoride Exposure and Neurodevelopmental and Cognitive Health Effects" (Sept 6, 2019)*

The NTP wrote that given the extent and consistency of the evidence, new studies would not change the hazard determination, which is the highest possible OHAT hazard categorization short of conducting human experimentation that intentionally causes brain damage in infants.

As you would expect, the NTP draft publication prompted considerable political backlash from fluoridationist organizations, including the American Dental Association (ADA) and Oral Health

Division of the CDC who have significant investment in fluoride treatments for children and water fluoridation programs. As a result, the NTP has undertaken a systematic review and a "state of the science" report which we've been expecting to be published for months. The current expectation is the final NTP package will be available by the end of March 2022.

All this is background. The fact of the matter is that there are even more studies published since 2019 that confirm everything to date regarding the toxicity and neurotoxicity of fluoride.

I am attaching an annotated bibliography validating harm to brains, bodies and bones, but would like to specifically point to these items which establish LOAEL:

I. The 2021 Benchmark Dose Analysis used fluoride studies from MIREC and ELEMENT cohort projects as input, the results of which are consistent with other studies finding neurodevelopmental harm. The authors identify 0.2 mg/L as having an adverse impact on neurodevelopment. "The prospective studies offer strong evidence of prenatal neurotoxicity, and the benchmark results should inspire a revision of water-fluoride recommendations aimed at protecting pregnant women and young children."
   - Grandjean P, Hu H, Till C, Green R, Bashash M, Flora D, Tellez-Rojo MM, Song P, Lanphear B, Budtz-Jørgensen E. A Benchmark Dose Analysis for Maternal Pregnancy Urine-Fluoride and IQ in Children. Risk Analysis. 8 June 2021.

II. This excerpt from a 2021 study on bones is succinct, "In this cohort of postmenopausal women, the risk of fractures was increased in association with two separate indicators of fluoride exposure. Our findings are consistent with RCTs and suggest that high consumption of drinking water with a fluoride concentration of ~1 mg/L may increase both BMD (bone mineral density) and skeletal fragility in older women."
   - Helte E, Donat Vargas C, Kippler M, Wolk A, Michaëlsson K, Åkesson A. Fluoride in Drinking Water, Diet, and Urine in Relation to Bone Mineral Density and Fracture Incidence in Postmenopausal Women. Environ Health Perspect. 2021 Apr;129(4):47005.

III. Low thyroid has many adverse effects on overall health and about 18% of people drinking 'optimally' fluoridated water (0.7 ppm) in Canadian communities have a heightened risk of low thyroid function because fluoride interferes with iodine metabolism. Many will be sub-clinical and not know they are mildly hypothyroid, which nevertheless increases their risk for diabetes, high cholesterol and birthing children with cognitive and behavioral problems.
   - Ashley J. Malin, Julia Riddell, Hugh McCague, Christine Till. Fluoride exposure and thyroid function among adults living in Canada: Effect modification by iodine status. Environment International. Volume 121, Part 1, December 2018, Pages 667-674.

The bottom line is that using the SF of ten applied to the LOAEL identified in studies as advised by the EPA in order to protect vulnerable populations with increased susceptibility for harm suggests that a **MCLG for fluoride in drinking water should be zero**, with a pragmatic MCL of no more than 0.3 ppm which accounts for approximately 96% of naturally occurring fluoride in US waters. This would result in two actions: charging polluters with cleaning up fluoride pollution above the MCL and issuing a directive to state health departments advising an immediate cessation of intentional pollution of national waters through artificial fluoridation programs. An additional benefit from these two actions will be reducing a stressor on ecosystems as fluoride is also harmful to flora and fauna.

Fluoridation policy is an issue that has caused perennial confusion in statehouses and city halls as they are the bodies charged with making fluoridation decisions, but they erroneously assume the EPA Office of Water endorses fluoridation programs with warranties of safety for all consumers. So, these legislative bodies defer to willfully blind advocacy/lobby groups and fluoridationist marketing material that repeats 20th century talking points.

Since the SDWA recognizes all drinking water supplies as national waters under its jurisdiction, I suggest this board should step up to offer the scientific assist expected by states and municipalities using water fluoridation chemicals (HFS, FSA, HFSA, $Na_2[SiF_6]$ & NaF). I suggest an evaluation of modern science and recalculation of a fluoride MCLG and MCL that protects susceptible subpopulations should be prioritized by your board for the safety of public health.

Respectfully,

Karen Spencer

att: Annotated bibliography of some of the science published after the Dept of Health and Human Services, parent of CDC, reduced its recommendation of 'optimal' CWF to 0.7 ppm in April 2015

cc: Senator Elizabeth Warren, Senator Ed Markey, Congressman Seth Moulton, Senator Bruce Tarr, Rep. Ann-Margaret Ferrante, Mayor Greg Verga

Fluoridation Policy:
An Annotated Bibliography of Published Science

A sampling of the scientific studies and reports relevant to water fluoridation published since the HHS 2015 recommendation to lower the fluoridation target to 0.7 ppm is listed below.

I suggest these items provide compelling evidence that 0.7 ppm is neither optimal nor safe and that any claims to the contrary are ill-founded. Moreover, protests that more study is required before banning fluoridation is a tacit endorsement of human experimentation without individual consent which is medical assault - *Karen F. Spencer*

2021

BENCHMARK DOSE ANALYSIS: Using fluoride studies from MIREC and ELEMENT projects as input, the results of which are consistent with other studies, authors identify 0.2 mg/L as having an adverse impact on neurodevelopment. “The prospective studies offer strong evidence of prenatal neurotoxicity, and the benchmark results should inspire a revision of water-fluoride recommendations aimed at protecting pregnant women and young children.”
https://pubmed.ncbi.nlm.nih.gov/34101876/

- Grandjean P, Hu H, Till C, Green R, Bashash M, Flora D, Tellez-Rojo MM, Song P, Lanphear B, Budtz-Jørgensen E. A Benchmark Dose Analysis for Maternal Pregnancy Urine-Fluoride and IQ in Children. Risk Analysis. 8 June 2021.

LIFETIME EXPOSURE: Fluoridation is the primary source of fluoride exposure for 1,629 Canadians between 3 and 79 that finds substantially higher lifetime fluoride exposure in fluoridated communities using CHMS data, increasing with age. Vulnerable subpopulations to adverse effects of fluoride noted as the young, those who are iodine deficient, and post-menopausal women. https://www.mdpi.com/1660-4601/18/12/6203/htm

- Julia K. Riddell, Ashley J. Malin, Hugh McCague, David B. Flora, and Christine Till. Urinary Fluoride Levels among Canadians with and without Community Water Fluoridation. Int. J. Environ. Res. Public Health 2021, 18(12), 6203.

KIDNEYS: This study of 1,070 adults found every 1 mg/L increment in the urinary fluoride concentrations was associated with significant increases of 22.8% in the risk of kidney function injury after adjusting for potential confounding factors. Authors conclude that long-term fluoride exposure is associated with compromised kidney function in adults, and that urinary NAG is a sensitive and robust marker of kidney dysfunction caused by fluoride exposure.
https://pubmed.ncbi.nlm.nih.gov/34478979/

- Wu L, Fan C, Zhang Z, Zhang X, et al. Association between fluoride exposure and kidney function in adults: A cross-sectional study based on endemic fluorosis area in China. Ecotoxicol Environ Saf. 2021 Aug 31;225:112735.

BEHAVIORAL CHANGES: Children in Cincinnati Childhood Allergy and Air Pollution Study (CCAAPS) assessed at age 12. Boys in particular did not experience significant anxiety or depression, yet had somatic behaviors based on their childhood urinary fluoride (CUF) concentrations, “seven times more likely to exhibit ‘at-risk’ internalizing symptomology.”
https://pubmed.ncbi.nlm.nih.gov/34755609/

- Adkins EA, Yolton K, Strawn JR, Lippert F, Ryan PH, Brunst KJ. Fluoride exposure during early adolescence and its association with internalizing symptoms. Environ Res. 2021 Oct 29:112296.

Fluoridation Policy:
An Annotated Bibliography of Published Science

CRITICAL WINDOWS: Using urine samples and test scores from 596 mother-child Canadian pairs in the MIREC prospective cohort, researchers found evidence that developmental neurological damage was based on timing of fluoride exposure and gender, “Associations between fluoride exposure and PIQ (performance IQ) differed based on timing of exposure. The prenatal window may be critical for boys, whereas infancy may be a critical window for girls.” https://pubmed.ncbi.nlm.nih.gov/34051202/

- Farmus L, Till C, Green R, Hornung R, Martinez-Mier EA, Ayotte P, Muckle G, Lanphear B, Flora D. Critical Windows of Fluoride Neurotoxicity in Canadian Children. Environ Res. 2021 May 26:111315.

GENES: Several genes make individuals more vulnerable to the neurotoxic impact with gender differences, also affecting mitochondria and suggesting vulnerability to dementia. Chinese study of 952 school children between 7 and 13 using water, urinary, hair and nail fluoride identified multiple neurodevelopmental metabolic pathways that result in adverse effects from low fluoride exposures. https://www.sciencedirect.com/science/article/pii/S0160412021003068

- Yu X, Xia L, Zhang S, et al. Fluoride exposure and children's intelligence: Gene-environment interaction based on SNP-set, gene and pathway analysis, using a case-control design based on a cross-sectional study. Environ Int. 2021 Jun 4;155:106681.

GENETIC VULNERABILITY: Dopamine relative genes affect the susceptibility of individuals to fluoride toxicity even in safe water concentrations which result in lowered IQ so that “low-moderate fluoride exposure is inversely related to children’s IQ.” https://pubmed.ncbi.nlm.nih.gov/33360592/

- Zhao L, Yu C, Lv J, et al. Fluoride exposure, dopamine relative gene polymorphism and intelligence: A cross-sectional study in China. Ecotoxicology and Environmental Safety. 2021 Feb;209:111826.

BRITTLE BONES: “In this cohort of postmenopausal women, the risk of fractures was increased in association with two separate indicators of fluoride exposure. Our findings are consistent with RCTs and suggest that high consumption of drinking water with a fluoride concentration of ~1 mg/L may increase both BMD (bone mineral density) and skeletal fragility in older women.” https://pubmed.ncbi.nlm.nih.gov/33822648/

- Helte E, Donat Vargas C, Kippler M, Wolk A, Michaëlsson K, Åkesson A. Fluoride in Drinking Water, Diet, and Urine in Relation to Bone Mineral Density and Fracture Incidence in Postmenopausal Women. Environ Health Perspect. 2021 Apr;129(4):47005.

OSTEOARTHRITIS: Identifies fluoride as an environmental chemical that has adverse effects on articular cartilage and osteoarthritis (OA) risk. “In full sample analysis, a 1 mg/L increase in UF (urinary fluoride) level was associated with a 27% higher risk of OA.” https://link.springer.com/article/10.1007/s12011-021-02937-2

- Sowanou, A., Meng, X., Zhong, N. et al. Association Between Osteoarthritis and Water Fluoride Among Tongyu Residents, China, 2019: a Case–Control of Population-Based Study. Biol Trace Elem Res (2021).

NO BENEFIT FOR PRESCHOOLERS: Polish study finds ‘optimal’ fluoride concentrations in water provide no dental benefit. Dental caries experience depended on oral hygiene and diet. https://www.sciencedirect.com/science/article/abs/pii/S0946672X2100016X

- Opydo-Szymaczek J, et al. Fluoride exposure and factors affecting dental caries in preschool children living in two areas with different natural levels of fluorides. Journal of Trace Elements in Medicine and Biology. Volume 65. 2021.

ALTERNATIVE: This systematic review and meta-analysis concludes that biomimetic hydroxyapatite-containing, fluoride-free oral care products are effective in reducing dental decay, especially in children without the risk of dental fluorosis and neurotoxicity inherent in topical use of fluoridated products. https://files.cdha.ca/profession/journal/2752.pdf

- Hardy Limeback, BSc, PhD, DDS; Joachim Enax, Dr; Frederic Meyer, Dr. Biomimetic hydroxyapatite and caries prevention: a systematic review and meta-analysis. | Can J Dent Hyg 2021;55(3): 148-159.

AMERICAN KIDNEYS: Using U.S. NHANES data from two recent cycles, finds 'optimal' amounts of fluoridated water results in high incidence of uric acid in adolescents suggesting higher risk of kidney disease and other illnesses. Identifies dose-response trend in plasma fluoride of teens.
https://www.sciencedirect.com/science/article/pii/S0147651320315074

- Yudan Wei, Jianmin Zhu, Sara Ann Wetzstein. Plasma and water fluoride levels and hyperuricemia among adolescents: A cross-sectional study of a nationally representative sample of the United States for 2013–2016. Ecotoxicology and Environmental Safety. Volume 208. 15 January 2021.

TODDLERS: The Programming Research in Obesity, Growth, Environment and Social Stressors (PROGRESS) cohort included 948 mother-child pairs from Mexico City. Blinded testing of children between one and 24 months to examine associations between maternal fluoride intake from food and beverages during pregnancy and offspring neurodevelopment in this prospective and longitudinal study found, "higher exposure to fluoride from food and beverage consumption in pregnancy was associated with reduced cognitive outcome, but not with language and motor outcome in male offspring over the first two years of life."
https://fluoridealert.org/wp-content/uploads/cantoral-2021.final_.pdf

- Alejandra Cantoral, Martha M. Tellez-Rojo, Ashley J. Malin, Lourdes Schnaas d, ErikaOsorio-Valencia, Adriana Mercadob, E. Angeles Martínez-Mier, Robert O. Wright, Christine Till. Dietary fluoride intake during pregnancy and neurodevelopment in toddlers: A prospective study in the progress cohort. Neurotoxicology 87 (2021) 86–93.

NO SAFE DOSE: Study of Mexican children and their mothers using measurements of urinary fluoride and water concentrations associated dental fluorosis and lowered IQ with fluoride dose consistent with findings of larger studies in other countries. Authors declare WHO fluoride guidelines are unsafe and hypothesize that 0.045 F^- mg/day is a protective exposure
https://www.mdpi.com/1660-4601/18/21/11490/htm

- Farías P, Estevez-García JA, Onofre-Pardo EN, Pérez-Humara ML, Rojas-Lima E, Álamo-Hernández U, Rocha-Amador DO. Fluoride Exposure through Different Drinking Water Sources in a Contaminated Basin in Guanajuato, Mexico: A Deterministic Human Health Risk Assessment. International Journal of Environmental Research and Public Health. 2021; 18(21):11490.

BABY BRAIN POISON: Exposure to fluoridated water (10 mg/L & 50 mg/L) beginning on the first day of pregnancy and continuing through the last day of breastfeeding shows chemical imbalances, cellular damage and changes in the hippocampus of Wistar rat offspring that would affect neurological development.
https://pubmed.ncbi.nlm.nih.gov/33096359/

- Ferreira MKM, Aragão WAB, Bittencourt LO, Puty B, Dionizio A, Souza MPC, Buzalaf MAR, de Oliveira EH, Crespo-Lopez ME, Lima RR. Fluoride exposure during pregnancy and lactation triggers oxidative stress and molecular changes in hippocampus of offspring rats. Ecotoxicology and Environmental Safety. 2021 Jan 15;208:111437.

Fluoridation Policy:
An Annotated Bibliography of Published Science

BAD TEETH - BAD BRAIN: Chinese study confirm 1.6 ppm v. 0.1 ppm results in children with both damaged teeth and lower IQ. Authors validate that fluoride affects thyroid function, neurotransmitters and mitochondrial energy enzymes. There were no students with low IQ found in the area with low F level. There was high IQ among the 96.6% of the students who did not experience fluorosis.
https://www.sciencedirect.com/science/article/pii/S0213911121001965

- Yani SI, Seweng A, Mallongi A, et al. The influence of fluoride in drinking water on the incidence of fluorosis and intelligence of elementary school students in Palu City. Gac Sanit. 2021;35 Suppl 2:S159-S163.

GUTS & BRAINS: Memory function was reduced and gut microbiota structure was significantly altered in fluoride-exposed mice.
https://www.sciencedirect.com/science/article/pii/S0147651321002190

- Xin J, Wang H, Sun N, Bughio S, Zeng D, Li L, Wang Y, Khalique A, Zeng Y, Pan K, Jing B, Ma H, Bai Y, Ni X. Probiotic alleviate fluoride-induced memory impairment by reconstructing gut microbiota in mice. Ecotoxicol Environ Saf. 2021 Jun 1;215:112108

INFLAMED GUTS: Exposure to fluoridated water at both doses (10 mg/L & 50 mg/L) inflame guts in rats and alters the gut microbiome as compared to control (0 mg/L).
https://pubmed.ncbi.nlm.nih.gov/33508686/

- Dionizio A, Uyghurturk DA, Melo CGS, Sabino-Arias IT, Araujo TT, Ventura TMS, Perles JVCM, Zanoni JN, Den Besten P, Buzalaf MAR. Intestinal changes associated with fluoride exposure in rats: Integrative morphological, proteomic and microbiome analyses. Chemosphere. 2021 Jan 11;273:129607.

HARMFUL ADEQUATE INTAKE (AI): Study found "the levels of dietary F- intake were below the current AI, were greater towards the end of gestation and in women who were moderately and highly compliant with Mexican dietary recommendation" in ELEMENT cohort and recommended changing future dietary recommendations due to evidence of developmental neurotoxicity at even low dose exposure. https://pubmed.ncbi.nlm.nih.gov/33602354/

- Castiblanco-Rubio, G., Muñoz-Rocha, T., Cantoral, A., Téllez-Rojo, M., Ettinger, A., Mercado-García, A., Peterson, K.E., Hu, H., Martínez-Mier, E. (2021). Dietary Fluoride Intake Over the Course of Pregnancy in Mexican Women. Public Health Nutrition, 1-25.

CALCIUM & FLUORIDE IN PREGNANCY: Calcium intake during pregnancy lowers urinary fluoride (UF) concentrations by some unknown mechanism in ELEMENT cohort.
https://pubmed.ncbi.nlm.nih.gov/34176079/

- Castiblanco-Rubio GA, Muñoz-Rocha TV, Téllez-Rojo MM, Ettinger AS, Mercado-García A, Peterson KE, Hu H, Cantoral A, Martínez-Mier EA. Dietary Influences on Urinary Fluoride over the Course of Pregnancy and at One-Year Postpartum. Biol Trace Elem Res. 2021 Jun 26.

SAFETY: Evidence of dental fluorosis and other adverse effects to bodies and brains from supposed safe concentrations is alarming. “The safety of public health approach of drinking water fluoridation for global dental caries reduction are urgently needed further research.”
https://www.sciencedirect.com/science/article/pii/S0147651321005510?via%3Dihub

- Dong H, Yang X, Zhang S, Wang X, Guo C, Zhang X, Ma J, Niu P, Chen T. Associations of low level of fluoride exposure with dental fluorosis among U.S. children and adolescents, NHANES 2015-2016. Ecotoxicol Environ Saf. 2021 Jun 22;221:112439.

SKELETAL FLUOROSIS: This Chinese study of the pathogenetic progression of skeletal fluorosis, details how local signaling pathways, hormones, promoter DNA hypermethylation, RNA expression etc. are affected by fluoride exposure leading to pain and disability. https://www.mdpi.com/1422-0067/22/21/11932/htm

- Qiao L, Liu X, He Y, Zhang J, Huang H, Bian W, Chilufya MM, Zhao Y, Han J. Progress of Signaling Pathways, Stress Pathways and Epigenetics in the Pathogenesis of Skeletal Fluorosis. International Journal of Molecular Sciences. 2021; 22(21):11932.

DEPRESSION: Animal study finds negative changes in brain structure and behavior with exposure to sodium fluoride (NAF). https://pubmed.ncbi.nlm.nih.gov/34735150/

- Zhou G, Hu Y, Wang A, Guo M, Du Y, Gong Y, Ding L, Feng Z, Hou X, Xu K, Yu F, Li Z, Ba Y. Fluoride Stimulates Anxiety- and Depression-like Behaviors Associated with SIK2-CRTC1 Signaling Dysfunction. J Agric Food Chem. 2021 Nov 4. PMID: 34735150.

DECEPTION: This historical analysis documents how the ADA suppressed the established science that vitamin D was necessary for healthy teeth and bones in order to promote falsely fluoride which was and is more profitable for their membership. “Public health may well depend on looking at professional societies no different than the way we look at the pharmaceutical industry—conflicted organizations with a power to shape conventional wisdom based on fragile evidence.” https://www.mdpi.com/2072-6643/13/12/4361/htm#

- Hujoel PE. How a Nutritional Deficiency Became Treated with Fluoride. Nutrients. 2021.

2020

AMERICAN FETAL EXPOSURE: Study on pregnant women in California and Montana find, “Fluoride concentrations in urine, serum, and amniotic fluid from women were positively correlated to public records of community water fluoridation” and that concentration is consistent with findings of Canadian studies that find these concentrations are associated with increased learning disabilities and lower IQ in offspring.
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7132865/

- Abduweli Uyghurturk D, Goin DE, Martinez-Mier EA, Woodruff TJ, DenBesten PK. Maternal and fetal exposures to fluoride during mid-gestation among pregnant women in northern California. Environ Health. 2020 Apr 6;19(1):38.

BLOOD: Canadian Health Measures Survey (CHMS) collects extensive biomonitoring data used to assess the exposure of Canadians to environmental chemicals finds higher fluoride in urine associated with significantly higher blood lead, urinary lead, etc. Also finds urinary selenium is significantly lower in fluoridated Canadian communities, “this is the first study where biomonitoring data from multiple cycles of CHMS were combined in order to generate robust estimates for subsets of the Canadian population. Such assessments can contribute to a regional-level prioritization of control measures to reduce the exposure of Canadians to chemicals in their environment.”
https://www.ncbi.nlm.nih.gov/pubmed/31972364?dopt=Abstract

- Valcke M, Karthikeyan S, Walker M, Gagné M, Copes R, St-Amand A. Regional variations in human chemical exposures in Canada: A case study using biomonitoring data from the Canadian Health Measures Survey for the provinces of Quebec and Ontario. Int J Hyg Environ Health. 2020 Jan 20;225:113451.

THYROID & IQ: Concentrations of fluoride in drinking water considered optimal and safe in the US result in altered thyroid function and lowered IQ in Chinese children.
https://www.sciencedirect.com/science/article/pii/S0160412019301370

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PINEAL GLAND & MELATONIN: Fluoride calcifies the pineal gland and interferes with enzyme function, hormones and sleep patterns. https://www.mdpi.com/2076-3417/10/8/2885

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http://fluoridealert.org/wp-content/uploads/neurath.2019-1.pdf

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ALZHEIMER'S: Even low concentrations of fluoride in drinking water at or below concentrations deemed optimal or safe by the WHO result in a pattern of increased dementia.
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DEMENTIA: Describes mechanism by which the effectiveness of the two most popular drugs used to treat Alzheimer's & other neurodegenerative dementia disease is reduced or blocked by fluoride. https://www.mdpi.com/1660-4601/16/1/10/htm

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DELAYED MALE PUBERTY: This 4th study from the NIH sponsored ELEMENT investigation of the prenatal impact of low-dose prenatal exposure found a significant pattern of delayed puberty for boys associated with maternal fluoride as measured in urine samples. Female data showed non-significant trend towards earlier menarche. More study needed to determine the impact on sexual development. https://www.ncbi.nlm.nih.gov/pubmed/30922319

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IODINE: Identifies and discusses the biochemical and hormonal impact of fluoride and fluoridation policy on iodine metabolism with consideration of related neurodevelopmental and pathological disorders. https://www.mdpi.com/1660-4601/16/6/1086

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MITOCHONDRIA: Prenatal and postnatal exposure to fluoride results in mitochondrial abnormalities, autophagy and apotheosis contributing to neuronal death.
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NUTRITION: The f-ion is a poison but the bioavailability of CaF is different than NaF as calcium is the antidote to fluoride poisoning. In addition to being in water and dental products, 20% of pharma and 40% of agrichemicals have a fluoride base. Consequently, people are exposed to excessive amounts of fluoride which contributes to chronic disease.
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THYROID: 18% of people drinking 'optimally' fluoridated water in Canadian communities have a heightened risk of low thyroid function because fluoride interferes with iodine metabolism. Many of them will be sub-clinical and not know they are mildly hypothyroid, which nevertheless increases their risk for diabetes, high cholesterol, and other problems. Study excluded those already diagnosed with thyroid disease. (CHMS)
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- Ashley J. Malin, Julia Riddell, Hugh McCague, Christine Till. Fluoride exposure and thyroid function among adults living in Canada: Effect modification by iodine status. Environment International. Volume 121, Part 1, December 2018, Pages 667-674.

THYROID: Even 0.5 ppm fluoride in water has an adverse impact on thyroid hormones. Water is currently fluoridated to 0.7 ppm, a reduction from up to 1.2 ppm in 2015.
https://www.NCBI.nlm.nih.gov/pmc/articles/PMC5805681/

- Z. Kheradpisheh et al. (2018) Impact of Drinking Water Fluoride on Human Thyroid Hormones: A Case-Control Study. Scientific Reports. volume 8.

OVERDOSED BABIES: Over one third of babies (37%) in fluoridated American communities consume amounts of fluoride in excess of the upper limits of fluoride considered safe per government regulations. Even 4% of babies in non-fluoridated communities are overdosed on fluoride due to consumption of products made with fluoridated water. At the very least, this puts these children at high risk for developing dental fluorosis. Dental fluorosis is associated with increased incidence of learning disabilities, broken bones and kidney disease.
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- Claudia X Harriehausen, Fehmida Z Dosani, Brett T Chiquet, Michelle S Barratt, and Ryan L Quock. Fluoride Intake of Infants from Formula. Journal of Clinical Pediatric Dentistry. 2018.

GOVERNMENT BIAS: A National Toxicology Program animal experiment studying the impact of fluoride consumption used the wrong rats, the wrong dose, and the wrong study design in order to manufacture a finding of no prenatal or postnatal effect.
https://www.sciencedirect.com/science/article/pii/S0306987718308600

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PREGNANT WOMEN: Pregnant Canadian women drinking 'optimally' fluoridated water had twice the fluoride exposure per individual testing as compared to pregnant women in non-fluoridated Canadian communities - and consistent with the range in the Mexican women in the ELEMENT cohort whose children had up to 6 points lowered IQ based on prenatal exposure to fluoride (from salt). The Canadian study excluded those with health conditions such as kidney disease as well as considered confounding factors such as tea consumption.
https://ehp.niehs.nih.gov/doi/pdf/10.1289/EHP3546

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LEARNING DISABILITIES: Over 200 children were individually tested. Study found attention deficit disorder apparently caused by their prenatal exposure to fluoride specific to dose. This is the 3rd report out of the NIH sponsored 12 year ELEMENT project that has confirmed low dose prenatal exposure to fluoride consistent with exposure in 'optimally' fluoridated communities causes subtle but permanent brain damage for many consumers. Excluded those with history of mental illness or conditions such as diabetes and renal disease.
https://www.sciencedirect.com/science/article/pii/S0160412018311814

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ALZHEIMER'S DISEASE: Describes impact of fluoride-induced stress and inflammation in the development of Alzheimer's disease and demonstrates the mechanism for cell death in its worsening over time. https://www.mdpi.com/1422-0067/19/12/3965

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CANCER: Researchers who include an IARC scientist find esophageal cancer is 9.4 times more prevalent among those with dental fluorosis in the endemic fluorosis regions of Kenya. Provides biological plausibility that inflammatory fluoride affects microbiome and other biological mechanisms. Recommends more study. https://www.ncbi.nlm.nih.gov/pubmed/30582155/

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KIDNEYS: Fluoride is a common exposure that is selectively toxic to the kidneys.
https://www.sciencedirect.com/science/article/pii/S0270929518301827

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IQ & DF: Between 0.5 and 3.9 mg/L, found every 0.1 mg/L increased dental fluorosis by 2.24% and every 0.5 mg/L decreases IQ by 2.67 points. Also found half as many kids with high IQ children with higher F- dose. https://www.NCBI.nlm.nih.gov/pubmed/29870912

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2017

REVIEW: Concludes that fluoridation schemes whether from water, food or salt programs "pose risks of various diseases in the asthmatic-skeletal, neurological, endocrine and skin systems. Dental and skeletal fluorosis are signs of chronic and excessive ingestion of fluoride."
https://www.NCBI.nlm.nih.gov/pubmed/28453591

- Verena Romero, Frances J. Norris, Juvenal A. Ríos, Isel Cortés, Andrea González, Leonardo Gaete, Andrei N. Tchernitchin. The impact of tap water fluoridation on human health. Rev. méd. Chile vol.145 no.2 Santiago Feb. 2017.

DOSE-RESPONSE: Validated that IQs of children are lowered on a dose-response trend line correlated with the amount of fluoride exposure as measured via urine tests of their mothers during pregnancy and individualized IQ tests of offspring. In the range consistent with doses in optimally fluoridated communities, there was up to a 6 point difference in IQ. This NIH sponsored 12 year longitudinal study conducted by researchers at world class American & Canadian universities excluded diabetics as well as those with kidney disease or pregnancy complications and allowed for many confounders.
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GENES & BONES: "This study provides evidence that chronic oxidative and inflammatory stress may be associated with the fluoride-induced impediment in osteoblast differentiation and bone development." http://link.springer.com/article/10.1007/s12011-016-0756-6

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PRESCHOOL DIET: Diet of two year olds contain unsafe levels of fluoride.
http://onlinelibrary.wiley.com/doi/10.1111/cdoe.12283/full

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APOPTOSIS: "Enamel fluorosis is a developmental disturbance caused by intake of supraoptimal levels of fluoride during early childhood.The enamel defects consist of horizontal thin white lines, opacities (subsurface porosities), discolorations, and pits of various sizes. The molecular mechanism underlying enamel fluorosis is still unknown.…. We can hypothesize that fluorosis is due to a combination of direct cytotoxic effects causing cell death, the delayed development of tight junctions, which are necessary to form a sealed barrier between apical and basolateral surfaces, and a direct inhibitory effect of fluoride on vectorial calcium and/or bicarbonate transport." https://www.NCBI.nlm.nih.gov/pmc/articles/PMC5770627/

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DNA: Finds that "prolonged fluoride intake at chosen concentrations caused imbalance of the cellular oxidative state, affected DNA and disrupted cellular homeostasis… It is recommended that fluoride supplementation requires a fresh consideration in light of the current study."
https://www.NCBI.nlm.nih.gov/pubmed/28089781

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PRENATAL POISON: "F can pass through the cord blood and breast milk and may have deleterious impact on learning and memory of the mouse pups."
http://journals.sagepub.com/doi/abs/10.1177/0960327117693067

- Y Zhang, X Xue, R Niu, J Wang. Maternal fluoride exposure during gestation and lactation decreased learning and memory ability, and glutamate receptor mRNA expressions of mouse pups. Z Sun, Human & Experimental Toxicology. February 13, 2017.

IMMUNITY: Prenatal and early postnatal exposure to fluoride impairs spleen function and development which damages spleen and lifelong immunity.
https://www.NCBI.nlm.nih.gov/pubmed/28846973/

- Yanqin Ma, Kankan Zhang, Fengjun Ren, Jundong Wang, Developmental fluoride exposure influenced rat's splenic development and cell cycle via disruption of the ERK signal pathway, In Chemosphere, Volume 187, 2017, Pages 173-180

NEUROINFLAMMATION: Toxic effects of fluoride on the central nervous system and immunity.
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2016

CRITIQUE HHS RECOMMENDATION: Pro-fluoridation team of dental researchers determined that the Department of Health and Human Services reduction of the optimal fluoride concentration to a single 0.7 ppm target is lacking in sound science, i.e. that "policy need to be cognizant of the balancing of risk and protective exposures across the entire population and potentially all ages and to be based on recent data that are purposefully collected, critically analyzed and carefully interpreted… (the recommendation seems) premature in terms of its rationale and its use and interpretation of sometimes dated data." These authors' bias is to maintain 1 ppm; nevertheless, their rationale against the HHS document is appropriate. The HHS document is political, not scientific.
https://www.NCBI.nlm.nih.gov/pubmed/26710669

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OSTEOPOROSIS: "Consequently, although the World Health Organization continues to support F schemes for caries prevention despite a lack of scientific proof, the F schemes are not able to improve the crystal quality but rather contribute adversely to affect tooth development and increases the risk of developing postmenopausal osteoporosis."
http://dx.doi.org/10.4172/2379-1764.1000170

- Mitsuo Kakei, Masayoshi Yoshikawa and Hiroyuki Mishima. Fluoride Exposure May Accelerate the Osteoporotic Change in Postmenopausal Women: Animal Model of Fluoride-induced Osteoporosis. Adv Tech Biol Med 2016, 4:1

DIABETES: Fluoridation policy significantly increases incidence of age related type 2 diabetes. https://www.NCBI.nlm.nih.gov/pubmed/27740551

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Fluoridation policy poses a hazard to an unsuspecting public

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DEFINITIONS:

- **Endorsement**: An endorsement is an authoritative statement reflecting a point of view for the purpose of exerting influence. An endorsement is ***not*** an expert opinion.
  - **Authoritative statement:** An opinion that interprets a rule, law or policy for the purpose of guiding, influencing, or mandating action. Authoritative statements are not inherently trustworthy or reliable, but they are inherently manipulative. "Testimonial propaganda" utilizes authoritative statements in marketing and in politics. The slogan "question authority" was intended to encourage critical thinking in order to combat the blind acceptance of biased authoritative statements that endorse policy and/or sanctioned narratives. (*Logical Fallacies: Appeal to Authority*)
- **Expert Opinion**: An expert opinion is dependent on evidence and the due diligence of someone with substantial study in a field. The Daubert Standard is a legal process that validates the trustworthiness of experts offering opinion in a court of law.

EXAMPLES:

ENDORSEMENT: The April 2015 HHS statement recommending 0.7 ppm fluoride concentration in drinking water for 'safe & effective' prevention of tooth decay promoted the long standing fluoridation policy of the agency.

vs.

EXPERT OPINION: The June 2015 Cochrane report finds no reliable evidence of dental benefit to adults or low income children, but documents substantially higher rates of dental fluorosis, some of which will likely result in costly cosmetic dentistry.
The 2019 National Toxicology Program systematic review offered an expert opinion based on the evidence that fluoride is a presumed hazard to human health specific to neurotoxic impact when exposure is pre- or post-natal.

☰
Bulletin
of the
Atomic
Scientists

Trinity: "The most significant hazard of the entire Manhattan Project"

By Kathleen M. Tucker, Robert Alvarez, July 15, 2019

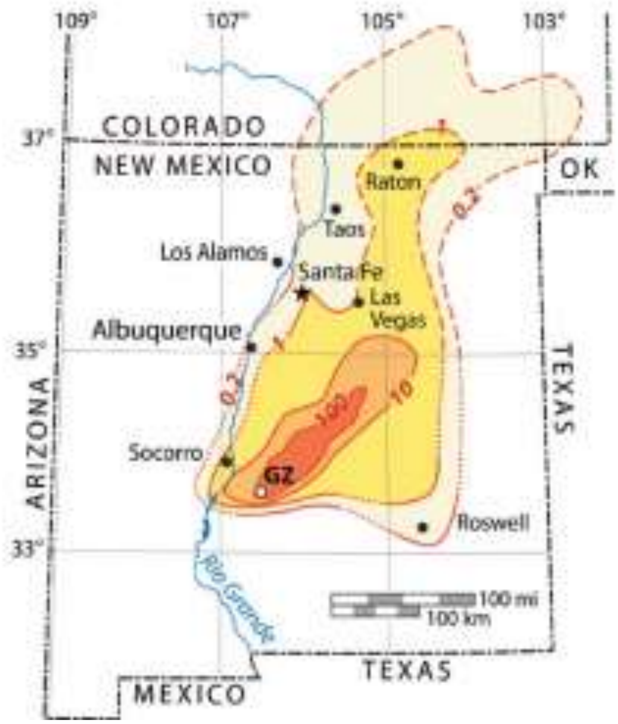
For the past several years, the controversy over radioactive fallout from the world's first atomic bomb explosion in Alamogordo, New Mexico on July 16, 1945—code-named Trinity—has intensified. Evidence collected by the New Mexico health department but ignored for some 70 years shows an unusually high rate of infant mortality in New Mexico counties downwind from the explosion and raises a serious question whether or not the first victims of the first atomic explosion might have been American children. Even though the first scientifically credible warnings about the hazards of radioactive fallout from a nuclear explosion had been made by 1940, historical records indicate a fallout team was not established until less than a month before the Trinity test, a hasty effort motivated primarily by concern over legal liability.

In October 1947, a local health care provider raised an alarm about infant deaths downwind of the Trinity test, bringing it to the attention of radiation safety experts working for the US nuclear weapons program. Their response misrepresented New Mexico's then-unpublished data on health effects. Federal and New Mexico data indicate that between 1940 and 1960, infant death rates in the area downwind of the test site steadily declined—except for 1945, when the rate sharply increased, especially in the three months following the Trinity blast. The 21 kiloton explosion occurred on a tower 100 feet from the ground and has been likened to a "dirty bomb" that cast large amounts of heavily contaminated soil and debris—containing 80 percent of the bomb's plutonium—over thousands of square-miles. (See Figure 1.)

After a nearly half a century of denial, the US Department of Energy concluded in 2006, "the Trinity test also posed the most significant hazard of the entire Manhattan Project."[1] Four years later the US Centers for Disease Control gave weight to this assessment by concluding:

"New Mexico residents were neither warned before the 1945 Trinity blast, informed of health hazards afterward, nor evacuated before, during, or after the test. Exposure rates in public areas from the world's first nuclear explosion were measured at levels 10,000-times higher than currently allowed."[2]

Figure 1.



Estimated exposure rate in milliroentgens per hour (mR h^{-1}) 12 hours after detonation; GZ = ground zero of Trinity. Source: Centers for Disease Control (2010).

Meanwhile the National Cancer Institute is conducting a study to model the dispersion and dose reconstruction for people who may have been exposed to fallout from the Trinity explosion. Regardless of the outcome of this study, it is clear the public was put in harm's way because of US government negligence in conducting and its participation in a coverup of the results of an exceedingly dangerous experiment.

Infant mortality concerns raised about Trinity. In October 1947, the first concerns over a rise in infant mortality along the fallout path of the Trinity explosion were raised in a letter to Stafford Warren, a medical radiologist and radiation safety chief of the Manhattan Project and the Trinity test in particular. "As I recall, in August 1945, the month after the first bomb was tested in New Mexico, there were about 35 infant deaths here…" Kathryn S. Behnke, a health care provider from Roswell, New Mexico, wrote. "I understand the rate at Alamogordo, nearer the site of the test, was even higher than Roswell."[3]

On December 4, 1947, Warren's medical assistant, Fred A. Bryan, replied to Ms. Behnke, writing that "we can find no pertinent data concerning infant deaths; in fact there is no report as to the number of or specific cause or dates and, as far as Alamogordo is concerned."[4] Bryan also wrote that he "wanted to assure you that the safety and health of the people at large is not in any way endangered."[5]

Bryan failed to mention that he did not bother to examine New Mexico’s vital statistics. About a month after Bryan’s reassured Behnke of no evidence of harm, a state health official sent the actual unpublished data on infant deaths collected by the state to Los Alamos. [6] Soon thereafter, in a letter dated, January 22, 1948 to Bryan, Wright Langham, biomedical group leader at the Los Alamos National Laboratory (LANL), forwarded hand-written sheets from the state of “the records of infant births and deaths during 1945-1947.” Langham added: “I am sure what I am sending you will not be of much help.” The New Mexico Health Department data indicated that the infant death rate increased by 38 percent in 1945 compared to 1946 and was 57 percent higher than in 1947.[7]

Finding the facts. More than 70 years later, we examined the vital statistics collected by the US government and the state of New Mexico in the 1940s to determine if area health patterns changed after the first atomic explosion. The data eventually provided to Los Alamos and Bryan in January 1948 indicated a sharp rise in infant deaths following the Trinity explosion. Later, between 1940 and 1960, infant mortality in New Mexico showed steady and deep annual declines—except for 1945, when it shot up.[8] The infant mortality rate in New Mexico in 1945 was 100.8 per 1,000 live births; the rate for 1944 was 89.1, and for 1946 it was 78.2.[9] (See Figure 2.) The unpublished data sent to Los Alamos indicated an infant death rate nearly 34 percent higher in 1945 than subsequently made public.

Figure 2

Month-by-month data for the years 1943 to 1948 revealed the highest infant mortality rates in late summer, following the Trinity blast, with a significant peak in September 1945. Infant mortality for the months August, September, and October after the explosion indicated that New Mexican infants had a 56 percent increased risk of dying, with less than a 0.0001 percent chance that this was due to natural fluctuation.[10]

In 1945, infant death rates increased on average by 21 percent (with a statistical error range of plus or minus six percent that applies to all the rates listed in this paragraph) in counties where fallout was measured by Manhattan Project personnel. Rates in these counties dropped by an average of 31 percent in 1946. The infant death rate in Roswell, where Ms. Behnke first alerted Warren of the problem, climbed by 52 percent in 1945, after falling by 27 percent between 1943 and 1944. The rate then dropped in Roswell by 56 percent in 1946. Rates in the downwind counties where fallout was measured dropped by an average of 31 percent (plus or minus eight percent) percent in 1946

We found no extraordinary metrological conditions, such as heat or heavy rains and floods, that may have competed with radioactive fallout as a factor in the increase in newborn deaths after Trinity. According to the CDC in 2010, risks to newborns were especially heightened as “residents reported that fallout ‘snowed down’ for days after the blast, most had dairy cows and most collected rain water off their roofs for drinking.”[11]

The Trinity Test was conducted on July 16, 1945. The rate of infant mortality began rising in July. The month of August showed an infant mortality rate of 152.3 per 1,000 live births. In September, the rate was 187.8, and in October 123.1. Infant mortality change rates for August, September, and October show a dramatic increase in 1945 when compared to the same three months for the years 1943, 1944, 1946, 1947 and 1948 (see figure 3)

Figure 3

Ionizing radiation is especially damaging to dividing cells, so the developing infant, both before and after birth, is susceptible to radiation damage, as Alice Stewart, an epidemiologist who first demonstrated the link between X-rays of pregnant women and disease in their children,[12] first warned in 1956.[13]This damage may be seen years later with the development of leukemia and other cancers in children exposed in utero to ionizing radiation, as Stewart and others confirmed in subsequent studies.[14] By 1958, the United Nations Scientific Committee on the Effects of Atomic Radiation recognized that, in the short term, radiation damage can be reflected in fetal and infant deaths.[15]

Fallout protection was not a priority for the Trinity explosion. The Trinity test was top secret to all but a few scientists and military officials. No warnings were issued to citizens about off- site fallout dangers, although off-site measurements done with a paucity of instruments and people indicated that radiation spread well beyond the test site boundaries. [16]

The Trinity bomb was detonated atop a 100-foot steel tower. With an estimated explosive yield of 21,000 tons of TNT, the fireball vaporized the tower and shot hundreds of tons of irradiated soil to a height of 50,000 to 70,000 feet, spreading radioactive fallout over a very large area. Fallout measurements taken shortly after the explosion were very limited and primitive instruments were used; the data suggest no measurements regarding inhalation or ingestion of radionuclides were taken.

Joseph Shonka, a principal researcher for the study of the Trinity shot for the Centers for Disease Control, recently concluded that the Trinity fallout “was similar to what might occur with a dirty bomb. A fraction of the plutonium [~20%] was used in the explosion [and] … the fireball contacted the soil. Because of the low altitude, fallout exhibited a ‘skip distance’ with little fallout near the test site. Although there were plans for evacuation, radio communication was lost as the survey teams traveled out to follow the overhead plume. Thus, the command center was unsure of whether that the criteria had been met … and failed to order the evacuation.”[17]

Scientists had stressed the importance of protection from radioactive fallout following a nuclear weapon explosion, five years before the Trinity test. “Owing to the spread of radioactive substances with the wind, the bomb could probably not be used without killing large numbers of civilians, and this may make it unsuitable as a weapon for use by this country,” warned Manhattan Project physicists Otto Frisch and Rudolf Peierls in their important memorandum of March 1940, which accelerated production of the first atomic weapons. “[I]t would be very important to have an organization which determines the exact extent of the danger area, by means of ionization measurements, so that people can be warned from entering it.”[18]

As preparations were being made to test the first nuclear weapon, warnings by Frisch and Peierls about fallout hazards were lost on the leadership of the Manhattan Project. Were it not for two physicists at Los Alamos who warned in a June 1945 memorandum that “radiation effects might cause considerable damage in addition to the blast damage ordinarily considered,”[19] little would have been done. Later Joseph O. Hirschfelder, one of the concerned scientists, recalled that “very few people believed us when we predicted radioactive fallout from the atom bomb. On the other hand, they did not ignore this possibility.”[20]

On first being warned by Los Alamos scientists, Gen. Leslie Groves, the Manhattan Project director, dismissed concerns about fallout as being alarmist. But Warren convinced Groves of the potential risk of legal liabilities, and Groves grudgingly agreed to assemble a team at the last minute to track fallout from the test.[21]

A lot was at stake. First, there was the enormous expense involved; the Trinity device cost approximately 15 percent of what the United States spent on all conventional bombs and other explosives during World War II.[22] Then again, there was great pressure to test the Trinity device before July 17, 1945, when the three heads of government of the United States, the Soviet Union and Great Britain were to meet in Potsdam, a German suburb of Berlin, to address the end-stage of World War II and post-war policies. Compared to the political imperative of Potsdam, the hazards of radioactive fallout took a back seat.

But five days after the explosion, Warren reported to Groves that “a very serious hazard” existed over a 2,700 square mile area downwind from the test that had received high radiation doses.[23] Tissue-destructive effects from fallout were observed in livestock in areas that were incorrectly assumed to be uninhabited by people.[24] After realizing the magnitude of the problem, Warren advised Groves that the fallout danger zone, originally set at a 15-mile radius, was too small by at least an order of magnitude and that “there is still a tremendous quantity of radioactive dust floating in the air.”[25]

After more than 70 years, radiation exposures from inhalation and ingestion of water and food contaminated by Trinity test fallout were never assessed,[26]and it may prove to be difficult, if not impossible, to reconstruct doses from internal exposures, given the deaths of residents living in the vicinities from the passage of time and the major changes in lifestyles and dietary habits that have occurred since 1945. Fallout maps of the Trinity test have been made, but they contain strong elements of speculation because of the paucity of radiological monitoring at the time.

The National Cancer Institute is near completion of a fallout dispersion study of the Trinity explosion. Regardless of the outcome of this study, it is clear the public was endangered because of US government negligence in conducting a highly dangerous experiment, as was the case for the downwinders living near the Nevada Test Site, where above-ground nuclear tests were conducted. Because of passage of the Radiation Exposure Compensation Act in 1990, 22,220 “downwinders” exposed to fallout from open air nuclear weapons tests near the Nevada Test Site received an official apology from the US Government for sending them in harm’s way through deception. Through 2015, they had also received nearly $2 billion in financial compensation.[27]

But the people downwind of the 1945 explosion in New Mexico have been denied official recognition, even though the Trinity shot was considered one of the dirtiest of American nuclear tests, with a significant absence of safeguards to protect people from dense radioactive fallout. Safety took a back seat to making sure the first atomic bombs would meet their enormously destructive potential. Alvin Weinberg, director of Oak Ridge National Laboratory during and after the Manhattan Project captured the prevalent mindset in his memoir by saying that "all else, including safety, was secondary."[28]

Several years ago, residents of central and southern New Mexico organized to fight for compensation. Known as the Tularosa Basin Downwinders, they have made a compelling case that cancers and other diseases are due to the Trinity blast and subsequent radioactive fallout from open air atomic bomb tests in Nevada.

Indeed, coming to terms with the legacy of the Trinity explosion through radiation dose reconstruction is further complicated by the fallout that drifted from the Nevada tests into New Mexico. As indicated by the Centers for Disease Control in 2005, northern and central New Mexico were among the areas where significant amounts of fallout were deposited from the Nevada open air atomic tests.[29] Even so, the strong correlation of increased infant deaths in the months following the Trinity explosion cannot be ignored.

We should remember that compensation for people near the Nevada test site was not exclusively based on abstract modeling of radiation doses. Rather, downwinders were also compensated because the burden of proof fell unfairly on them. They were victims not just of willful negligence, but also the government's purposeful deception and suppression of evidence about the high-hazard activity that the US nuclear weapons program constituted. The current body of historical evidence of harm, negligence, and deception—especially the evidence of increased infant death following the first nuclear explosion—should be more than enough for long overdue justice for the people in New Mexico who were downwind of Trinity.

Endnotes

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[7] Letter from Wright H. Langham,

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Keywords: Trinity test
Topics: Analysis, Nuclear Risk, Nuclear Weapons

Tularosa Basin Downwinders Consortium (TBDC) is a grassroots organization that was founded in 2005. Our purpose is to bring attention to the negative health effects suffered by the people living adjacent to the Trinity test site subsequent to their overexposure to high levels of ionizing radiation that occurred on July 16, 1945, with the explosion of the Trinity atomic bomb test. For more information about our organization, please go to: www.trinitydownwinders.com or we would be glad to meet with your organization for a full presentation of our work.

- **DID YOU KNOW**

- **There were families living as close as 12 miles to the Trinity test site in 1945 and there were thousands of families, men, women and children living in a 50 mile radius. If you extend the radius to 150 miles it encompasses Albuquerque to the north and El Paso to the south. That would mean that hundreds of thousands of people were exposed.**
- **The bomb was a plutonium based bomb and it was packed with 13 pounds of weapons grade plutonium but only 3 pounds of the plutonium fissioned. The remaining 10 pounds of plutonium was joined with the soil, sand, animal and plant life and incinerated. The resultant fireball exceeded the atmosphere and penetrated the stratosphere traveling more than 7 miles high. An ash fell from the sky for days afterwards.**
- **The bomb produced more heat and more light than the sun. Many people who we've spoken to that were alive at the time thought they were experiencing the end of the world.**
- **Plutonium has a half life of more than 24,000 years. Once the radioactive ash fell from the sky as fallout it settled on everything on the soil, in the water and on the skin of every living thing both human and animal.**
- **In 1945 most if not all the small villages inside a 50 mile radius of the Trinity Site had no running water. The water sources at the time were cisterns, holding ponds or irrigation ditches. As a result of the fallout the water sources were contaminated.**
- **In 1945 there were no grocery stores in the small villages surrounding the Trinity site. All the meat, dairy and produce people consumed was raised, harvested or grown by them. It too was contaminated.**
- **Since 1990 the US Government has been compensating "Downwinders" of the Nevada Test Site. The fund set up to extend compensation and medical care is called the Radiation Exposure Compensation Act (RECA). The Downwinders in New Mexico have never been included or compensated although they were the first people to be exposed to radiation any place in the world. Furthermore, it is well documented that the people of New Mexico were downwind of the**

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above ground tests that took place at the Nevada Test Site through the summer of 1962 yet the compensation ends at the Arizona, New Mexico border.

- **The fund has paid out more than 2.5 billion dollars in claims and has provided much needed health care coverage to some of those that qualify. If the health care coverage was extended to the Downwinders of New Mexico it would save lives and reduce the financial burden to patients and families as they travel from their rural communities to receive treatment traveling long distances and sometimes out of State.**
- **The TBDC is fighting for the same compensation and for health care coverage. We often say we don't want one dime more or one dime less than what other Downwinders are receiving today and have received for over 31 years.**
- **On June 27, 2018, Tina Cordova as a representative of the TBDC testified before the U.S. Senate Judiciary Committee about the need to amend the RECA in order to compensate the New Mexico Downwinders. The testimony is available at: https://www.judiciary.senate.gov/meetings/examining-the-eligibility-requirements-for-the-radiation-exposure-compensation-program-to-ensure-all-downwinders-receive-coverage. The hearing begins at 20 minutes. Tina Cordova, co-founder of TBDC, begins her testimony at 1:02:20 minutes.**

On March 24, 2021, Tina Cordova as a representative of the TBDC testified before the U.S. House Judiciary Committee, Subcommittee on the Constitution, Civil Rights and Civil Liberties. The testimony is available at; Examining the Need to Expand Eligibility Under the Radiation Exposure Compensation Act | U.S. House of Representatives Judiciary Committee Tina Cordova, co-founder of the TBDC, begins her testimony at 51:14 minutes.

- **The TBDC is working on behalf of bills introduced in Congress to Amend the RECA to include the people of New Mexico and the Post 71 Uranium miners/workers. 2021 will be the 11th year bills have been introduced in both the House and Senate without a vote on the floor. The original RECA bill will sunset in 2022 so time is of the essence.**

To help the TBDC, sign-up to receive alerts, or learn more about our work please go to: www.trinitydownwinders.com.

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Training School Planes

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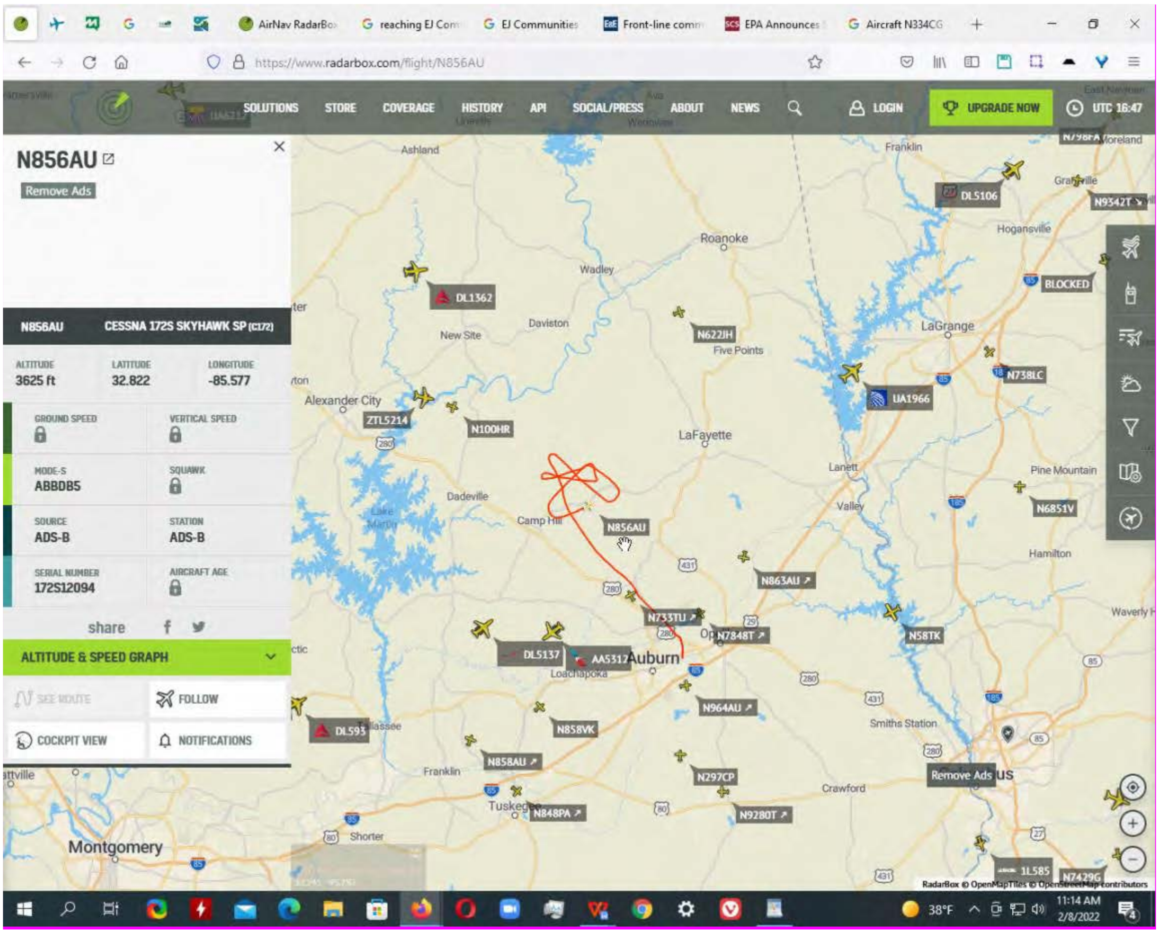

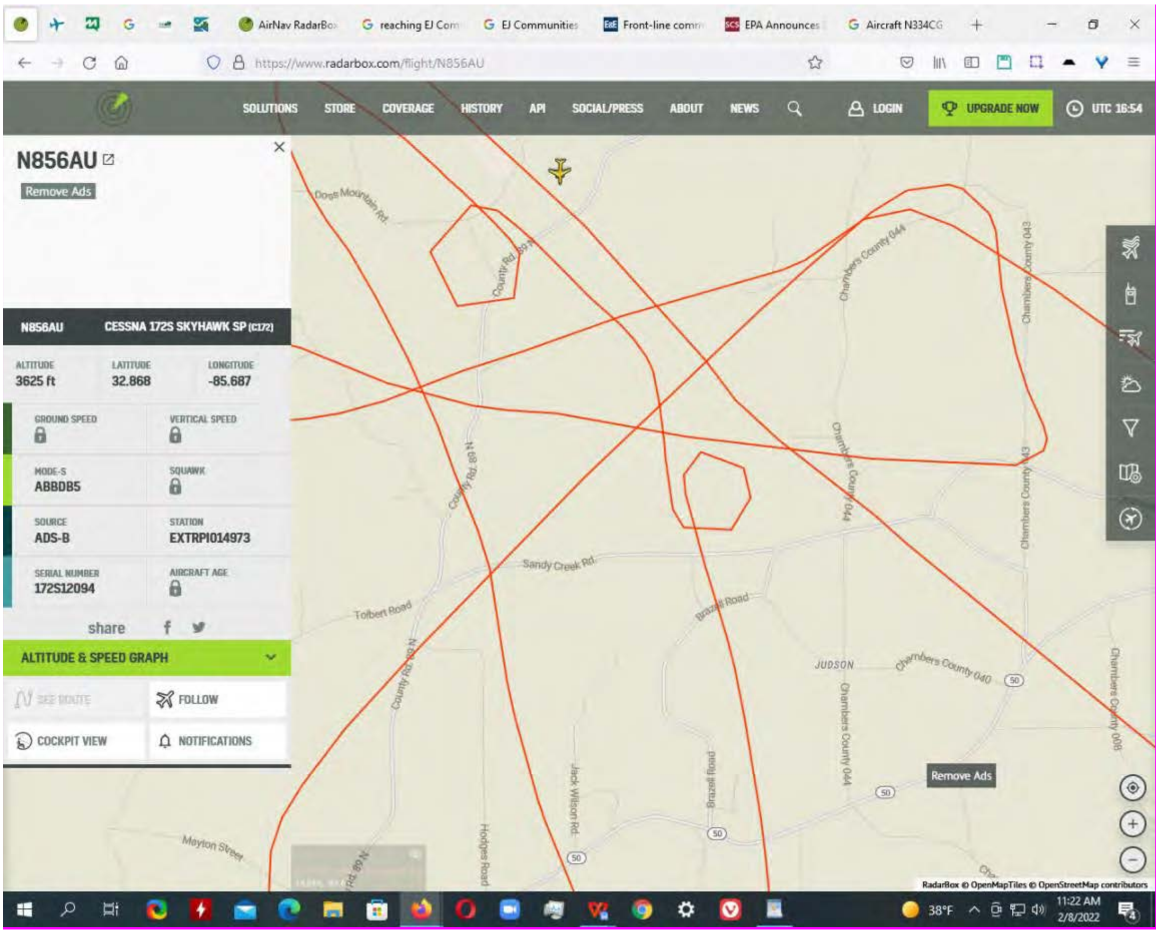
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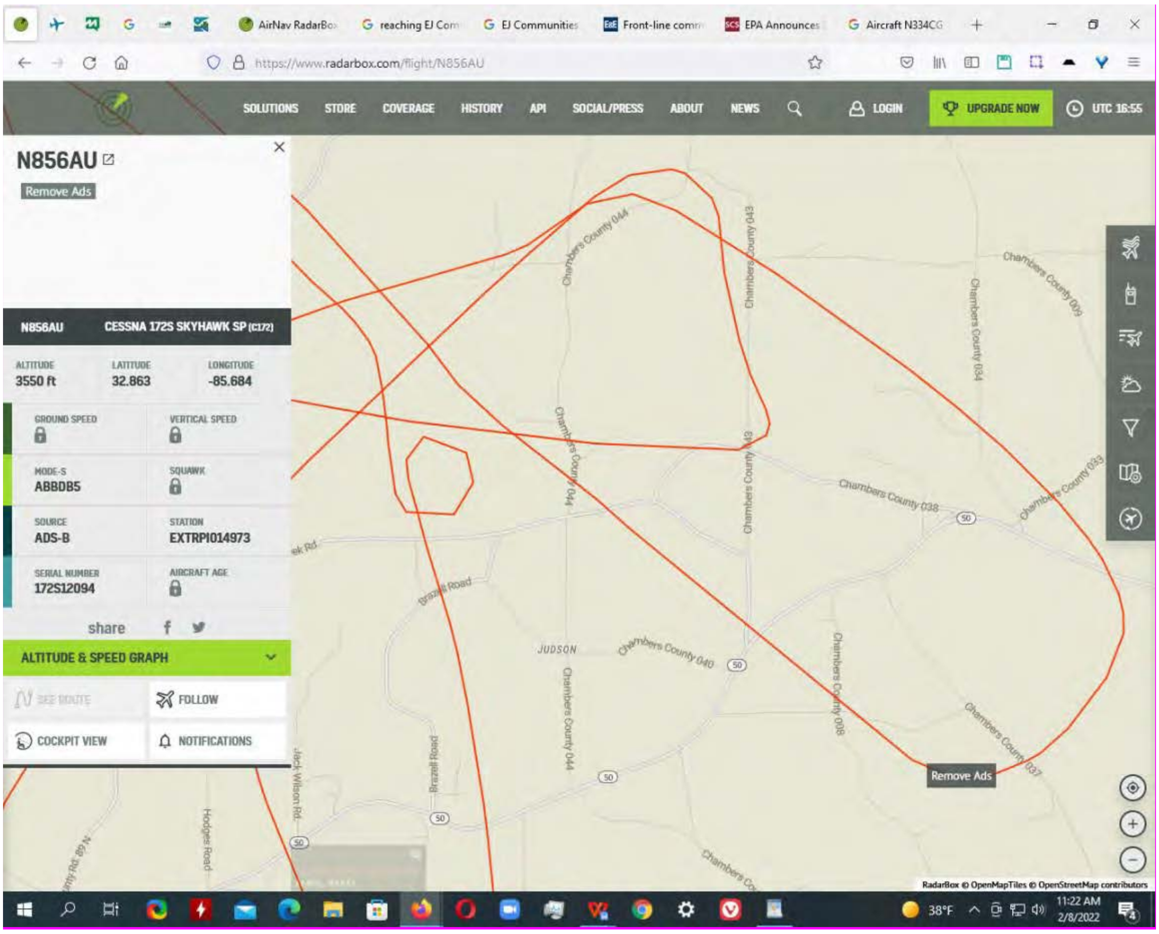
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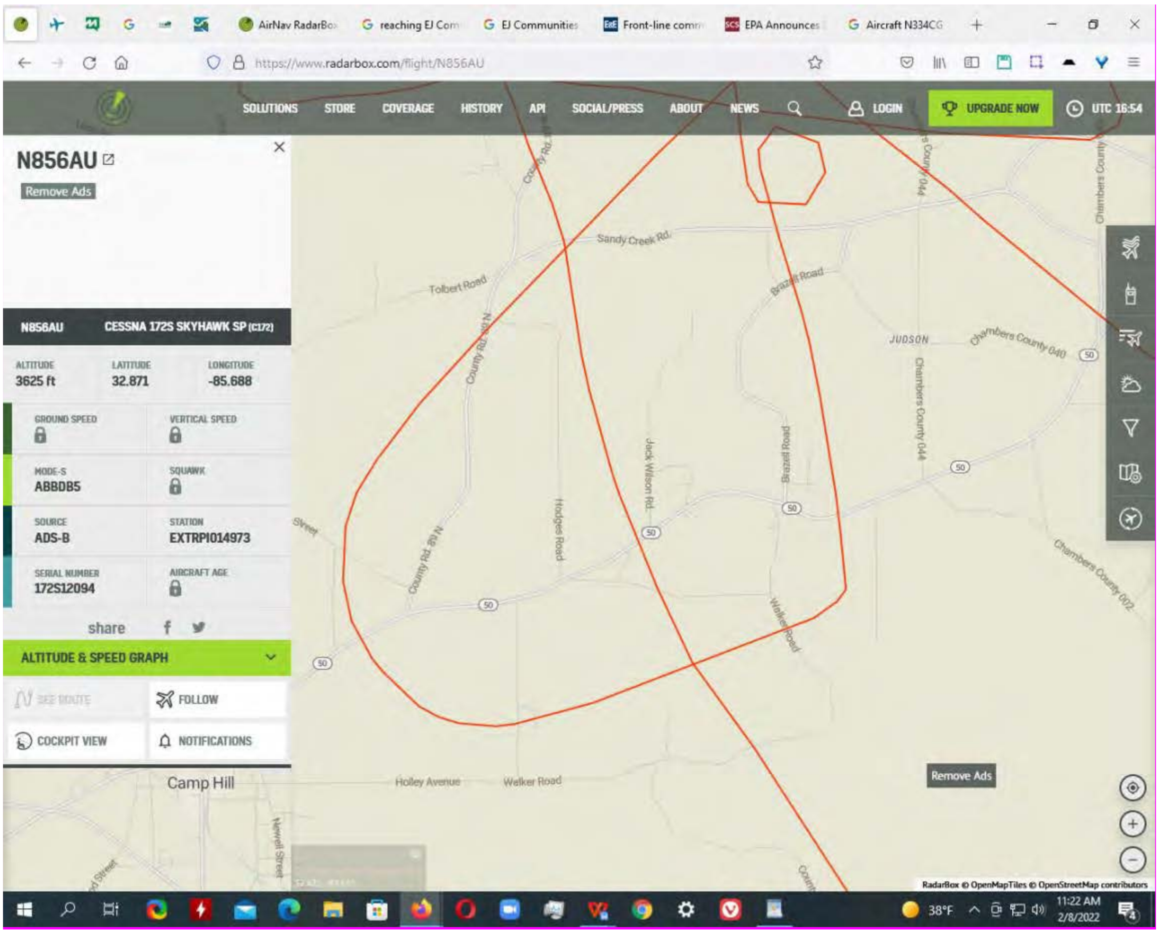

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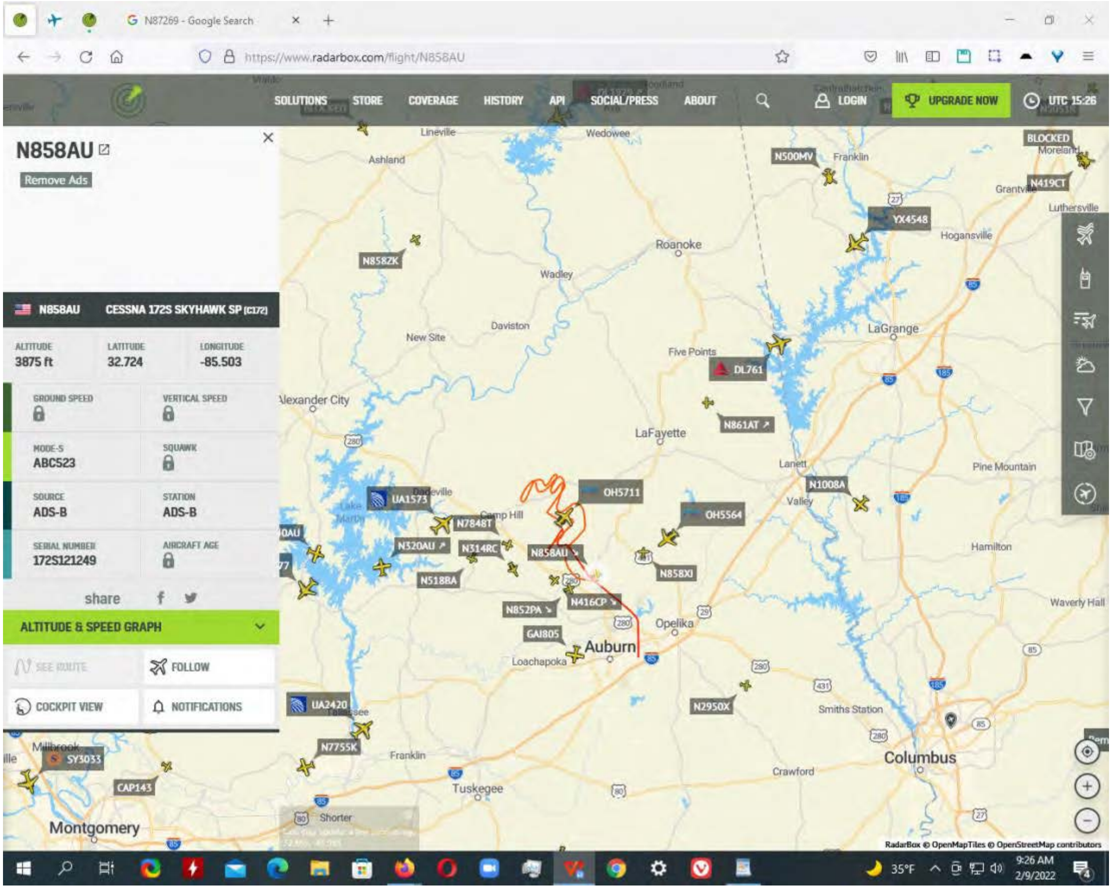
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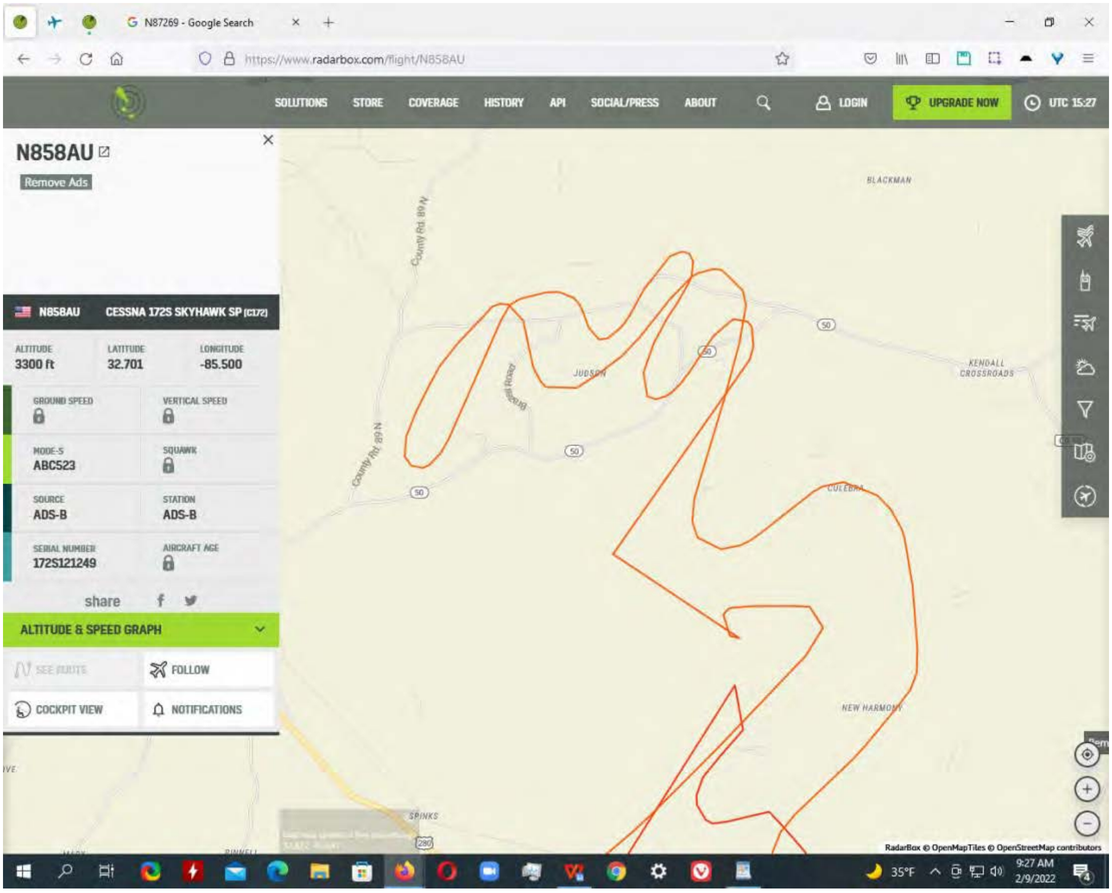
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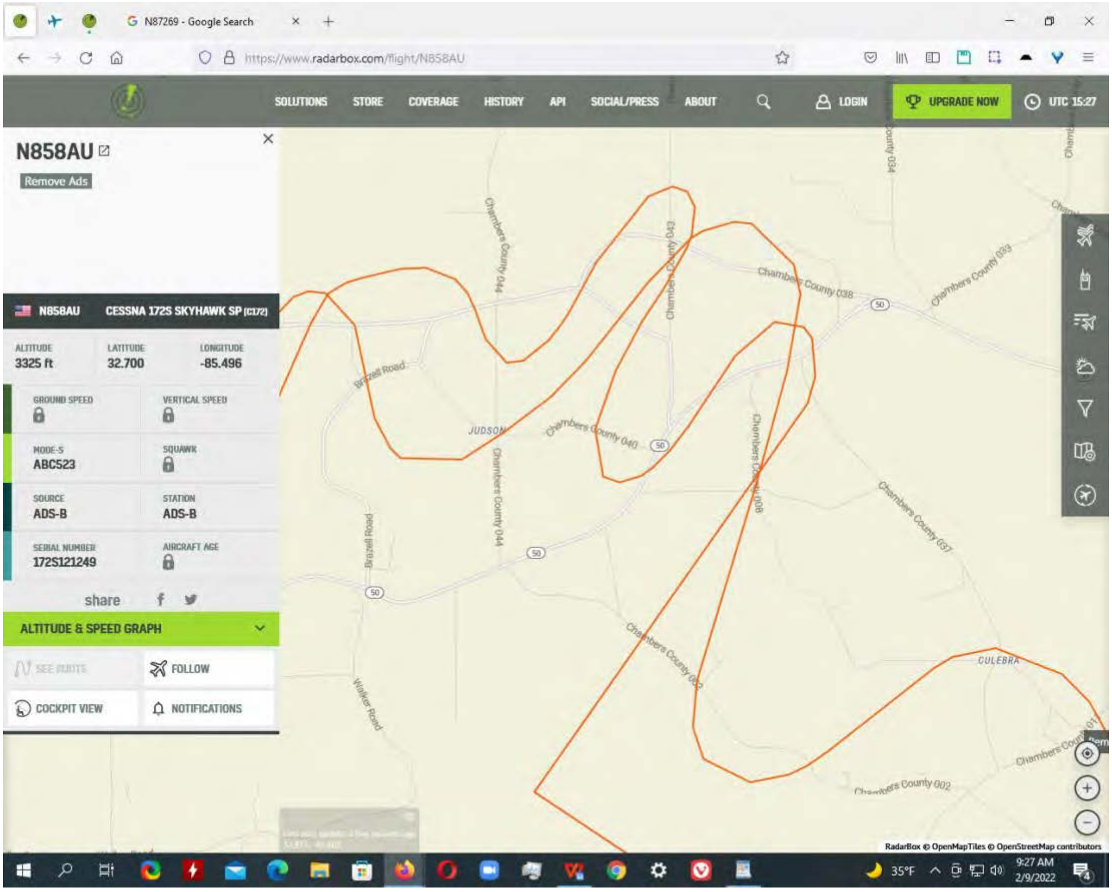

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RESOLUTION
UTE MOUNTAIN TRIBAL COUNCIL
REFERENCE: Opposition to the Proposal by United States National Nuclear Security Administration, Department of Energy to Establish a Strategic Uranium Reserve and Authorization to Submit Comments in Opposition

WHEREAS, the Constitution and By-Laws of the Ute Mountain Ute Tribe, approved June 6, 1940 and subsequently amended, provides in Article III that the governing body of the Ute Mountain Ute Tribe ("Tribe") is the Ute Mountain Ute Tribal Council and sets forth in Article V the powers of the Tribal Council exercised in this Resolution;

WHEREAS, the Tribal Council is responsible for protecting and promoting the best interests of the communities and the members of the Tribe and the resources of the Reservation;

WHEREAS, the White Mesa community sits three miles south of the White Mesa Mill owned and managed by Energy Fuels Resources (USA) which process radioactive waste materials, such as uranium, and has not only far exceeded the period of time for its operations, but has accepted and processed materials that were not a part of its original design and has gone beyond the purposes for which the Mill operates;

WHEREAS, the operations of the White Mesa Mill has had severe health impacts on the residents of White Mesa and should cease entirely;

WHEREAS, in the Consolidated Appropriations Act, 2021, $75,000,000 is set aside for the Department of Energy for a Uranium Reserve Program;

WHEREAS, the Department of Energy, through the National Nuclear Security Administration, has published a Request for Information from the public and has set a deadline of September 10, 2021, to receive comments (See Fed.Reg. Vol. 86, No. 152 pp 44007 to 44009);

WHEREAS, the action taken by this Resolution is in the best interests of the Tribe.

NOW THEREFORE BE IT RESOLVED that the Tribal Council hereby opposes the creation of a Uranium Strategic Reserve and directs the Tribe's Justice Department and Environmental Programs Department to respond to the request for information by September 10, 2021; and

BE IT FINALLY RESOLVED that the Chairman of the Ute Mountain Ute Tribe is authorized to sign this Resolution and to take such further action as may be necessary to carry out the intent of this Resolution.

The foregoing Resolution was duly adopted this 24th day of August, 2021.

Manuel Heart

Manuel Heart, Chairman
Ute Mountain Ute Tribal Council

CERTIFICATION

This is to certify that there was a quorum of 6 Tribal Council Members present at the official meeting of the Ute Mountain Ute Tribal Council held on August 24, 2021, that 5 voted for this Resolution, that 0 opposed with 0 abstaining, and that this Resolution was, therefore, duly adopted.

M. House

Marilynn House, Council Secretary
Ute Mountain Ute Tribal Council

WHEJAC PUBLIC COMMENTS

Submitted by Brenda Vallee, Colfax LA
Central Louisiana Coalition for a Clean & Healthy Environment
bfvallee@bellouth.net

My name is Brenda Vallee, and I represent the Central Louisiana Coalition for a Clean & Healthy Environment. I live near the town of Colfax and near an EJ community known as The Rock. Both are made up of predominately African American residents. The Rock community and my home are less than 2 miles from the OB/OD site operated by Clean Harbors Colfax.

Open burning/open detonation (OB/OD) began at the site in the 1980's. The property was sold several times, and the amount of waste received and burned has grown over the years. Currently Clean Harbors Colfax is the **only commercial** OB/OD facility in the United States. Waste is accepted from many states and some from outside the United States.

So why would a facility that conducts OB/OD want to operate here. Below are some primary reasons.

1. Low income area
2. Property values lower than many places
3. Area of low education
4. Lower property taxes in comparison to facilities that conduct enclosed burning
5. Company could operate and not tell the public what they were really doing; no information given and few questions asked

All of this makes for a quiet, underdeveloped area where people accept what is there without knowing much about it. They just accepted it without questioning.

So what changed? Camp Minden had an emergency. Old munitions exploded and put the area at great risk. Plans were put in place, funds for container b burning were secured and actions began. To help speed along the process Clean Harbors began receiving some of the munitions (with approval from Louisiana Department of Environmental Quality –LDEQ). It did not take long until the loud detonations were heard and offsite plumes became more common. A local newspaper ran a story on the situation, and people became angry, afraid and upset. This was the point at which our coalition began to evolve.

In 2017, our coalition conducted a health survey of many residents of The Rock community and areas close to The Rock. After reviewing the health survey we realized that we had a major health crisis on our hands.

Next we began looking at information from the Louisiana Tumor Registry and the census tracts surrounding the Clean Harbors site. The census tracts involved the Town of Colfax, The Rock and areas close to Clean Harbors. Comparisons were made with the State of Louisiana statistics and specific census tracks around Clean Harbors. What we found alarmed our group. There were elevated cancer rates for lung and bronchus, colon and rectum cancer, female breast and prostate cancer. Many other problems were noted in the survey, namely thyroid problems, skin and eye irritations, respiratory and lung issues plus many more.

At that time we did not have information regarding the burn pits located in the war zones. Today we do, and the health issues regarding our residents and those soldiers who suffered and are suffering from various illnesses are parallel. The OB/OD that occurred in both areas is the common thread.

A bill was presented in a legislative session to ban OB/OD, but it failed. It seemed that profits were more important than people.

It was after that LDEQ began making some efforts to see what was happening at Clean Harbors Colfax. All reports prior to that read **no issues of concern.** Now inspections began taking place and violations were noted. Below is a list of what LDEQ has issued to Clean Harbors Colfax as of this writing.

1. Five (5) Consolidated Compliance Orders & Notice of Potential Penalties

2. Four (4) Notices of Potential Penalties
3. One (1) Penalty Assessment for over $880,000 issued November l8, 2018
4. One (1) Penalty Assessment for $605,000 issued April 7, 2021
5. Over fifty (50) warning letters

It was after further negotiations between Clean Harbors and LDEQ that the $880,000 penalty assessment was put aside in favor of the $605,000 penalty assessment. A major difference between the two(2) penalty assessments was that the complaints from the citizens were left out of the $605,000 penalty assessment,

We wrote a letter in opposition to the $605,000 penalty assessment and were given an appointment with LDEQ staff members to reinforce our opposition. As of now there has been no public release by LDEQ as to what actions will be taken and what the penalty assessment might be.

In the meantime OB/OD continues, people are dying, smoke plumes travel offsite and into homes and yards of the residents, eye and skin irritations continue, breathing problems persist and thyroid issues abound. The many write-ups and other action taken by LDEQ (refer to 5 listed above) have not changed anything for the people here.

Clean Harbors submitted a new RCRA application with OB/OD as the only method to be used in the hazardous waste operation. LDEQ sent it back and told them it was not acceptable. The next application submitted included two (2) methods of destroying hazardous waste, but it also contained OB/OD for about 20% of the materials. There has been no listing of the materials that Clean Harbors said could only be destroyed using OB/OD. **That 20% in the future could amount to the same destroyed in OB/OD there today.**

Open burning/ open detonation of hazardous waste needs to stop. People should not continue to suffer and die.

Below you will find a few pictures with captions that might explain things more clearly.

Offsite plume from Clean Harbors as seen from my home. I live less than 2 miles from the OB/OD facility.

This picture of the OB/OD site has been taken from the LDEQ website. Please note the concrete pads that have been found with cracks on each inspection. This is not a fixable problem. These cracks are a violation of the RCRA permit The site pictured is part of 73.9 acres more or less that is in a conveyance notification filed in our local court house. Ground water contaminants are listed. Document was dated August 16, 2019. On April 30, 2013 another conveyance notice was recorded at our local court house. This involve about 5 acres and is located on what is referred to as Old Burn Area.

Skin irritations from a local Rock Community resident is shown in the picture. Since the time that this picture was taken the resident has died from pancreatic cancer. He lived very close to the Clean Harbor property line.

This is of the lady's arm show skin problems. Residents in nearby houses have similar skin conditions. Please note that the person in previous picture of skin irritations live about a mile apart.

Northeast -1

Maine, Massachusetts, Rhode Island, Connecticut, New Hampshire, Vermont, New York, Pennsylvania, New Jersey, Delaware, Maryland, DC

Dear PA Officials, My ten-year-old granddaughter wants to know why the backyard is without the constant racket when she is outside playing in the snow, unlike when it is warmer and the annoying racket from loud motorcycles bother her and upsets her three year old brother. These loud motorcycles, nearly all Harley-Davidson's, are heard from neighbor's driveways, blocks away on local roads, and from a State Highway located more than a quarter mile away. And no, this is not a local matter, as attested by complaints heard across the Commonwealth, this is a statewide health and safety issue that is being ignored - contributing to the harm of children, the elderly, and those with illnesses aggravated by loud motorcycle exhaust noise. Which elected officials have the guts to stand up to motorcycle special interest groups and will do something to stop these unsafe and harmfully loud motorcycles. In addition to other motorists and the environment, noisy motorcycle exhausts are a constant threat to the welfare of children, the elderly, and those with medical conditions. The current motorcycle exhaust system and noise level regulations in Pennsylvania are useless, and owners of loud motorcycles are well aware of that fact. Motorcycles with harmful and annoying loud exhaust systems cannot be failed at inspection stations, and can operate freely on public roads - intimidating, annoying, and distracting motorists, pedestrians, and cyclists. Regulations need to include 'excessively loud or unusual noise' for exhaust noise violations, with the factory exhaust as the benchmark. Any motorcycle with an exhaust system that can be audibly heard beyond 500 feet should be deemed excessively loud or unusually noisy. [The EPA has required every new motorcycle since 1985 to be no louder than 80 decibels, under all conditions, the same as every other new car, van, and pickup truck] In addition, every motorcycle should be required to have mufflers bearing a valid EPA certification label that matches the motorcycle's EPA emissions label. Motorcycles should be in violation if the factory catalytic converter was tampered with or removed, and not replaced with an EPA certified catalyst. Motorcycle exhaust system and noise compliance regulations should be applied and enforced by inspectors and enforcement. Please reply with what actions you will be taking to ensure my grandchildren, and every other child, will be able to enjoy the outside year-round without hearing unsafe and harmfully loud motorcycle exhaust noise. If no action is to be to be taken, then please explain why not. P.S. - If motorcyclists are relying on the false Loud Pipes-Saves Lives claim to be safe, then they should be required to participate in the Pennsylvania Motorcycle Safety Program training course to learn how to properly operate their motorcycle and stay safe, which is done daily by conscientious motorcyclists who have quiet EPA labeled factory or aftermarket mufflers on their motorcycles. Respectfully, Anthony Lombardy. Besides licensed inspection stations not being permitted to fail a motorcycle with a noisy exhaust, regardless of how loud the exhaust is, PennDOT has acknowledged roadside motorcycle exhaust noise testing by enforcement is not being conducted. It was suggested current Pennsylvania regulations for determining exhaust noise compliance are too complex and cannot be realized outside of a controlled environment. Plus, vehicle inspectors at licensed Vehicle Inspection Facilities are not permitted to test noise levels by any means. They are only permitted to perform a visual inspection of the motorcycle exhaust system. Besides being a nuisance, loud motorcycles are a health and safety concern for citizens – harming the hearing of children, aggravating medical conditions, and intentionally distracting other motorists. Citizens should be able to go into their backyard to enjoy a nice day with their children or grandchildren without the loud obnoxious roar from loud motorcycles that scare and upset these young children, driving them back inside the house. Or the homeowner who wants to enjoy a peaceful day in their backyard but is unable - not due to cars, trucks, buses, or tractor trailers - but the motorcycles that can be heard well above every other vehicle. Multiple studies have proven that the 'Loud Pipes-Saves Lives' mantra that is repeated by supporters of loud motorcycles is false and dangerous. Loud motorcycle exhausts prevent the rider from hearing emergency sirens and vehicle horns, as well as dangerously distract drivers around them. Plus the noise is heard at the back of the motorcycle, which is too late to be heard when the majority of accidents involving motorcycles is from other vehicles making a turn in front of them, or the motorcycle turning into another vehicle. Enforceable regulations that are being successfully applied in other venues: 1- Vehicle code violations that ban an exhaust system that emits a loud or unusual noise. 2- An exhaust system that can be heard more than 500 feet away. 3- A muffler that does not have a valid EPA label that matches the motorcycle frame EPA Emissions label; 4- Disallowing exhaust systems louder than factory. [Since 1985, new motorcycles, including Harley-Davidson's, are equipped with mufflers that limit exhaust noise to 80 decibels, the same as every other new vehicle] 5- Informing vehicle inspectors and enforcement that motorcycles are no louder from the factory than other vehicles. 6- Requiring vehicle inspectors to check motorcycle exhausts for non-complying noise levels in addition to a visual inspection of the exhaust system. 7- Banning the removal or tampering of the factory installed catalytic converter, and requiring an EPA certified catalyst be properly installed. Many motorcycle organizations and groups, such as ABATE, routinely lobby for the ability of their members to install loud mufflers on their motorcycles. This is accomplished by ensuring motorcycle inspection and noise compliance laws are useless. One way is by giving the false impression that changing or enforcing the laws is unpatriotic, dishonoring veterans who ride unsafe and harmfully loud motorcycles.

Hello WHEJAC, I understand that the CHPE path goes very close to this site. One cannot know for sure as Champlain Hudson Power Express (CHPE) /Transmission Developers Inc. (TDI) still has not chosen to reveal it's final trajectory. The coal ash pile was subdivided out of the major parcel of the Lovitt property at the point in time when Mirant was financially restructuring. In light of the attached letter, we know the coal ash pile is leaking. What might the effects be of high definition drilling, jet plowing, trenching and ground disturbance on the coal ash pile which has been leaking toxic chemicals without remediation for the past ten years? The entire site has been totally ignored. This issue should have been included in the EIS except that as CHPE has changed paths so many times already and refuses to reveal the actual trajectory, this issue was not included. Because the issue of the toxic coal ash waste into the Hudson River has not been addressed, what will the impact be for the 7 Towns and their drinking water supply (already concerned about PCB's PFA's and other toxins) considering the Hudson River Flows Both Ways? Why wasn't the issue of the 7 Towns drinking water part of a FEIS? No one has even brought up the issue of the leakage of radioactive nuclides/isotopes from Indian Point Nuclear Power Plant that leak directly into the Hudson River. One may be able to capture some of the PCB's, PFA's may be able to be captured with new technologies in the future (but what to do with the PFA waste - like nuclear waste - PFA's are forever chemicals!) but radioactive nuclides and isotopes cannot be contained or captured. There are NO safe levels for drinking radioactive waste. Because of the deep and destructive process of jet plowing in the Hudson River - 105 miles - for CHPE, I fear this action will create several serious health issues that must be looked at. I feel that even the potential for radioactive nuclides and isotopes in drinking water should be enough to stop this project. I saw a news release from the EPA today regarding

49 Superfund sites that are having money allocated through Biden's infrastructure Bill for remediation/clean up. This one in Stony Point has been on the National Priorities List Superfund Site for much longer than many of the others that were picked. Why not bump it up and make this one No. 50? - a nice round number. Thank you once again and have a happy holiday and New Year! Thank you so much, jacqui Drechsler

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Forty-five degrees on Christmas Day is unusual in these parts. Unfortunately, inconsiderate neighbors took out their loud Harley-Davidson's and are revving the engines, ruining what would had been an enjoyable Christmas with our grandchildren. Besides terrorizing the children, the racket is detrimentally effecting my wife's fibromyalgia and upsetting her mother who has dementia. These wannabe 'bikers' are now riding around the neighborhood blasting their exhausts, which can be heard blocks away. Disgraceful. Cannot complain to the local authorities because the borough exempts motorcycles with valid safety inspection stickers from noise codes. State and local enforcement also wrongly use the issuance of an approval sticker as proof that the motorcycle mufflers are compliant. This could not be further from the truth. Inspection stations are NOT permitted to fail motorcycles for noise, regardless of how loud and noisy the exhaust. [PA law states inspectors are not to issue an approval sticker unless the motorcycle complies with established noise levels, but PennDOT does not enforce that part of the regulation] Someone once 'jokingly' said fire truck sirens are so loud because they need to be heard above loud Harley-Davidson motorcycles. Guess what. Respectfully, Anthony Lombardy

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Full Name (First and Last): Karen Spencer
Name of Organization or Community:
City and State: Gloucester, MA
Brief description about the concern: "New evidence questions existing policies about the safety of fluoride for babies' developing brains. Given that safe alternatives are available and that there is no benefit of fluoride to babies' teeth before they erupt or appear, it is time to protect those who are most vulnerable." - *Bruce Lanphear MD, PhD; Christine Till PhD; & Linda S. Birnbaum PhD in "It is time to protect kids' developing brains from fluoride." Environmental Health News (October 7, 2020)* "I would advise them (pregnant women) to drink bottled water or filtered water..." *- Dimitri Christakis, MD, MPH, editor in chief of JAMA Pediatrics on "Association Between Maternal Fluoride Exposure During Pregnancy and IQ Scores in Offspring in Canada" (August 19, 2019)* "There are fluoride-free strategies in which to prevent dental caries. Given the current levels of exposure, policies should reduce and work toward eliminating avoidable sources of fluoride, including water fluoridation, fluoride-containing dental materials, and other fluoridated products, as means to promote dental and overall health." - *International Academy of Oral Medicine and Toxicology (IAOMT 2017)* WHEJAC - Over 98% of the fluoridation chemicals added to minimal water supplies go directly to waste water. These chemicals are sourced from the pollution control systems of industry. My community imports our product from Shanghai; it is literally Shanghai smog that we add to city water which drains into the harbor of America's oldest seaport, Gloucester, MA. When we clean our equipment, the residue is sent to hazardous waste disposal sites. Although our local Conservation Commission and Shellfish Commission object to this policy, fluoride holds a protected pollutant status, ostensibly because a handful of local dentists and the local BOH claim it prevents cavities - a tooth-fairy myth. Fluoridation policy damages the teeth and health of millions of Americans with heightened susceptibility to harm in environmental justice communities - scientific and medical facts which are supported by countless studies, many NIH funded and published in recent years. I am attaching the following documents on this matter: An

annotated bibliography of dozens of scientific studies & reports published since 2015 documenting harm A bibliography of environmental science with my city's conservation commission statement attached A one page handout on the political squashing of disproportionate harm to environmental justice communities Frankly, I am amazed that the fluoridation cult continues to hold sway within the US government. Consider: EPA scientists documented that fluoride (a regulated water pollutant) is a 'gold standard' developmental toxicant with substantial evidence of harm to humans (Mundy et al. 2009 & 2015). LULAC identified fluoridation as a civil rights violation in 2011 and civil rights leaders such as Ambassador Andrew Young have repeated spoken out against fluoridation as has environmentalist Lois Gibbs of CHEJ and safe water advocate Erin Brockovich The 2006 National Research Council recommended then that the MCLG be lowered as it was not supportive of health. Moreover, the NRC chided the EPA that there was no evidence of safety to any population, let alone susceptible subpopulations that included pregnant women & their fetuses, bottle-fed babies and young children, the elderly and any with chronic conditions such as low-thyroid, kidney disease or diabetes. Sixteen years later, and the EPA has failed to do address these gaps. BOTTOMLINE: If an individual doesn't believe topical use of fluoride in dental products is sufficient for their needs, it is cheap to buy a gallon of fluoridated drinking water. However, for low-income populations, many with circumstances putting them at heightened risk of toxicity which include diabetes and CKD, conditions that result in higher than average water consumption and which that disproportionately affect EJ populations, it is very costly to have to purchase bottled water for all consumption and in some cases topical use to prevent even more costly harm to bodies, bones and brains.

Full Name (First and Last): Jack Crowther
Name of Organization or Community:
City and State: Rutland, VT
Brief description about the concern: Dear White House Environmental Justice Advisory Council, Our experience as a society beset with problems points to a truism: When systemic problems develop, when numbers of people suffer injury, the less-advantaged suffer disproportionately. This is injustice. My specific concern is fluoridation of public water supplies, a practice that science, ethics, and common sense increasingly discredits. Fluoridation is known to have negative health impacts across a wide spectrum that includes brain damage to infants, tooth mottling, bone fractures, harm to thyroid and kidney function, gastric distress, and chemical sensitivity. Others will have provided the peer-reviewed science to support these claims. What we all know by now is that economic privilege can reduce the harm of health stresses. We know that the profiles of the certain minority populations, notably Black, may include generally lower income, health vulnerabilities, and toxic exposures driven by lack of housing choices. Ending fluoridation of public water supplies will greatly reduce exposure to a protoplasmic poison, a developmental neurotoxin, and an endocrine disruptor — all potentially harmful to people in general and particularly to those whose environment, broadly speaking, makes them particularly vulnerable.

Full Name (First and Last): Paul Connett
Name of Organization or Community: Fluoride Action Network
City and State: Exeter, NH
Brief description about the concern: Dear White House Environmental Justice Advisory Council, In 2015 I co-authored a 135-page paper entitled Water Fluoridation and Environmental Justice. This paper was

written in response to a request for public comment from the newly formed US government Environmental Justice Interagency Working Group. Despite the many hours of work I and other members of the Fluoride Action Network (FAN) put into this paper we received neither acknowledgement nor a thank you from those who solicited public comment. Nevertheless, some 6 years later much of what we wrote stands up well today and I encourage Council members to give it a read. In particular, section 9 points out the lack of U.S. studies up to that time (2015) which had seriously investigated fluoride's potential to cause harmful health effects. There have been some very important developments since 2015. Two FAN initiatives in 2016 In 2016, FAN did two things which focused on fluoride's neurotoxicity, an issue FAN has followed closely since its formation in 2000. 1) FAN requested the National Toxicology Program (NTP) to undertake a systematic review of all the studies (animal, human and cellular) pertaining to fluoride's potential to damage the brain, and 2) FAN petitioned the EPA under provisions in the TSCA law to ban the deliberate addition of fluoridation chemicals to the drinking water supply because it poses an unreasonable risk to the developing brains of children. A game-changing development in 2017 Both these initiatives have yielded very important results as I will discuss below. Both were enhanced by a dramatic game-changing development. Even though many fluoride-IQ studies have been published in China, India, Mexico and Iran since the late 1980s, in 2017 the first of four US government-funded studies (Bashash, 2017, 2018; Green, 2019 and Till, 2020) were published in major journals including Environmental Health Perspectives and JAMA Pediatrics. The earlier studies (particularly from China), while yielding very consistent results, were of an ecological design (exposures were based on community water levels not individual measurements) and a number were at a higher concentration (2 to 11 ppm, see the 2012 Harvard meta-analysis of 27 studies, Choi et al., 2012) than used in water fluoridation programs in the USA (0.7 to 1.2 ppm). The very rigorous high-quality US government funded studies were based on individual measurements of both exposure and outcome, controlled for a large number of confounding variables and were conducted either in fluoridated communities at 0.7 ppm (Green, 2019 and Till, 2020) or in communities with exposures (from other sources) in the same range as fluoridated communities (Bashash, 2017 and 2018). Moreover, these US government funded studies for the first time examined in utero exposure. Fetal exposure was measured via examining the pregnant women's urine fluoride levels. Such measurements indicate total exposure to fluoride from all sources, which allows comparisons between communities and countries regardless of water fluoridation status. From the results obtained in both the Bashash and Green studies it is clear that the most sensitive period as far as fluoride impacting brain development is concerned (at least for boys) is during the fetal stage. However, Till's research in 2020 also showed that early infancy is another very vulnerable period. Till found a large significant lowering of IQ for children who were bottle-fed in fluoridated communities in Canada (F level = 0.7 ppm) compared to those who were bottle-fed in non-fluoridated communities. So while the water fluoridation debate has framed the issue of whether or not we should add fluoride to the drinking water, maybe a better frame would be should we add fluoride to the amniotic fluid of the fetus from its first day of existence! Based on the findings in the Bashash and Green Studies the shocking conclusion is that today water fluoridation is causing a greater overall loss of IQ points to America's children than any other factor including lead exposure and preterm birth The NTP's systematic review of fluoride's neurotoxicity (2016-2022). This has been the most thorough review of fluoride's neurotoxicity ever undertaken. Their final report is due in March 2022. Draft versions have indicated that of 29 High Quality (i.e. low risk of bias), 27 found a lowering of IQ. Of these 13 were conducted at 0.7 ppm or lower; another 5 conducted between 0.7 and 1.5 ppm and 5 at 1.5 ppm or higher. In other words, the claim by fluoridation promoters that the lowering of IQ only occurs at much higher concentrations than used in artificial fluoridation programs is false. Please Note: finding a lowering of IQ at 1.5 ppm offers no adequate margin of safety when you are exposing a large population of children to 0.7 ppm of fluoride in their drinking water. There are two reasons for this a) children drink different amounts of water and b) there is a wide range of sensitivity to any toxic substance among a large population. Typically, regulatory agencies like the EPA would like a margin of safety of 10, in this case 1.5 ppm only offers a margin of safety of 2. The TSCA lawsuit against the US EPA In 2017, the EPA rejected our TSCA petition on scientific grounds which allowed us to take the matter to Federal court (Region 9, San Francisco). Our case was held in June 2020 (via Zoom – with 500 observers). FAN was able to obtain expert testimony from Howard Hu (director of the ELEMENT cohort in Mexico City which was used in the Bashash, 2017 and 2018 studies; Bruce Lanphear, a world-renowned expert on lead's neurotoxicity and co-author of the Green, 2019 and Till, 2021 studies and Philippe Grandjean, a world-renowned expert on mercury's neurotoxicity and author of a risk assessment (BMD analysis) on fluoride's neurotoxicity (LINK). One couldn't imagine a more qualified team to have available for establishing that the addition of fluoride to public drinking water poses an unreasonable risk for the brain development of America's children. To almost everyone's surprise (including our own) for its experts the EPA lawyers chose not to use scientists from the agency, but instead used experts from the firm Exponent, Inc. This firm is notorious for being highly industry-friendly defending the safety of such chemical villains as dioxins, PCBs, PFOS and Monsanto's glyphosate. The judge in this case (Judge Chen) is following the science very closely and this trial offers the world a rare opportunity to examine the science of this matter on a level playing field. In this situation the EPA cannot expect any deference based on its regulatory authority. The Judge will rule on the scientific merits of our case. One important concession made by Exponent's experts is that the four US government-funded studies (Bashash, 2017, 2018; Green, 2019 and Till, 2020) included as evidence in our case, are the highest quality human studies on fluoride conducted to date. The Judge has delayed his ruling on this case until he has been able to read the final report of the NTP's review of fluoride's neurotoxicity) expected in March 2022, Grandjean's BMD analysis, a study from Spain and possibly other studies which have been published since June 2020 (of which there are several). Unless the EPA concedes the case, it is anticipated there will be another round of expert testimony probably sometime in the fall of 2022. The glaring irony in this case, however, is that the science being presented as evidence for the plaintiffs comprises the essential science EPA needs to revise its regulatory limits for fluoride in drinking water (currently the Maximum Contaminant Level Goal (MCLG) and the Maximum Contaminant level (MCL) are both currently set at 4 ppm, based on crippling skeletal fluorosis as the most sensitive end point!) The need for such revision was the overarching conclusion in the NRC's 2006 landmark report on fluoride in drinking water, a need EPA has not been able to properly address since that report was published at their own (EPA's) request. A victory in our lawsuit would most likely necessitate setting the MCLG at zero (like lead and arsenic) and the MCL (an enforceable standard) no higher than 0.1 ppm, based on protecting children's brains. Such an MCL would doubtless trigger opposition from states with high natural levels of fluoride who would probably fight spending money to remove the natural fluoride from their water supply. The beauty of FAN's TSCA lawsuit is that it begs that debate by simply calling for a ban on the deliberate addition of fluoride to drinking water. So what has all this cutting edge science have to do with the Environmental Justice community? In our 2015 paper we drew attention to the many ways that communities of color are disproportionally impacted by fluoride. This is especially true of fluoride's impact on the brain because inner city children are known to have been historically exposed to higher levels of other neurotoxic substances in their air, water and in local soils. Knowingly, adding to this burden is unconscionable. Fluoridation has been promoted as closing the gap in dental care between rich and poor, which is certainly a noble intention but whether that has been achieved or not is debatable (see the Cochrane Review of 2015 which found “insufficient evidence to determine whether water fluoridation results in changes in disparities in caries levels across socio-economic status”) it is time a safer way be found to bring better dental care to low income families. Childhood tooth decay notoriously remains at epidemic levels in the inner cities and other disadvantaged areas. The quickest and simplest way of achieving better dental care as well as improving the overall health of communities of color In our 2015 paper we provided some very positive suggestions on how better dental care could be delivered to low income families and communities of color, which would not only improve dental care but also improve overall health and community well-being. I urge you to review section 22, on page 64 of our 2015 paper. Finally, on behalf of all the disadvantaged and disproportionately harmed environmental justice communities who have no choice but to drink fluoridated tap water, FAN hereby requests that WHEJAC recommend, in strongest terms possible, that EPA leadership resolve to concede in the referenced TSCA lawsuit. Thank you for this opportunity to contribute to your valuable mission during these challenging times.

Full Name (First and Last): Rick North
Name of Organization or Community:
City and State: Wellesley, MA
Brief description about the concern: My name is Rick North. I’m the former executive vice president (CEO) of the Oregon American Cancer Society and former project director for the Oregon Physicians for Social Responsibility. Now retired, I have over 30 years’ experience in nonprofit health and environmental management. Most of my life I believed the CDC’s and American Dental Association’s assertions that water fluoridation was “safe and effective.” When I actually examined the science, I was taken aback. Fluoridation’s effectiveness was minimal, at best, and there were numerous associated health risks, as identified by the National Academy of Science’s (NAS) authoritative 2006 review, Fluoride in Drinking Water (https://www.nap.edu/catalog/11571/fluoride-in-drinking-water-a-scientific-review-of-epas-standards). Since its publication, hundreds of other peer-reviewed, published studies have added even more evidence of these risks. My main purpose in writing is to document the evidence that fluoridation harms, not helps, low-income families. First, please consider fluoridation’s lack of effectiveness and clear evidence that fluoride’s preventive actions are mainly topical, not ingested. The Cochrane Collaboration is considered the gold standard for evaluating effectiveness of medical interventions. Its 2015 report on fluoridation (https://www.cochranelibrary.com/cdsr/doi/10.1002/14651858.CD010856.pub2/full) found 4,677 published studies in its exhaustive literature search. Of those, only 277 studies qualified for the first round of review, with 155 meeting Cochrane’s highest quality criteria for inclusion in the study. The report concluded “There is insufficient evidence to determine whether water fluoridation results in a change in disparities in caries levels across socio-economic status.” (https://fluoridealert.org/articles/fan-brochure-fluoridation-efficacy-one-pager/) CDC itself has said “. . . fluoride prevents dental caries predominately after eruption of the tooth into the mouth, and its actions primarily are topical for both adults and children.” https://www.cdc.gov/mmwr/preview/mmwrhtml/mm4841a1.htm) Even CDC’s belief that fluoridation results in a 25% reduction of caries, which in itself is highly questionable, only equates to half a cavity per child. Finally, World Health Organization data clearly show that cavity rates in children have dropped as much in nations that don’t fluoridate as in nations that do.

(https://fluoridealert.org/issues/caries/who-data/) Then consider ingested fluoride's health risks, which, as cited in NAS's Fluoride in Drinking Water, include brain damage, hypothyroidism, kidney damage, diabetes and fluorosis. To take just one example, neurotoxicity, please note the National Toxicology Program's 2020 systematic review (https://fluoridealert.org/wp-content/uploads/ntp.revised-monograph.9-16-2020.pdf), which found compelling data linking fluoride to IQ loss in children. Several of the most recent studies were on pregnant women consuming fluoride or babies being fed formula mixed with fluoridated water: – 25 of 27 of the studies determined to be the highest quality linked higher fluoride levels to substantially lower IQs – 11 of 11 studies detected this IQ loss at levels found in fluoridated water Low-income families also use more infant formula. A 2019 high-level Canadian study funded by the U.S. National Institutes of Health determined that babies fed formula mixed with fluoridated water averaged 4 IQ points less than those mixed with non-fluoridated water, 9 points lower in non-verbal IQ. (https://www.sciencedirect.com/science/article/pii/S0160412019326145?via%3Dihub) The chances for academic achievement for low-income children and future success as adults are already diminished by poor nutrition and other environmental pollutants, such as lead in pipes, as shown in Flint, MI and other cities. It is not right to expose them to another toxic substance further reducing their capabilities. Cheap water filters don't eliminate fluoride. Low-income families can't afford expensive filters (typically at least $300/$400) or bottled water to avoid the health risks of fluoridated water. They have no choice. And since Black and Hispanic families are more likely to be below the poverty level, they are disproportionately harmed. Such notable Black civil rights leaders as former U.N. ambassador Andrew Young, Rev. Gerald Durley and Rev. Bernice King, daughter of Martin Luther King, Jr., have publicly opposed fluoridation. And LULAC, the nation's oldest and largest Hispanic advocacy organization, is also formally opposed. (http://fluoridealert.org/wp-content/uploads/FAN-Environmental-Justice-Brochure-Final.pdf) If people want fluoride, they can get it very inexpensively in toothpaste or mouthwash and apply it topically, where it's most effective. But no one should have the right to force anyone else to ingest a drug that they don't want in their drinking water. This is clearly a social and environmental justice issue. Fluoridation is unethical for low-income families and should be ended immediately. Thank you for your consideration.

Full Name (First and Last): Heidi S. Slatkin
Name of Organization or Community: Post Road Cooperative
City and State: Bronx, NY
Brief description about the concern: Pre-war buildings need to be insulated and electrified to prepare for a fossil-free future. The cost will be astronomically expensive for all owners except for the ultra-wealthy. We need federal and state subsidy to pay for these upgrades. Otherwise, we'll lose our pre-war buildings to either the ultra-wealthy, including mega landlords who own dozens of buildings, or to new construction. The middle class of cooperative owners will disappear if we don't get funding for these upgrades.

What do you want the WHEJAC to advise the White House Council on Environmental Quality to do?: Initiate federal and state programs for co-operatives to apply for grants to upgrade buildings on a building-by-building basis, to prepare for a fossil-free future.

To Whom It May Concern; I was glad to participate as a registered call-in person during the meeting regarding the Strategy Plan. Although most people focused on issues to do with their own communities, I focused on the damages of large-scale non-renewable hydropower worldwide as it is a major contributor of emissions that are not counted, creating great harms to Indigenous People's, the environment and all life, with green house gasses, methane and methylmercury. I do hope my comments were not only recorded but transcribed as well. I will take this opportunity to comment on issues in my community. 1.We fought off an approved by the PSC, Desalination Plant that wanted to be built by a private water company supplier - Suez. We won on two counts: Radioactive nuclides from Indian Point Nuclear Power Plant could not be removed from the water supply of the Hudson River and that Suez supplied fraudulent information on "need" to the PSC which we proved was misinformation. We, however, are paying the surcharge of 83 million dollars for a plant that was never built and should not have been approved. 2. We successfully fought off a "gasification plant" to be built on the shores of the Hudson River - whereby NYC waste would be brought by barge and "gasified." 3, We fought to successfully shut down Indian Point Nuclear Reactor. We have NOT won the fight to get a 42 inch high pressure gas pipeline from landing at Indian Point and although the facility is now shut down, the Spectra Enbridge pipeline - and others in the vicinity remain a huge risk to 20 million New Yorker's safety. This pipeline was approved by the NRC, PHMSA, PSC and FERC. We are still fighting to get this 42 inch high pressure pipeline shut down during the remediation of Indian Point. Furthermore, this pipeline goes through Rockland County and has a dirty compressor station, which when combined with the SouthEast Compressor Station has cumulative negative effects on people's health. 4. The criminal company Holtec won the bid for decommissioning Indian Point. The NRC and PSC have gone against public interest by allowing a company that has no experience and produces faulty thin casks to have the job. Which leads me to Holtec's plan to transport hi-level, high-burn fuel nuclear waste from Indian Point to Utah. Jonathan Perry, who was on the January 5, 2022 call - in from Region 6 should be very worried. I cannot attach as a document but refer you to the news release from Trains News Wire on Spent Nuclear Fuel Railcar Testing, The DoE has contributed more than10 million dollars to "move by rail". 5. In Rockland County we have a very limited water supply which must be gained in - county. The PSC has determined that a hostile take-over by Veolia of Suez is "in the public's interest." It is not in our interests. Veolia is not only a bigger and badder French owned company than Suez, it also is partly owned by the French Government and has a government official as a sitting Board Member.We used the opportunity of the public hearing to advocate for a feasibility study on the possibility to create our own Public Water Authority. 6. Pollution knows no bounds. We continue to have an alliance with our Ramapough -Lenape relatives in Rockland County and Ringwood and Mahwah, New Jersey, who have been fighting for years regarding the Ford Plant Sludge. Absolutely criminal. Considered remediated, oversight every five years??! 7.Bomb Trains which go around Rockland Lake with the possibilty of polluting our water supply. I personally fought to make sure that our Sheriff' and first responders have real-time knowledge of what, when, where and how they come through Rockland County. Bomb Barges in the Hudson River. Riverkeeper successfully fought this off. CAN YOU IMAGINE A BOMB TRAIN COLLIDING WITH A HIGH-BURN-UP NUCLEAR FUEL WASTE TRAIN? CAN YOU IMAGINE OUR ONLY WATER SUPPLY POISONED BY A BOMB TRAIN?? CAN YOU IMAGINE THE HI-PRESSURE PIPELINE EXPLODING AT INDIAN POINT??? I was recently on a webinar with Secretary Granholm regarding Hydro Quebec's Champlain Hudson Power Express - during which she said: " Getting to the hearts and minds of opposition activists - to change their minds and get them to accept Canadian Hydropower - this is what keeps me up at night." All of the above and Canadian Hydropower is what keeps ME up at night. Champlain Hudson Power Express aka CHPE owned by Hydro Quebec, Blackstone and Transmission Developers Inc. is not only not actually needed as a power source for NYC, but is incredibly damaging to New York - the economy, our environment and the world. 105 miles of the Hudson River - a Scenic

Historic National Site, will be power plowed, dredged and immensely disturbed. The electro magnetic fields created will interfere with the fish in the Hudson River- they have their own EMF systems they rely on - their entire life cycles will be hurt, the PCB's (from GE which have not been fully remediated) toxins and radioactive nuclides will be shot up in huge plumes which may affect the water quality for the 7 Towns that rely on the Hudson River water (above Indian Point) for their drinking water and daily needs. By the way - Indian Point still leaks radioactive water into the Hudson River - the river known as The River That Flows Both Ways. There is a Coal Ash Pile very close to the shores of our Hudson River in Stony Point which did not make the cut for remediation for the EPA's new picks. Blackstone and TDI have bought the land surrounding that Coal Ash Pile and we fear that the disruption of the building of CHPE will create more leakage from that Pile into the Hudson River. The maritime industry is opposed.The "right" to allow CHPE, for "public need" will create an opportunity for many other "cable transmission projects" to go through. The Great, Wide and Beautiful Hudson River - all Rivers for that matter- should not be a transmission transportation system for for-profit, privately and foreign government owned private merchant lines. This sets a very bad precedent.The environmental organizations such as Riverkepper, Sierra Club and the North American Megadam Resistance Alliance (NAMRA) are opposed. Former Governor Cuomo wanted it (one of his staff advisors was Mulrow - a Blackstone man} and current Governor Kathy Hochul wants it. Pretty sure Mulrow is still on staff. Therefore NYSERDA and the PSC wants it. Large-scale non-renewable hydropower is created with cement/ concrete - which all by itself contributes around 8% of green house gasses (GHG) world wide. In Canada there is the flooding and cutting down of nature's wonder - carbon capturing Boreal Forests. Then the impoundments/reservoirs are created - flooding entire communities. This also includes the diversion of Rivers.The communities must relocate - sometimes at a moments notice, losing their land, sacred sites and burial grounds, Then they have to live with poisoned waters - methane is created and the very dangerous neurotoxin Methylmercury. These waters poison their sources of food, and now - they live with food scarcity, most do not have electricity or running - clean water themselves. All for us. Would we accept this here? No. Why then, do we accept it there and accept truly dirty energy as a "clean energy" for us? Just because when it arrives - there are "no emissions" - there are great emissions from the creation of large-scale hydro energy. The production of hydroelectricity worldwide is melting the permafrost. The companies engaged in this dirty creation - self- report "no emissions" as the emissions are considered non-combustible.I have spent the past ten years fighting against CHPE. I have recently been told by Mr. Peter Keane, Esq. of NYSERDA that I must get all of the big environmental agencies to put in comments by February 7, 2022 in order to fight this.Why must there always be a fight? By the time this is in place, it will be totally moot as New York works towards true renewable energy for our State - wind farms and solar. CHPE is now greenwashing health as the reason this needs to go through. That they will be cutting down on approximately 3 billion dollars of health care related costs by not having bad air quality from gas fired polluting power plants. Yes, stop the plants - but at the same time they deny they have any emissions and that they do no harm. Governor Cuomo wanted this so much - he changed the rules to allow a non-renewable and polluting source of energy, which in it's production creates more GHG emissions than gas or coal fired power plants to be considered clean and renewable. I am 63 years old and have spent much of my life involved in fights for justice and for the environment. I know you are working to make sure the two go hand-in-hand, but everything always seems too little too late. Why have I written so much - and what I wrote is only half of what I could have written? To make a couple of points. 1. All Federal agencies need to know what the other is doing. Is the NRC pushing moving radioactive materials and the DoE encouraging this? Does the PSC really work on behalf of the public or are they working for corporate greed? Does the EPA work with DEC? Why did FERC allow a design on the back of a paper napkin presented to NRC and PHMSA for a high-pressure pipeline at Indian Point, to be considered a good thing? Does one hand know what the other does and does anyone care? 2. Pollution -whether from hydroelectricity or gas or coal fired power plants finds it's way through the air, land and water, across all borders. The United States Policies on Clean Air and Water and Environmental Justice MUST extend beyond our borders. Our policies must include others - the ones who bear the brunt of energy needs for us. Is a disadvantaged community only here in the United States?We must not be complicit in harm to others. The word collusion has two meanings. 1. To "play nicely" with others and 2. To collude - "secret or illegal cooperation or conspiracy, especially in order to cheat or deceive others." I offer all of the examples above as what I consider collusion. From water issues, to bomb train issues, to Indian Point issues, to Pipeline issues. PSC, FERC, PHMSA, NRC, and many more - all supposed to be for the environment and the public's interest. - pick any acronym and any issue and there will be a connection between politicians, agencies and corporations. Please make EJ Policies not just National - but International. Sincerely, jacquelyn Drechsler

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Full Name (First and Last): Angeline Cione
Name of Organization or Community: Federal Community
City and State: Washington, DC
Brief description about the concern: The most economically disadvantaged communities and businesses lack access to energy efficiency and water efficiency services that could save them money and empower them to be part of the climate solution. The common question is how to unite environmental justice goals with federal goals to address climate change.

What do you want the WHEJAC to advise the White House Council on Environmental Quality to do?: In your implementation guidance for EO 14008, require federal agencies that use performance contracting to require bidders to provide evidence (reference projects) that they provide energy efficiency services to low-income communities. Energy Act of 2020 requires federal agencies to use performance contracting for 50% of their energy efficiency measures. This mechanism provides a quicker funding source for agencies, but the federal government pays the price over time. That money benefits the (already big businesses), the Energy Service Company (ESCO) but doesn't benefit the broader community - unless those ESCOs have an incentive to provide their services to the struggling private sector. This would help tie together the EO's Environmental Justice goals with its goals to tackle climate change.

Full Name (First and Last): Catherine Skopic
Name of Organization or Community: Grow Renewable New York - NO Canadian Hydro
City and State: New York, New York
Brief description about the concern: Renewable Energy Only - Preserve Hudson River - No CHPE

What do you want the WHEJAC to advise the White House Council on Environmental Quality to do?:

1. Rescind the Presidential permit allowing energy cables from Canada to New York City 2. Recognize the racial, environmental, economic injustice of CHPE - Champlain-Hudson Power Express 3. Reject the CHPE Project

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Full Name (First and Last): Elisabeth Anderson
Name of Organization or Community: Medford, MA
City and State: Medford, MA

Brief description about the concern: It is unconscionable to subject tax-paying citizens to the concentrated flight paths and resulting dramatic increase in airplane noise, as a result of NextGen navigation. There are frequently days of 200-350 flights directly over "sacrificial" communities such as mine, at low altitudes and with decibel meter readings of 70-85 db per flight. Let's not pretend to think it a fair approach that the FAA calculates the "average" of decibel readings over a given site, a calculation which incorporates flights at high altitudes with low decibel readings, resulting in a skewed average. Let's take the average decibel readings of those flights with decibel readings over and above the noise limits allowed by city noise ordinances to fully realize the deafening and damaging effect these flight paths are having on communities. Citizens are selling their homes to escape the noise, are uprooting children from their schools to escape the noise. NextGen navigation is destroying lives and communities.

What do you want the WHEJAC to advise the White House Council on Environmental Quality to do?: The concentrated flight corridors as a result of NextGen navigation are having a deleterious impact on "sacrificial" communities. Every community wants and benefits from the advantages of an airport; no community wants the concomitant airplane noise. The only fair solution is to disperse the airplane noise, disperse the flights.

Full Name (First and Last): Shawn Merritt
Name of Organization or Community: Ayer Ma.
City and State: Ayer MA
Brief description about the concern: The noise in Ayer MA from the train depot (including the loading and driving of tractor trailers at the train depot), air traffic, Army base and local auto cross track makes for a a very uncomfortable living experience in Ayer. We have more than our fair share of loud noises and noise pollution.

What do you want the WHEJAC to advise the White House Council on Environmental Quality to do?: I would like to see a reduction of excessive industrial noise through the use of electric trains *and the greasing of all squeaky parts of rail cars etc, rerouted trucking routes and a relocation of the autocross track to another facility. I think there should be a nation wide effort to reduce excessive industrial noise wherever possible.

Full Name (First and Last): Sofia Petsas
Name of Organization or Community: Plane Sense
City and State: Flushing NY
Brief description about the concern: Noise levels in minority areas are unacceptable. Government must regulate towards communities first and foremost.

What do you want the WHEJAC to advise the White House Council on Environmental Quality to do?: Plane patterns must adjust for noise pollution and community concerns first if government is at all helpful in fixing this crisis.

Full Name (First and Last): Elaine M. Miller
Name of Organization or Community: Plane Sense 4 Long Island
City and State: Malverne
Brief description about the concern: Since the implementation of NextGen flight paths communities across America have been subjected to hundreds and hundreds of low flying planes over their homes on

a daily basis. Noise and toxic emissions are a major health and environmental issues that must be addressed. Numerous studies have shown the harmful impact of noise and emissions on the people living directly under the flight paths. It is time for our elected officials in government to take a strong position to protect people from dangerous health impacts.

What do you want the WHEJAC to advise the White House Council on Environmental Quality to do?: The living conditions and exposure to harmful levels of noise and emissions due to excessive overflights MUST be effectively dealt with by WHEJAC. You can no longer turn a blind eye to the devastating living conditions which are communities are forced to endure. It is incumbent upon WHEJAC to take positive measures to solve this problem. The American people should not have to suffer because our government refuses to act.

To Whom it may concern, I am writing to make the following noise statement: Mara Van Fleet Administrator for A Peaceful Sky Facebook group Chappaqua, NY HPN Westchester county airport has more than quadrupled its air traffic since 2017. The main issue is that private and business aviation has grown to 85% of the traffic and has no limitations at all. We have planes frequently every 2 minutes. At times every 90 seconds for a long duration of time. There are many flights that are scheduled after midnight that run all night waking residents up. We can only have a voluntary curfew. There are a significant number of private planes that use leaded fuel that fly low and frequently over our homes and schools. We need to be tested for lead poisoning. We have engaged with our airport extensively and with our county executive and BOL to zero effect. In fact it only has gotten worse. An airport official told me that the customs office at HPN closes at 11:00 pm and that no internationals arrive after midnight. This is not true. See attached photo of a plane that arrived after midnight from Saudi Arabia last night. Where do these people go if the customs office is closed? Why would this plane land here and not the main NYC airports? We desperately need help. They are landing every plane over the same homes when flights can be dispersed to alleviate suffering. For myself personally, the increased and incessant air traffic has given me a heart condition (hypertension) for which I am on medication now permanently since the planes aren't dispersing and I get nearly every single one over my home. Please see attached photos. I am 8 miles out from HPN yet planes fly this low over my home. They used to fly higher and no one can answer us as to why this has changed. Jet Blue in particular seems to fly in ways that are outright deliberate, flying low and making sharp turns with severe banking (without any other traffic nearby), similar to how the planes looked going into the trade center on 911. This is a frightening sight, and to me terrorizing. Again, trying to get an explanation as to why they do this failed. No other airline flies like this over my home. Thank you for taking the time to read my statement. We are suffering 24/7.

Full Name (First and Last): Amy McCoy
Name of Organization or Community: Town of Ayer
City and State: Ayer, MA
Brief description about the concern: Thank you for the opportunity to comment. My area in MA has been in a long battle against the noise and lead pollution caused by recreational aircraft flight maneuvering repeatedly over a small geographic area. Even though noise is a state and local issue, all local officials point to the FAA as the regulator with the power to make a change, however FAA's Regional Administrator for New England, Colleen D'Alessandro, stated to me, "The FAA does not engage with individual private citizens regarding aviation noise" and "repeat complaints of the same or similar nature will not be responded to by either my office or the Boston FSDO." Two of the busiest flight training areas established by area flight schools are over Massachusetts EJ communities. This comes as no surprise. When area residents first brought noise concerns to FAA's Nancy Risso of the Boston FSDO,

she stated flight training was not able to take place "over wealthy housing." 200,000 propeller planes are registered in the United States and countless people on the ground are subjected to the noise and lead emissions from these aircraft.

What do you want the WHEJAC to advise the White House Council on Environmental Quality to do?: Ban leaded aviation fuel. Support regulations that give the power to enforce noise and pollution laws, from any source, back to local control. Support controls that establish mandatory fines for violating noise ordinances. Disallow the concentration of flight maneuvers over areas that are not airports. Require noise impact studies with real noise measurement, not noise modeling. Formulate regulation to keep quiet places quiet (state and national parks for example) so there is a place for all to experience quiet.

Full Name (First and Last): Jean Quartiano
Name of Organization or Community: 10,0000 Hawks
City and State: East Haven, CT
Brief description about the concern: Concerns are the terrible loud noise and pollution caused by Avela 737's planes that fly directly over my house several times a day and night beginning 7:15am and into the late evening. It's affecting my sleep and nerves. The planes fly so low I freeze in anticipation for it to crash into my house, it's very scary.

What do you want the WHEJAC to advise the White House Council on Environmental Quality to do?: To please listen to the little guy and help, we need a voice for us. I invite anyone to spend a day at my residence and see first hand how terrible it is to have to live with this problem.

Full Name (First and Last): Mayce Torress
Name of Organization or Community: Resident
City and State: New haven ct
Brief description about the concern: We own a house near airport and the noise is a problem.
This residential property near airports was cheaper and more affordable to a person dreaming about a better especially belonging to black and brown vulnerable communities, the concern is impact it has on residents' health. People who live near an airport have a higher risk of developing high blood pressure (hypertension) and heart disease, asthma, lung cancer, chronic lung disease, and depression. The pollution of the aircrafts are a hazard. ... Emission from the aircraft can also contain soot. All houses in the immediate surrounding the entire airport should get aid windows airtight. Sun panels to help efficient. Air conditioning that is clean.

What do you want the WHEJAC to advise the White House Council on Environmental Quality to do?: Because we are living and raising families. We pay taxes. We matter!

Full Name (First and Last): Rachel Winston
Name of Organization or Community: 10,000 Hawks
City and State: East Haven
Brief description about the concern: The Tweed New Haven airport expansion is a deep concern of mine. When I moved into my home, I did research on potential expansion and I was told that there was an agreement that it would not expand. So clearly this has been a concern since I have lived in my home. I have serious concerns for my two daughters, my husband and I, in terms of the increase in pollution

due to the bigger plans and more frequent flights. This has a huge impact on our community and we have to live with it daily, for others to be convenienced intermittently. They can go to Hartford instead. Thank you for your time!

What do you want the WHEJAC to advise the White House Council on Environmental Quality to do?: Not allow the bigger planes and the frequency. Go back to how it was before. Thank you!

Full Name (First and Last): Maya Bentz
Name of Organization or Community: Quiet Skies JFK - Farmingdale, NY
City and State: Farmingdale
Brief description about the concern: We live across the Bethpage State Park in the Village of Farmingdale, Nassau, NY, 11735. We are tormented with unbearable noise from JFK's arriving planes affecting our health and well-being. We filed hundreds of complaints with JFK but have not heard back from them. Planes fly over our house day and night, polluting our environment, causing excruciating noise, damaging our health, and making it impossible to enjoy outdoors in our backyard. We already live in the noise-sensitive area due to circling our houses, low flying student pilot planes, helicopters, and private planes from the Republic Airport at Farmingdale, NY. There is no reason for JFK to include our neighborhood in the loop of their arriving planes. They can easily redirect planes to the areas not affected by the Republic Airport noise. In addition, we pay high taxes in the Village of Farmingdale and cannot find any quiet time to rest in our backyard after work or during the weekends.

What do you want the WHEJAC to advise the White House Council on Environmental Quality to do?: Stop hundreds of JFK Airport planes from daily intruding into the Republic Airport airspace and regularly flying over Farmingdale and Bethpage State Park. The residents are already suffering from constant aircraft noise and negative environmental impact from the Republic Airport without added noise and pollution from the JFK planes.

To whom it may concern, I am reaching out to express my experience with aircraft noise.
Before I moved to New Hyde Park, I lived in Queens. Then, we decided to move to LI because my two older sons had joined the military and were no longer living home. That, plus the idea that my daughter needed to go to a different sort of high school to attend than what the city had offered.
Little did I know that realtors were not under any mandate to inform us that our home was under a flight path for JFK. ————-That is when the problems began-Twenty -four hour landings of planes rocketing over my home. Fuel turning my siding grey. The pictures in my home perpetually crooked from the vibrations from the loud , large planes going overhead. The worst came- I started having breathing problems and am now on singular for allergy problems.————Do not tell me this is my imagination. Do not tell me planes make noise.This is due to the collaborative effects of the FAA and the airlines to deprive me of my life, liberty and happiness. T his is due to insensitive,self centered denial. This is the antithesis of the American ideal. Please help our family and others like mine before the environmental damage will be too great to fix. Stacey Korman Vargas Planesense4LI New Hyde Park, NY

Full Name (First and Last): Lisa Avery
Name of Organization or Community: Medford, MA
City and State: Medford
Brief description about the concern: I've lived in Medford, Massachusetts under Boston Logan airport's hyperconcentrated flight paths years. Beginning in 2013, Boston Logan implemented the FAA's horrendous and cruel Next Gen flight navigation, which concentrated flight paths into narrow corridors

over the airport's sacrifice communities. Since then we have lived with a nightmare of near constant noise and pollution from flights arriving and departing, very often, every single minute for hours and hours and days on end. Medford is also the site of a TEKK point where ALL planes departing specific runways must fly to, before they are routed to their destinations. If the daytime noise and pollution weren't enough, we wake to flights low and loud enough to shake the house- at all times into the late night and early morning. We live in fear of the return of sleepless nights, constant noise and showers of pollution post COVID. Numerous studies have made clear the detrimental impacts of both noise and air pollution; this cannot be disputed. Studies have also shown that it is a small percentage of frequent fliers that account for a majority of flights. It is no surprise that the world's wealthiest are accountable for the most emissions - on the back of the poorest communities, while airlines continue to rake in the profits with no accountability.

What do you want the WHEJAC to advise the White House Council on Environmental Quality to do?: Noise proof windows are not the answer. Airports and airlines need to be accountable to their communities and accountable for their pollution. Community concerns are completely ignored in favor of airline and airport priorties. WHEJAC needs to push for stronger FAA oversight. No one seems to know who the FAA is accountable to. Is it the airlines, lobbyists or Americans? WHEJAC needs to make sure community and environmental concerns are a priority - not airline profits. States and cities need more say in airport expansions, and when and where planes can and cannot fly. Having been involved in the community appeal around the FAA's unjust RNAV implementation for years, and witnessed FAA's series of delay tactics, I hold little hope that any council recommendations will result in any meaningful change. If it isn't obviously clear that moving ALL airport noise and pollution over a small sacrifice community isn't just completely wrong, then what else is there to say?

Full Name (First and Last): Marilyn Dolgoff
Name of Organization or Community: BWI/DCMetroplex
City and State: Ellicott City
Brief description about the concern: We live under the FAA Nextgen flight paths out of BWI and surrounding airports. We live 21 miles from the nearest airport. Without warning or environmental review, the FAA moved flight paths over our area in April 2018, two years after they were established in other areas. Since that time we have been tasked with saving our lives from the FAA and those serving in government at all levels. We moved into our home in a suburban area 29 years before the flight paths were moved over us. We depend on our well for safe water, our house was built with most of the rooms exposed to the roof. As a result of low flying aircraft, it is nearly impossible to get enough sleep, scientists found heavy metals with an airport signature in our dust sample, and now our medical tests indicate high lead levels. We contacted legislators, local and state, and environmental agencies, also local and state about the catastrophic changes to our environment. They have all used deception and deflection when we seek help. After many requests for help, and countless complaints, we only get lower flying aircraft from all over. That is why I use the word catastrophic to describe our alarm about a U.S. government agency causing serious health damage and destruction to a once healthy environment. I have information I would like to share that provides clear evidence of retaliatory behavior since we live in an area with 10,000s of open acres. I would like to submit videos, data, and screen shots of hundreds of low flying helicopters, as low as 95 feet, private jets, propellers buzzing our roof, freight jets doing drive and dive arrivals (same jet 3 times in 13 min, Amazon, UPS, etc., along with SWA, UAL and many other airlines. The brutality of the actions of FAA/MAA is clear and potentially deadly.

What do you want the WHEJAC to advise the White House Council on Environmental Quality to do?:

Please look into why we must try protect ourselves from the damage caused because we live under nearly all BWI departures and many arrival paths(some as low as 1,400 feet) . Thank you for taking the time to read our story. There are so many like us all over this country seeking protection from the FAA and local airport authorities.

Full Name (First and Last): Jackie Toth
Name of Organization or Community: Good Energy Collective
City and State: Washington, D.C.
Brief description about the concern: (Please see email attachment sent to whejac@epa.gov on Feb. 7 at 8:21pm ET for our full comment.) Injustice has been a defining characteristic of the United States' legacy with nuclear energy. If nuclear energy is to continue to play a part in a sustainable, progressive vision for our domestic energy system, the federal government and the nuclear industry must first work to address the historically inequitable balance between who has experienced the benefits and the detriments, respectively, of nuclear technology.

What do you want the WHEJAC to advise the White House Council on Environmental Quality to do?: (Please see email attachment sent to whejac@epa.gov on Feb. 7 at 8:21pm ET for our full comment.) Recommendation 1: Recommend to the CEQ that the following investments be explicitly included in the definition of what counts as a benefit for disadvantaged communities under Justice40 in the category of Remediation and Reduction of Legacy Pollution: "Restoration of uranium mines, uranium mills, and nuclear weapons test sites." Recommendation 2: Establish a WHEJAC working group to explore potential recommendations across the full suite of legacy waste issues in the United States, inclusive of uranium mining and uranium milling and military toxics. The working group could in part act to identify the most polluted among the abandoned uranium mines and military testing sites to help establish a list of the top-priority sites for clean-up. Recommendation 3: As the WHEJAC members' time allows, consider reaching out to the DOE Office of Nuclear Energy to exchange information and ideas and to hear what the department is doing to embed environmental justice considerations into the agency's work.

Full Name (First and Last): Amy Myers
Name of Organization or Community: 10,000 Hawks
City and State: New Haven, CT
Brief description about the concern: Noise and environmental pollution. There is a new airline in the airport near where I live. There used to only be puddle jumpers because this airport is in the middle of a coastal neighborhood. Now there are huge jets taking off several times per day. This coastal neighborhood is a designated Important Bird Area and is home to hundreds of salt water marsh bird and other animal species that are being impacted by the air and run off pollution of these jets. I live 1.7 miles from the airport and every time a jet takes off, the noise over my home is incredible, my house shakes. My home has also become my workplace in the past 2 years. This noise is enormously disruptive to my sleep, my quality of life and to my workday

What do you want the WHEJAC to advise the White House Council on Environmental Quality to do?: Place regulations on this airport. It is a tiny airport that operated for decades flying tiny puddle-jumper planes a couple of times per day. There is no reason to have an airport that is flying these kind and size of jets numerous times per day in this neighborhood. There is an international airport 55 miles away and three more airports 80-90 miles away. Those airports are appropriately not in the middle of a neighborhood. This airport should have never expanded and is needs to be regulated back down.

Full Name (First and Last): Mrs Kathryn M Gallicchio
Name of Organization or Community: 10,000 Hawks, New Haven, CT
City and State: New Haven
Brief description about the concern: For over 20 years, my family has lived in a residential section of New Haven which surrounds Tweed-New Haven Airport to the north and west. The eastern side of the airport is bordered by the town of East Haven, another residential zone. Both neighborhoods are working class, composed of small single family or multi-unit, older homes. To the south, the flight paths from Tweed overfly protected greenspace which is the site of a yearly raptor migration that draws ornithologists and birding enthusiasts from all over the country. Once known as an Italian-American enclave of New Haven, my own neighborhood has in recent years drawn many Black and Hispanic families. The East Haven side is also heavily populated by recent Hispanic and Asian immigrants. There are several schools within a few miles of Tweed, including one large elementary school only 4 blocks away. This community already has some of the highest rates of asthma in the state. Tweed is a small, regional airport which opened in 1931. It was rather foolishly located in a flood plain encompassed by tidal salt marshes, and parts of it are only 2-3 feet above current sea levels. FEMA's published projections show that much of it will be underwater by 2050, due to climate change. Local access roads already flood regularly during high tide or storm events, and the homes surrounding the airport suffer from run-off from its parking and runway pavement. Nevertheless, the airport is there, and it has continued to operate since then primarily for small, general aviation aircraft, student pilots, and emergency flights to/from local Yale-New Haven Hospital. Several commercial airlines have attempted to maintain flight schedules between one larger air hub or another, but none have found demand sustainable at the price point they offered, and they have all eventually pulled out. Bradley International Airport is only a 75 minute drive up Interstate 95, Providence airport is 90 minutes to the east, and of course the large facilities at JFK and LaGuardia are only 2 hours to the west. Private corporate jets make frequent use of the current runway at Tweed, so local power brokers (read: Yale University) who want more direct access to New Haven proper already have it. However, there is currently a 'master plan' being advanced by the Tweed-New Haven Airport Authority to expand the main runway at Tweed to almost the length of the runways at LaGuardia, and to build a new, six-gate terminal on the eastern side of the airport in order to accomodate a new, budget airline which has contracted to "manage" the airport for the next 43 years (getting out just before the airport floods for good, per FEMA) The "improvements" in this plan also open the door to freight traffic, and if it is enacted, the airport could soon rival Bradley, hosting between 15-30 take offs/landings of Boeing 737s every day/night.
The plan is currently undergoing an environmental assessment, according to the demands of the FAA, prior to approval. Unfortunately, the FAA is not an objective overseer of this process, as its role is expressly to promote as well as regulate aviation in the US. While the EA must follow guidelines set forth under NEPA, many of those considerations amount to nothing more than checking a box (“yeah, we considered the effects on rates of children's asthma--we're not worried about it”) rather than a serious investigation of the long-term, cumulative impacts of such a huge expansion of air traffic in this dense residential community and the fragile ecosystem it forms a part of. There is a strong sense among many in this community that this expansion is a "done deal", that the fix is in, that the concerns of local residents for the health of their children, the local wetlands, their economic well-being and the shared planet amount to nothing when pitted against corporate and political interests. The way in which the process is unfolding is the very definition of environmental injustice: it is being controlled and manipulated by the very people who stand to gain from the project--the wealthier, whiter portions of the city and state--while the people who will bear the harms are being ignored. The EA process is supposed to have full public noticing and opportunity for input, but the first meeting announcing it was streamed over Facebook (a private platform) and not well publicized in the community, nor offered with Spanish language interpretation. Covid restrictions have limited the opportunity for in-person

public meetings, and the process to comment online is both opaque and one directional. And so Tweed's master plan marches toward FAA approval with little attention given to the voices of the environmental justice community being directly affected.

What do you want the WHEJAC to advise the White House Council on Environmental Quality to do?: I applaud the Biden administration's declaration that issues surrounding climate change and environmental justice will be made integral to federal actions. With that in view, I ask that a moratorium be placed on the FAA's approval of the Tweed plan or any other aviation infrastructure project until the EA/EIS/FONSI process can be reconfigured to give higher priority to those issues, so that decisions regarding our children's and planet's health are no longer a matter of "business as usual". NEPA guidelines need to be rewritten to reflect more current scientific consensus about the cumulative effects of particulate pollution, VOCs, noise levels, etc. FAA approval for aviation expansion should REQUIRE that adequate carbon offsets be included in all plans. The FAA's oversight of the EA process should itself be subject to review, since the agency's mandate creates a conflict of interest in these matters, stacking the deck against local communities. Thank you for your time. Sincerely, Kathryn Gallicchio

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Full Name (First and Last): Alex Vassallo
Name of Organization or Community: Plane Sense 4 Long Island
City and State: Long Beach, NY
Brief description about the concern: The Federal Aviation Administration (FAA)'s flight paths of arrival into John F. Kennedy International Airport (JFK)'s runways 31L and 31R are a source of constant noise bombardment to the residents of Long Beach. This constant airplane noise hits this community every 90 seconds at altitudes under 2,000 feet, and the practice continues at all hours of the day or night, often waking me up and causing substantial stress. I have recently been told that I had elevated blood pressure, possibly the result of this situation. The FAA, despite being offered specific suggestions to address the noise, has not taken any significant action and has shown no concern to the people of Long Island. Indeed, it appears FAA has even started to lower altitudes of these arriving flights from their existing levels. The FAA needs to be held accountable, reverse their Performance Based Navigation (PBN) procedures, contained within the agency's 'NextGen' policies, and bring real noise relief to all Americans who are being affected by these wrongheaded, industry-biased decisions.

What do you want the WHEJAC to advise the White House Council on Environmental Quality to do?: I want the WHEJAC to recognize the real world effects of airplane noise on human health and begin to more strongly regulate the issue. The EPA should no longer recognize the FAA's Day/Night Average (DNL) sound metric to assess the impact of aviation noise on communities and implement a more strict metric to control the impact of this pollution. The FAA should no longer be in charge of the process of environmental review of flight paths and any associated changes.

Full Name (First and Last): Davie Matheu
Name of Organization or Community: Private Citizen
City and State: Arlington, Massachusetts
Brief description about the concern: The FAA introduced a new plan for flight paths in 2012 in our community, which focused all departing aircraft into an exceedingly narrow corridor causing an enormous increase in noise pollution in our neighborhood, as well as in others, and a likely increase in dangerous particulate pollution. This cynical program was intended to boost intensity of aircraft operation on the same airport "footprint", at the behest of the airline industry that the FAA de-facto works for. The noise pollution caused by this is beyond maddening. I am forced to leave my home on

RNAV days. The noise is endless, ceaseless, drives us out of sleep and makes outside conversation impossible. It is unjust to pour all the pollution and all the consequences of increased airline activity onto a small population, just so that Logan Airport operators can make larger bonuses, and airlines can save money consolidating operations into Logan. Undoing RNAV might make flights might cost more and take longer -- but this is just! Flyers and the flying industry should pay the full costs of flying -- not privatize the gains and discharge the losses onto communities below! The FAA has polluted communities with noise, across the country, through RNAV and through the badly flawed and unbalanced 1990 Airport Noise and Capacity Act, so that airlines can make more money and airport operators can collect more landing fees. This regulatory-capture behavior needs to stop.

What do you want the WHEJAC to advise the White House Council on Environmental Quality to do?: Confront the FAA and force them to end the hyper-concentration of aircraft flight paths through RNAV. Force the FAA to disperse flights and the accompanying noise pollution. Force the FAA to use the N-above metric to judge the effects of noise pollution -- not their favorite, "DNL". DNL is useless as a noise metric in the face of endless repeated aircraft noise. Force the FAA to report N-above noise metrics, and require airport operators to disclose as public record deals and arrangements made concerning flight capacity with airline industry operators. Promote the substitution and improvement of less polluting alternatives, such as high speed trains. End the cycle of regulatory capture at the FAA. Stop letting former airline CEO's run the agency that is supposed to regulate them.

Full Name (First and Last): Marcia Munro
Name of Organization or Community: 10,000 Hawks
City and State: East Haven, Connecticut
Brief description about the concern: The new commercial jets at Tweed New Haven Airport (HVN), Boeing 737-700 - Avelo Airlines, are flying extremely low at departure and arrival. I live directly in the flight path on the shoreline South of Runway 02 for takeoff, and the noise and especially vibration Is intense as the planes go over the house. Arrivals also go over the property, continuing more to the East, but often are even lower in height. Not only does this cause sleep disturbance, but the vibration and low glide paths are very worrisome and stressful. We are surrounded by marshland and it is frequently foggy.

What do you want the WHEJAC to advise the White House Council on Environmental Quality to do?: Please investigate whether FAA rules are being followed. Feel free to contact me by telephone or e-mail (as above). I have other concerns re the proposed expansion of Tweed and would like to see more transparency and input from residents.

Southeast -2

West Virginia, Virginia, Kentucky, Tennessee, North Carolina, South Carolina, Georgia, Alabama, Mississippi, Arkansas, Louisiana, Florida

Dear President Biden, We the undersigned are elevating a matter in urgent need of your immediate attention and action. Many of us live in the Gulf coast region directly impacted by the 2010 BP Deepwater Horizon oil disaster and use of toxic chemical dispersants during spill response. Hazardous dispersants were staged in our public marinas and neighborhoods; they were sprayed in coastal seas where we swim and fish; they were used long after the "official" end date in July 2010. Hazardous oil spill waste was disposed of in several of our municipal landfills. Now our families, friends, and neighbors suffer with long-term diseases, disabling chemical illnesses, cancers, and early deaths linked with toxic exposures from this oil spill, and childhood cancer rates have soared in our coastal communities. Many of us have participated in the long-term studies that found that dispersants make oil more toxic. Local wildlife is also experiencing long-term health harm from this oil disaster. Yet dispersant use continues even now. We are joined by others who share our concerns, having experienced oil spills and dispersant use in other areas. We all pray for relief from these toxic chemicals. We have sought relief from both the EPA and the courts, and we have played by the rules only to be frustrated by industry pressures. Some of us filed a rulemaking petition with EPA on November 14, 2012, urging an NCP update to rules governing dispersant use. On January 22, 2015, EPA issued a Notice of Proposed Rulemaking to revise the NCP's Subpart J, the rules governing dispersant use. EPA stated the proposed rule was "anticipated to encourage the development of safer and more effective spill mitigating products, and would better target the use of these products to reduce the risks to human health and the environment." Sounded good, but for years, EPA took no action to finalize its proposed rule. After waiting through two federal administrations for a final rule, a group of environmental justice and tribal advocates and individuals, including some of us, sued EPA under the Clean Water Act (CWA) and the Administrative Procedures Act (APA) (*Earth Island Institute/ALERT Project et al. v. Andrew Wheeler et al.* 3:20-cv-00670-WHO). We were elated when, on August 9, 2021, the federal district court ruled in our favor on both claims! Indeed, the court clarified an important part of the CWA, finding that "EPA has a nondiscretionary duty to revise or amend the NCP ***when there is new information that shows that the current standards for efficient, coordinated, and effective action to minimize damage from oil and hazardous substance pollution are insufficient to safely provide for mitigation of any pollution***." See Order at 8 (p. 6, lines 9–12), interpreting 33 U.S.C. §1321(d)(2). And the court agreed that waiting through now three administrations and eight years "was unreasonable and compelling agency action." (p. 16, lines 12–13). The court imposed a deadline of May 31, 2023, for EPA to issue a final rule, under court supervision, in its 2015 proposed rulemaking. Despite our victory, we have several reasons to remain concerned that EPA will continue to ignore critical new information in this rulemaking. On July 27, 2021, just prior to the court decision in August, the EPA signaled its intent to allow dispersant use in future spills when it issued a final rule, based on the 2015 Proposed Rule, to require monitoring of dispersant use in certain situations. Yes, that's right, it did. It's almost like EPA didn't really hear the court since this final rule does not include the current science from May 2015 to present. Given this action, we now also fear that EPA will ignore this science in its final rule in May 2023. This latest science contains the bulk of a growing number of independent studies that show deadly harm to human health and the environment from dispersant use during oil spill response under the NCP. ***This is a solid block of new information that clearly meets the federal court's recent interpretation of the CWA and compels EPA to update the NCP accordingly.*** Mr. President, we have done everything we can to hold EPA accountable to the spirit and intent of the laws that are designed to protect the waters of the U.S. and our health and wellbeing. But we feel a higher hand is now needed to compel EPA to do the right thing. This is why we now ask *you*, the President of the United States, to take two specific and immediate actions: 1) order EPA to withdraw its final rule issued on July 27, 2021, before it goes into effect on January 24, 2022; and 2) order EPA to issue one comprehensive rule in its 2015 proposed rulemaking, on or before May 31, 2023, based on the latest science, i.e., to present. (More support for each of these requests is provided in Appendix A and B.) Mr. President, we have paid the ultimate price for short-sighted, industry-driven public policy. Yet we are willing to wait until May 31, 2023, for one comprehensive rule that is revised to reflect the emerging proof of deadly harm to people and sealife from dispersant use. It is time to recognize that oil dispersants are a net environmental *loss*, not benefit. We believe that only you can make this happen. Please hear us. Sincerely, Gulf Coast advocates and allies

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Full Name (First and Last): Michelle Rutledge
Name of Organization or Community: St. Peter and Pinesville neighborhoods of Archer, FL
City and State: Archer, FL
Brief description about the concern: My concern is the siting of industrial facilities (including renewable power plants) in historically Black and rural residential / agricultural neighborhoods. My neighbors and I are community members of the Saint Peter Saint Paul Community Council in Archer, Florida. Our rural, historically African American neighborhood intact since slavery successfully fought an industrial solar facility sited in a residential and agricultural zone. However, the threat remains. Some background, Archer is a small town with one traffic light. Yet, the small town of Archer, FL has experienced two (2) large utility-scale solar proposals in size of 600+ acres recently in historically Black rural residential and farming neighborhoods. As we advocate for a Just Transition to renewable energy, I feel our small rural town is an early warning on how renewable energy can be unjust as well in regard to land use/zoning/siting.

What do you want the WHEJAC to advise the White House Council on Environmental Quality to do?:
I would like to ask the establishment of siting / land use / and zoning guidelines which will protect the culture, wealth accumulation, heritage, and mental health of historically Black and rural residential and agricultural communities (i.e., no siting in residential neighborhoods). In addition, as there is a transition to the use of renewable energy, I would like to request there be a just, fair and equitable process which includes community engagement, benefits, historical lookback and reparations for current and past harms to Black property owners, increase of programs (i.e., grants and or tax incentives) for energy efficiency and weatherization of homes in minority communities, and development of pathway programs to increase full-time job opportunities in the renewable energy sector for minorities, etc. Thank you for your time and consideration.

Full Name (First and Last): JACQUELINE Mary JONES
Name of Organization or Community: Reidsville Georgia Community Floods
Brief description about the concern: Educating citizens to what flooding is and how hazard it is to home and health

What do you want the WHEJAC to advise the White House Council on Environmental Quality to do?:

My name is Jacqueline Jones and I am the leader of a group called Reidsville Georgia community floods, zip code 30453. My group is a member of Anthropocene alliance, a national nonprofit that educates and organizes individuals and communities harmed by environmental abuse and climate change Reidsville Georgia is an impoverished community and the people in this area are basically closed off from the rest of society. The nearest college is about an hour away and many do not have any means of transportation to get to the grocery store, let alone to get an education. So if you asked most people in this area, they will not know what environmental justice is What is environmental justice? These words can mean many things to many different people. Environmental justice is desperately needed in this area even though because of generations of oppression, people have normalized the erosion, the devastation, the mold the sewage, the nuisance and inconvenience of flooding. Shockingly people in this area do not look upon the water that stands for months at a time on their properties as flooding, drains being 50 years old or there not being a drain whatsoever, or being incapable of getting in or out of their homes as being anything but normal because they have lived with it for decades. I moved to this area four years ago and immediately experienced flooding with almost 3 ft of water up to my window sills that sat in my yard for months with nowhere to go! In my opinion it's not just the fact that many homes were allowed to be built in this area on top of swamps, it's not only the fact that the city and county officials refuse to do anything including applying for grants that would help resolve the situation, it is the mentality of the citizens of this area. Although I am not in the best of health I have passed out flyers to close to 300 people so far and only one person has contacted me for help. There needs to be a federal outreach program to inform people that yes there is help and assistance available. This could be accomplished through direct mailing using the us postal service Reidsville Georgia in desperate and serious need of environmental justice through educating citizens of the hazards and health issues that are caused by flooding. Thank you for your time

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I am the Executive Director of Little Growers Inc an Environmental and Food Justice Organization in Brevard County Florida where poor communities of color are inundatede with corporate pollution, environmental racism, and rampant food insecurity. Comment: We already know that communities of color are disproportionately impacted by environmental racism, and injustice. Yet resources, specifically funding that is meant for what is now labeled BIPOC(black indigenous people of color) and Latinx communities is never actually getting into the hands of grassroots leaders who are on the frontlines of this work. Often because white led orgs hijack our narratives' to access resources that are specifically meant to help grow our work but that never happens! Dr. Bullard spoke to this in the meeting held 01/26/2022 and he spoke truth to power, but still skilled educated black and indigenous grassroots leaders who are on the ground with our feet in the mud of this work are being ignored because of this systematic effort to continue to fund the same white led orgs that steal our NARRITATIVE and leave with money that was meant to support those most impacted. Its comparable to how during slave times the slave would birth, raise, clean and cook the hog but the slave would only get the scraps like pig intestines basically the shit, and then be expected to thank the master for the scraps. Like this community leaders like myself who build green infrastructure, and design other actionable solutions that are well formulated and supported never get the resources we need to push or work forward always the shit always the crumbs but still we press on sustaining our work without any of the resources we need. We are exhausted, overwhelmed, and severely undervalued but still will press on. This systematic effort to keep resources out of the hands of the ones who are actually doing the work HAS TO STOP! Advocacy for communities should not look like government or others who have not lived our experience telling us how they think we should solve our problems. We are capable of solving our own problems if we stop being locked out of access to resources needed. Thank you Camille T. Hadley

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Full Name (First and Last): Kent Minault
Name of Organization or Community: Sierra Club's Harvey Broome Group in Knoxville TN
City and State: TN
Brief description about the concern: Coal ash impoundments near TVA coal plants in Tennessee

What do you want the WHEJAC to advise the White House Council on Environmental Quality to do?:
Here in Knoxville, TN, the Sierra Club is working with environmental justice communities near coal ash impoundments - particularly near TVA's Bull Run Coal Plant. This impoundment in Claxton is a mountain of coal ash right next to a soccer field and playground. The main pollution from the coal ash is from seepage into groundwater. Most impoundments in our state sit below the groundwater level. TVA and local water testing seem to indicate that drinking water in the area is safe. But heavy metal toxins in the leachate tend to settle on the river bottom where they don't show up in the tests. Local residents have noticed a rash of dead ducks by the Clinch River that don't seem to have come to the attention of TVA. Ducks are well known to eat river bottom mud. Reports show methyl mercury, a toxin derived from coal ash, moves up the food chain from the bottom mud and ends up in the tissue of fish that are often eaten far away. Local testing doesn't really assess the environmental harm. Many local residents were first responders to clean up the Kingston coal ash spill of 2008 20 miles to the west. Over 50 of these workers have died in the intervening years with symptoms characteristic of coal ash toxicity. Local citizens want the coal ash in their community moved to impoundments that are high, dry, lined and capped. They want coal ash officially declared a hazardous substance, which it certainly is.
I have two questions for the Council: first, what agencies can citizens access to get coal ash out of their communities and get support for their health care needs in dealing with the effects of toxicity.
Second: how can citizens get federal help to bypass TVA's blockage and get real help to get the blight of coal ash out of their communities?

Full Name (First and Last): Tina Smusz
Name of Organization or Community: Preserve Montgomery County Virginia PMCVA
City and State: Catawba, Virginia
Brief description about the concern: Lack of acknowledgement by state agency staff that a linear construction project like the Mountain Valley Pipeline is rife with Environmental Justice Issues. DEQ staff's erroneous denial of EJ issues connected with projects can sway the decisions of citizen boards toward certifying harmful projects for Virginia communities

What do you want the WHEJAC to advise the White House Council on Environmental Quality to do?:
Offer MANDATORY training programs in Environmental Justice for all state depts of environmental quality (or protection) - titles for these agencies vary by state.

Full Name (First and Last): Tiffany Grantham
Name of Organization or Community: Florida Rights of Nature Network
City and State: Hollywood, FL
Brief description about the concern: In western Palm Beach County Florida the areas where sugar cane is grown there are multiple concerns with air, water and even soil quality. When the sugar cane is burning and the winds are blowing the smoke over brown and black neighborhoods, it's ok. If the wind is blowing toward the affluent east it is not allowed. When the fields and waters are being sprayed with pesticides there is also no certain for the folks who live, fish and breathe in the surrounding neighborhoods. And with the burning of the fabulous muck tremendous amounts of carbon that have

been sequestered for years goes straight into our atmosphere. The asthma and cancer rates prove these issues.

What do you want the WHEJAC to advise the White House Council on Environmental Quality to do?: Ask Nikki Fried our Ag Commissioner to stop the burn and go with worldwide favored green harvesting and figure out a better way with the spraying of algicides and pesticides. This is a most blatant and horrific act of Environmental injustice especially when compared to the other half of the county.

I am Linda Parsons, a private citizen from rural Montgomery County, Virginia. "Thank you for the opportunity to speak to the White House Environmental Justice Advisory Council today. My story involves my fight against Mountain Valley Pipeline, the Federal Energy Regulatory Commission, Cardno, the Virginia Department of Environmental Quality, and the Virginia Department of Conservation and Recreation. I made appeals to all of these groups without success. They have in turn ignored my issues and denied my claims. Until 2018 my family's water source for 3 houses was from a perpetual spring that had produced clean, clear water since before my grandfather's time. He bought the farm in 1944 and subsequently operated a Grade A dairy, using the water from the spring. My family's farm also has another spring on the property nearer to the pipeline's path which also produces clear water used for livestock. When I learned of the pipeline's proposed path over nearby Johnson Ridge in 2014, I was alarmed. Dye tracing done in the 1970s showed our springs' water sources originate under Johnson Ridge. Furthermore I knew Johnson Ridge was not like normal terrain since it is full of karst features like sinkholes, swallets and caves that can direct rainwater into the underground waters. I sent FERC documents and letters pointing out the danger to my springs. I participated in public comment periods. I was ignored by both FERC and Virginia DEQ which are agencies set up to protect our water resources. We were told the pipeline trench was only 10 feet deep and would not affect my springs. Then after the blasting on Johnson Ridge commenced in May of 2018, the problems started with the very first rain. Muddy sediment boiled up in the waters to the spring closest to the pipeline. MVP denied they were the cause. Then after 3 months MVP found a swallet opening in the uncovered pipeline trench itself. They dye-traced this swallet opening to our spring. MVP immediately mitigated the swallet so they could install the pipeline. We were told the mitigation would fix the problem. Yet, more than three and a half years later, the sediment contamination continues. Our spring has been visibly harmed with decreasing water flow and sediment coatings over the aquatic plants. Not only am I incensed by the destruction of our spring with sediment contamination, I am fearful that this sedimentation is evidence of hidden erosion around the pipeline. Erosion can destabilize the pipe which in turn could result in a devastating explosion. There are 6 houses close to the swallet and I fear for the safety of my neighbors. I am in the process of writing Senator Tim Kaine, a letter that outlines my belief with backup documentation questioning MVP's mitigation of the swallet. I have just presented two examples of what I see as environmental injustice. I hope the White House Environmental Justice Council can channel their power to turn around these examples into environmental justice. Linda Parsons Sink - Blacksburg, VA 24060

Full Name (First and Last): Mary Tatigian
Name of Organization or Community: Quiet Florida
City and State: Naples
Brief description about the concern: We live in Naples FL and have been inundated with low, loud frequent aircraft this last year. The noise is making us sick; I cannot imagine what the particles being dropped from fuel exhaust down upon us is doing. This should be illegal as it is harming human beings,

affecting their health.   We also are listening to traffic noise/modified mufflers, inside our homes, while we are in bed. This is insanity, where is the respect for our Quality of Life? We Matter Too.
STOP NOISE POLLUTION

What do you want the WHEJAC to advise the White House Council on Environmental Quality to do?: STOP NOISE POLLUTION There is much research that has been done and suggestions given by Noise Org to quiet the noise, choose any and act upon this. The FAA needs to be reigned in, too many flights in the sky, do you have any idea that millions of people are affected negatively by these flights? Thank you.

Full Name (First and Last): Jerry Leonard
Name of Organization or Community: Melrose Place
City and State: Baton Rouge, LA
Brief description about the concern: Our neighborhood, Melrose Place, in Baton Rouge, Louisiana, has been having extreme diffuculties with a company on the close backside of us that is a nuisance. We've been forced to deal with several noise related changes that they've made that have a noise induced affect on the community that requires therapy & medication. We've also been dealing with a state regulated department building that has loud equipment that affects the quality of life in the community. Memebers of Quiet Communities, Inc. in Lincoln, Massachusetts, have been helping us in any way possible to eliminate the issues.

What do you want the WHEJAC to advise the White House Council on Environmental Quality to do?: Please take the proper steps to ensure that harmful noises & their effects on the human body are taken into serious consideration & develop grant programs for harmful noise elimination.

Full Name (First and Last): Christina Cummings
Name of Organization or Community: Partnership for Southern Equity, Inc.
City and State: Atlanta
Brief description about the concern: The public comments that we offer today are a summary of our learnings as we designed and launched the Justice40 Accelerator. Our goal is to help ready frontline communities to prepare for these investments and navigate the complexities of applying for federal, state, and local funding. Lastly, we have posed questions regarding the efficacy of the WHEJAC and seek to better understand the accountability structure of the body.

What do you want the WHEJAC to advise the White House Council on Environmental Quality to do?: We would like for the Council to respond to the various questions we proposed below: 1.How will WHEJAC be different and more effective than NEJAC- the National Environmental Justice Advisory Council established in 1993 by President Clinton? Erecting prestigious bodies to confer on the injustices bestowed upon frontline communities has proven to be more ceremonious than effective when it omes to getting the environmental justice communities deserve. 2.To what extent will the WHEJAC develop accountability metrics and transparent communications to the community of the progress and activities of the body? 3. How will WHEJAC ensure that the funds being deployed from the Bipartisan Infrastructure Bill adhere to President Biden's Justice40 Initiative? Recognizing that oversight and compliance at the federal level has traditionally excluded historically marginalized EJ communities. 4.What will the WHEJAC do to ensure that the impact of the body stands as more than another vestigial consortium whose outputs culminate in more than a report? 5. What role does the WHEJAC play in supporting the Biden Administration to implement the 10-12-30 provision? 6.How can WHEJAC ensure the Department of Energy actively participates in a whole-of-government response to work with every single Rural Electric Cooperative in the country to chart a technology transition path?

Full Name (First and Last): Virginia Richard
Name of Organization or Community: SouthWings
City and State: New Orleans, LA
Brief description about the concern: For over two decades, SouthWings has been working with frontline communities and grassroots organizations to expose the threats to ecosystems and human health posed by extractive industries and now, false climate solutions -- including CAFOS, mountaintop removal mining, coal burning power plants, coal ash ponds, deforestation and processing for biomass, offshore drilling, the breakneck speed at which liquid natural gas facilities are being built, and more. CAFOS- Confined animal feeding operations produce an enormous amount of animal waste that contributes to water and air pollution. Hog waste at a CAFO is usually stored in a pit of water or a "lagoon." This waste often leaches into groundwater and during flood events can spill into nearby surface waters. The liquid waste is also sprayed on fields, allowing it to become airborne, and polluting neighboring properties. CAFOs are disproportionately located in low-income communities and communities of color. Wood pellets/Biomass- There has been a significant increase in demand for woody biomass as an energy source in Europe and other parts of the world. Burning wood pellets for energy has falsely been encouraged as a climate change solution. However, burning wood pellets produces more carbon dioxide per unit of energy produced than coal AND cutting forests to produce wood pellets releases carbon dioxide and reduces carbon sequestration abilities. The industry claimed that only scrap from timber harvest would be used for wood pellet production. However, SouthWings' partners have collected evidence that natural forests are being clear cut to supply wood pellet mills. This destroys habitat, impacts biodiversity, and harms water quality. In addition, the vast majority of wood pellet plants are located or proposed in rural, low-income, communities of color. Coal- From extraction, to combustion, to disposal, burning coal is destructive to nearby communities and the environment, and accelerates climate change. Pipelines- Pipelines built to transport oil and liquified natural gas cause widespread harm to the environment and communities. It is not a question of if a pipeline will leak and/or explode, but a question of when and where. Pipelines are often routed through low-income and communities of color, as are compression stations. They are neglected in favor of building new pipelines and new construction often flouts regulatory requirements with creative permitting and lack of enforcement. LNG- Liquid natural gas is being touted as a cleaner, greener fuel that is environmentally friendly, despite the fact that it is a fossil fuel and its production and use contributes a significant amount of carbon to the atmosphere. This is nothing more than greenwashing. Even more concerning is the break-neck speed at which LNG infrastructure is being built in areas already on the front lines of the climate crisis. Southwest Louisiana has endured many climate events back to back over the past two years that have not only impacted people, but also made it very clear that building fossil fuel infrastructure in such a vulnerable and over-polluted area is unwise. Not only do the frequency and strength of storms and rising seas threaten infrastructure, they also mean more pollution events for residents who are already struggling. Plastics-The world is already overwhelmed with plastics. Plastics never fully disintegrate; they just get smaller and smaller. We are only beginning to learn the breadth of plastics' carcinogenic nature, and already know that microplastics harbor toxins and bacteria, especially in warmer waters. Plastics have made their way into every part of our food chain and into our bodies, which will have untold effects for generations to come. Our oceans are glutted with them and they are disrupting life on earth as we know it. However, communities like St. James Parish, Louisiana, which is part of Cancer Alley, are targets for plastic production sites like the proposed Formosa mega-complex. Not only does plastics production and consumption harm our communities, but emissions from the production of raw and finished materials drive climate chaos.

What do you want the WHEJAC to advise the White House Council on Environmental Quality to do?: It's long past time that we stopped subsidizing fossil fuels and false climate solutions. Instead, we must invest in real, just solutions to climate change. We have less than 10 years to get it done. Fortunately, we already have all we need to achieve this; we just need the funding and the willpower to do it. That includes following the leadership of BIPOC and oppressed communities. It also means respecting the sovereignty of Indigenous communities, regardless of federal recognition status. Environmental justice means more than mitigation and resilience. It means stopping climate change now, and doing so in the most equitable way possible. And so this is simply the essence of our comment: SouthWings urges WHEJAC and the Biden administration to take decisive action now. Stop extractive practices in all sectors: fossil fuels, forest, and human life. Stop the production of harmful materials. Stop the false climate solutions which only exist to prop up industry while the world burns. We have no time to waste.

Midwest -3

Ohio, Indiana, Michigan, Illinois, Missouri, Wisconsin, Minnesota, Iowa, Kansas, Nebraska, South Dakota, North Dakota

Full Name (First and Last): Dr. Jim Van Keuren
Name of Organization or Community: Ashland University, Ashland,
City and State: BROOK PARK, Ohio
Brief description about the concern: The Presumptive List of Conditions for those exposed to Toxins at Superfund site is ambiguous and unclear. For those to toxins the litigious road is an expensive time-consuming process and often leaves the plaintiffs with large debt and no solutions.

What do you want the WHEJAC to advise the White House Council on Environmental Quality to do?: Recommend that that WHEJAC create a study group to examine a Presumptive List of Conditions for those exposed to toxins at Superfund sites. THIS IS THE MISSING PIECE TO SUPERFUND CLEANUPS.

Full Name (First and Last): Pat Van Keuren
Name of Organization or Community: Ashland University, Ashland,
City and State: BROOK PARK, Ohio
Brief description about the concern: This is an expanded version of what I sent on your public comment section for the January 22nd meeting. I wrote a book entitled The School Poisoning Tragedy in Caledonia, Ohio, and from that work I found a missing piece in the Superfund site cleanups which is that a Presumptive List of Conditions for those exposed to toxins at Superfund sites is ambiguous and unclear. The following is one avenue of addressing a missing piece of Superfund site cleanups **Assumption:** 1. The Presumptive List of Conditions for those exposed to toxins at Superfund sites is ambiguous and unclear. **Known Facts:** 1. The detailed data on the population within 1 and 3 miles of Superfund sites makes up approximately 22% of the total population in the United States. * 2. The most common carcinogenic to humans frequently found at Superfund sites are **lead** (43%), **Trichlorethylene** (42%), **Chromium** (35%). **Benzene** (34%), **Perchloroethylene** (28%), and **Arsenic** (28%) leading to different forms of cancer. * 3. The International Agency for Research for Cancer for Toxic Substances and Disease Registry Substance Priority List ranking for carcinogenic to humans at Superfund Sites is

Lead (2), **Trichloroethylene** (16), **Chromium** (17), **Benzene** (6), **Arsenic** (1) and **Perchloroethylene** (probably carcinogenic to humans). ** 4. The carcinogenic to humans by organs found at Superfund sites are blood disease (Leukemia Lymphoma), brain, bladder, colon, kidney, immune system, liver, lung, nervous system and skin cancers. ** **Problems: 1.** The barrier of meeting a Presumptive List of Conditions has been the documentation of an environmental exposure which is difficult to prove because the requirements focus on specific time, place and level of exposure. **2.** The litigious road is an expensive time-consuming process and that often leaves the plaintiffs with large debt and no solutions.

What do you want the WHEJAC to advise the White House Council on Environmental Quality to do?: Create a study group to examine a Presumptive List of Conditions for those individuals who were exposed to toxins at Superfund sites. **Possible Study Group Members:** 1. Identify legislative aides with appropriate background of the issue being discussed where consideration might be given to those who worked with H.R. 2372 and the Senate's *Comprehensive and Overdue Support for Troops (COST) of War Act of 2021.* 2. Representative from the EPA who works with Superfund sites. 3. Representative from the International Agency for Research for Cancer. 4. Representative from the VA who has knowledge of the Presumptive List of Conditions used with veterans. 5. Epidemiologist or like representative. 6. Individuals who have been exposed to toxic waste, resulting in cancer or some other disease with a prime source being from book from my book *The School Poisoning Tragedy in Caledonia, Ohio* and possibly from the Diamond Shamrock contamination in Painesville, Ohio. 7. Representative from some other outside resource groups. **Recommended Study Group Work:** 1. Meetings would be held virtually which would eliminate travel time and provide for more flexibility in convening meetings. 2. Meeting sequence can be altered based on data gathered, plus some of the meetings might be done via a consensus vote without holding a meeting. First meeting would be introductions and sharing of background information with identification reading material such as *A Spatial Study of the Location of Superfund Sites and Associated Cancer Risks by* Arlene Nelson and Shannon McDougall (1 hour). Second meeting would be the discussion on the type of surveys that may be needed, plus what kind public outreach might be done during the study group work. Identify existing surveys and assign staff to develop surveys, plus have study group members and/or staff develop a public outreach program if so desired. Third meeting would be to examine surveys and public outreach programs before release with surveys results being integrated throughout the study group work (1 ½ hours). Fourth meeting would be the discussion of Presumptive List of Conditions and the latency period impact that it may have on future illnesses (1 hour). Fifth meeting would be to bring more clarity to the meaning of Presumptive List of Conditions with examples of toxins impacting those diseases (1 hour). Sixth meeting would be to identify existing remedies available to veterans and non-veterans who been exposed to toxics resulting in cancers and other diseases (1 hour). Seventh meeting would be to recommend 4 or 5 Presumptive List of Conditions and request a cost analysis of those conditions (1 ½ hours). Eighth meeting would be to examine the cost analysis options and possibly ask for more data for clarifications purposes (1 to 2 hours). Ninth meeting would be to identify existing and/or potential revenue streams (1 hour). Tenth meeting would be to identify federal funds that could be redirected to incrementally fund this program. This could take several meetings. Eleventh meeting would be a wrap-up session with discussion of whether or not there is an agreement on a **Presumptive List of Conditions** and make a recommendation that the study group work be moved to the next level (1 to 2 hours).

Full Name (First and Last): Lisa DeVille
Name of Organization or Community: Ft. Berthold Protectors of Water Earth Rights
City and State: Mandaree, ND

Brief description about the concern: Environmental impacts from oil and gas.

What do you want the WHEJAC to advise the White House Council on Environmental Quality to do?: Need help creating laws, regulations, and studies. There was no EIS completed prior to oil and gas extraction on Ft. Berthold.

Full Name (First and Last): SUSAN Liley
Name of Organization or Community: Citizens Committee For Flood Relief
City and State: De Soto,MO
Brief description about the concern: De Soto MO flash floods. EpA comes on digs yards up and leaves contaminated foundations and home. It costs the same if the would buy repetative loss homes.

What do you want the WHEJAC to advise the White House Council on Environmental Quality to do?: Buy these Repetative loss home out that keep flooding. Stop just fixing the yards.

Full Name (First and Last): Clifford Stuehmer
Name of Organization or Community: Concerned citizen
City and State: Port Hope, Michigan, 48468
Brief description about the concern: The Air Force and Air National Guard continue to push for F-35 basing at civilian airports (Burlington, VT, Madison, WI, Selfridge ANGB in Michigan). The areas around the airports are generally low income/people of color and bear the brunt of the noise pollution. The Air Force's own data shows that thousands of people living near the airports will be subjected to noise levels "incompatible with residential living", 65dB DNL. This standard was set in the 1970s and subsequent research has shown this to be too high, notwithstanding the 115dBA ear damaging noise generated by the F-35s.

What do you want the WHEJAC to advise the White House Council on Environmental Quality to do?: Basing F-16s and F-35s at civilian airports and populated areas is not acceptable and should be stopped as this is clearly a public health and environmental justice issue. The EPA should also examine and update the 65dB DNL standard to 21st century science in order to protect public health.

Dear Staff, It is an honor to bring my thoughts to the table for environmental justice. I am an college educated engineer and military veteran. I have seen many types of chemical operations in industry and the management of safety in and around chemicals. I think we need to look at the Fentanyl crisis from a different perspective to be effective at managing this issue. We need to prioritize the environmental impact and environmental justice that Fentanyl is causing within our entire biosphere. Fentanyl is an extremely dangerous chemical and should be handled with caution. It should be classified as Hazardous Waste when not used for medical purposes because of its known physiological effects both short term and long term. When we allow this chemical to cross our borders we allow environmental hazardous waste to cross our borders and we fail the intent of environmental justice. I think it is only a matter of time before a terrorist uses this chemical as a weapon like any other hazardous chemical can be used. Here are some of the biological effects of Fentanyl from the Pfizer Material Data Sheet: Section 2:
May be absorbed through the skin and cause systemic effects. May be fatal if swallowed (based on components) . Use of this drug is habit forming. Addiction may occur. Animal studies have shown a potential to cause adverse effects on the fetus. Animal studies indicate that this material may cause adverse effects on the reproductive system. Ingestion of this material may cause effects similar to those seen in clinical use including dry mouth, drowsiness, headache, dizziness, nausea, vomiting, weakness,

anxiety, and dilated pupils. Cases of severe overdose may lead to respiratory depression, hypotension, coma, convulsions, cardiac arrhythmia, and tachycardia. The EPA has guidelines for handling Fentanyl pharmaceutical waste. Within these guidelines it is treated as hazardous waste. Management Standards for Hazardous Waste Pharmaceuticals and Amendment to the P075 Listing for Nicotine Management Standards for Hazardous Waste Pharmaceuticals and Amendment t... Some pharmaceuticals are regulated as hazardous waste under the Resource Conservation and Recovery Act (RCRA) wh...Lets standup for environmental justice and fix this Fentanyl issue at our borders with more assertive international and national environmental laws like we are doing with lead in our water, clean air to breath, and the protection of our environment as a whole system, not just picking political "cherries" to satisfy a special interest group. respectfully, Robert Gustason Woodbury, MN

Full Name (First and Last): Julie Geisinger
Name of Organization or Community: Sierra Club
City and State: Birmingham, MI
Brief description about the concern: I am writing you about the Line 5 oil pipeline -- specifically the section that runs under the Straits of Mackinac. This nearly 70-year-old pipeline is a disaster waiting to happen. The pipeline was never intended to be in service so long. What's more frightening is that Enbridge, the owner and operator, does not have a good safety record and the line has spilled at least 33 times. A spill in the Straits, a vital waterway that connects the Great Lakes, would be absolutely devastating for Michigan's economy, our drinking water, wildlife, and the native tribes that have hunting and fishing rights in this area. Enbridge is now trying to secure permits to move forward with a tunnel to replace the section under the Straits, but that is not the answer. Building new infrastructure for fossil fuel companies (especially foreign companies) does not make sense. Since Biden announced his plans of moving forward with a clean energy future and decreasing greenhouse gases to combat climate change, building new fossil fuel infrastructure continues to contribute to the problem.

What do you want the WHEJAC to advise the White House Council on Environmental Quality to do?: -Urge President Biden to revoke the presidential permit for Line 5 -Ensure Michigan's tribal governments have a seat at the table in any treaty talks with Canada. -Follow through climate commitments by shutting down Line 5.

Full Name (First and Last): Erik Hatlestad
Name of Organization or Community: Rural Power Coalition
City and State: New London, MN
Brief description about the concern: Rural Electric Cooperatives (RECs) serve 42 million Americans and power 56% of the nation's landmass. RECs cover 90% of counties federally-recognized for persistent poverty, yet the member-owners of those utilities face some of the highest energy burdens—percentage of income spent on energy bills—in the country. These counties include broad swaths of the Black Belt region, Appalachia, Tribal Lands, and the US border, where many economically-disadvantaged Americans live. For this reason, it is essential that the path to a lower carbon future does not drive electric cooperatives, or the communities that own them, further into debt. RECs directly own over 300 fossil fuel power plants in the United States, emitting over 130 million metric tons (mmt) of carbon pollution annually. Though they serve just over 12% of the U.S. population, RECs account for over 20% of all electrically-generated carbon emissions, with nine of the most carbon-intensive electric utilities in the US energy sector being owned by RECs. To meet the necessary goal of creating a carbon pollution-free power sector by 2035, the REC sector must retire these plants and replace them with carbon-free energy sources—and they must do so in just 14 years. Without federal relief, the only way that RECs can

finance clean energy investments is via new debt. Unfortunately, the REC sector already holds roughly $100 billion in debt, nearly half of which is financed directly by the federal government. To put this into context: East KY Power Cooperative owes the Rural Utility Service (RUS) more than $2.1 billion for energy infrastructure that needs to be retired. They carry daily debt service of $563,000. That's an average of $29/month in debt service for every meter served. Electric cooperatives and member-owners have been at a historic disadvantage. Even in the face of insufficient energy infrastructure and devastating environmental impacts (like mountaintop removal coal mines and air and water pollution from coal plants) rural communities were last to get electricity to their homes. RECs have also been historically disadvantaged by federal incentives for clean energy, while their investor-owned counterparts have been able to leverage robust state and federal tax credits for proactive investments. Without federal funding, these communities will be the last to transition to clean energy, or will be unable to decide their own clean energy futures, while outside companies buy up rural land for large-scale solar projects, lining the pockets of out-of-state developers. Rural communities require an equitable and just energy transition that promotes local ownership and local wealth building.
Federal investments in rural America have been proven to be effective and necessary. The Rural Electrification Act of 1936 brought electrification to the countryside. As a result of this renowned New Deal program, the electrification of rural areas (including homes and businesses) increased from 10% to 80% between 1936 and 1950. Unfortunately, the economics and social conditions have changed relatively little since the first power lines went up—some have even gotten worse. Today, faced with the economic and climate imperative to transition to a new energy system, rural communities will once again require major federal investment.

What do you want the WHEJAC to advise the White House Council on Environmental Quality to do?: The Rural Power Coalition (RPC) represents the national movement for electric cooperative reform that is being led by member-owners and advocacy groups across the country. Specifically, the RPC is asking the White House Environmental Justice Advisory Council to recommend the Biden Administration: - Ensure any debt relief to Rural Electric Cooperatives is conditional on new investment in local energy upgrades in rural communities on terms that are broadly inclusive. (It's imperative that the benefits of federal support reach rural communities!) - Ensure that Justice 40 recognizes and includes in its definition the counties that are already federally recognized for persistent poverty. These are counties that have had more than 20% of the population living below the federal poverty line for at least three census reports, or 30 years. - Ensure that President Biden fulfills his pledge as a candidate to implement the 10-20-30 rule. This is a policy championed by Rep. Clyburn and Sen. Booker to assure that at least 10% of federally funded programs be directed toward persistent poverty communities. - Ensure the Department of Energy actively participates in a whole-of-government response. The DOE has the capacity to work with every single Rural Electric Cooperative in the country to chart a technology transition path, but right now, it is helping very few. - Ensure meaningful engagement at a community level for every new application requesting federal financing from the Rural Utilities Service—and for every requested issuance of debt for the Tennessee Valley Authority. -Treat electric cooperatives fairly in reform of renewable energy tax credits so that they receive the same vital option for direct payment that is afforded to most other utilities. Sincerely, Rural Power Coalition 2/3/22 Appalachian Voices (NC) CURE (MN) Kentuckians for the Commonwealth (KY) Mountain Association (KY) Partnership for Southern Equity (GA) RENEW Missouri (MO) Western Organization of Resource Councils (MT)

Full Name (First and Last): Leatra J Harper
Name of Organization or Community: FreshWater Accountability Project
City and State: Bowling Green

Brief description about the concern: The Ohio River Valley has become a sacrifice zone for the fracking industry and its associated waste and infrastructure. We have been working diligently to stop the planned PTT Global petrochemical plant planned for the river in Dilles Bottom, Belmont County, Ohio. The region cannot tolerate the Shell cracker plant already coming on line this year - let alone another heavy toxic polluter.

What do you want the WHEJAC to advise the White House Council on Environmental Quality to do?: Take a very close look at what is being done to the Valley - especially because of fracking, its toxic, radioactive waste, and associated polluting infrastructure like compressor stations, fractionation plants, pipelines and petrochemical plants and a planned WV Methanol plant along the river. The Biden Administration needs to be alerted to this serious EJ issue in Appalachia and look at cumulative pollutants as well as the heavy health effects already experienced in the Valley!

Southwest -4

Texas, Oklahoma, New Mexico, Arizona

Full Name (First and Last): Rebecca Jim
Name of Organization or Community: Local Environmental Action Demanded Agency, Inc.
City and State: Vinita, OK
Brief description about the concern: People living near or downstream of superfund sites throughout the country want them cleaned up and in the meantime to be protected from the Toxic Flooding that can and does occur each time heavy rains displace contaminated materials and they move to harm those residing downstream, including entering drinking water reservoirs. More funding, more serious eyes on the most vulnerable.

What do you want the WHEJAC to advise the White House Council on Environmental Quality to do?: The CEQ has been manned in the past with passionate scientists who have come to the Tar Creek Superfund Site, listened and understood our issues. Use this time, use every ounce of influence to push for more to be done and done to protect the environment and the people who are most at risk at all the superfund sites throughout the nation as well as those sites longing to be placed with that status.

Full Name (First and Last): Alicyn Gitlin
Name of Organization or Community: Sierra Club - Grand Canyon Chapter
City and State: Flagstaff
Brief description about the concern: Thank you for taking our comments. My name is Alicyn Gitlin and I am representing Sierra Club's Grand Canyon (Arizona) Chapter. I am in Flagstaff, AZ, in the Grand Canyon region, the ancestral and current homeland of at least 14 Tribal nations. This region is the site of ongoing environmental injustices related to several stages of the nuclear fuel chain, from a legacy of abandoned mines on the Navajo Nation to the Pinyon Plain Mine (formerly known as Canyon Mine) that is extracting 10 million gallons per year of uranium and arsenic contaminated water from the Havasupai Tribe's watershed, despite never producing an ounce of uranium ore, to the White Mesa Mill which was just cited for carelessly polluting the air of the Ute Mountain Utes with radon while their outdated containment cells threaten the Ute's water supply. These Tribal nations bear the brunt of ongoing intergenerational health risks while foreign corporations play a careless money game. We were

therefore happy to see in the Advisory Council's Final Recommendations, plainly stated "Examples of the Types of Projects that will not benefit a community," number 4, "The procurement of nuclear power." We support the following from the Council's Final Recommendations:
"Permitting processes are key areas where Tribes must be involved as decision-makers, planners, and leaders. Regarding projects affecting public lands and other relevant lands, Tribal co-management must be an available option." (p. 53) "Tribes should be part of the earliest discussions about infrastructure development and design, which means as early as any other governing entity, such as states, are involved in such discussions." (p. 54) "Cultural impacts of infrastructure are critical to consider, whether for projects operated by Tribes or for projects that will affect Tribes. Tribal ecological knowledge has an important role in the design of projects and the assessment of risks. (p. 55) and "100% of investments must do no harm to Environmental Justice communities." (p. 57)

What do you want the WHEJAC to advise the White House Council on Environmental Quality to do?: This and all future administrations must operate on the principle that Tribes have a right to free, prior, and informed consent. Consultation is important, but we all know that consultation is not consent. Tribal nations have dealt with enough, and they deserve better. Green infrastructure must not be built by sacrificing the health of Indigenous peoples, destroying their most important religious sites, their water supplies, or their clean air. We are seeing too many examples of mining proposals being marketed as green while they lie within Traditional Cultural Properties and vitally important watersheds, threatening to contaminate and deplete the springs and drinking water supplies that affected communities depend upon. Nuclear power should not be part of any climate solution, and nothing should be part of a climate solution if it harms Environmental Justice communities. We can find a pathway to a healthy future, and Tribal nations and other affected communities should be at the forefront of recommending, designing, and benefitting from climate investments.

Full Name (First and Last): Sally Stueber
Name of Organization or Community: Moral ReSources
City and State: Harlingen, TX
Brief description about the concern: You reported during presentations Jan. 27 (WHEJAC) that among the major "achievements" so far, of Justice 40, (which was never fully explained, e.g. what 's the "40" about? how many agencies involved?) was to grant MILLIONS to the Army Corps of Engineers! Really?!!! ?? I was horrified to learn this, as an experienced environmental advocate, and closely familiar with recent changes in the Rio Grande Delta and the Kissimmee River catastrophe in Florida (due to Army Corps' misguided straightening and creation of a concrete channel, ignorantly assuming this would reduce flooding, which turned out to be a completely false assumption! Is this new JUSTICE 40 push to fund the Army Corps too generously, in my view, designed to RESHAPE their approach to hydrology and flood prevention? I hope so, since it is very clear their methods are NOT using the latest environmental science about the health and benefits of riverine habitats, role of trees in taking up water, etc. How did Army Corps get so much money? ARE their methods being scrutinized by qualified ecologists and hydrologists? Concrete only INCREASES flooding! Impervious cover is the worse cause of floods! This is basic information, well-verified, which was partly learned from terrible errors the Army Corps and others made.

What do you want the WHEJAC to advise the White House Council on Environmental Quality to do?: To scrutinize the METHODS of the Army Corps, and resist the straightening of rivers, and reducing, not increasing impervious cover! Across the board, indigent people here in the Rio Grande Valley suffer from flooding yearly now, and water is standing in and near the colonias, which often lack basic infrastructure, but are more easily harmed by floods from unethical owner/developers building in flood zones. Even when housing developments are raised to a higher level, the cities here are not planning for access by emergency vehicles to such areas, since the access roads will be completely full of water. Boats and ATVs would be needed for rescues in many of the newer housing developments. This is a key interface issue between proper ecological methods of flood management, and building codes, and who governs the Army Corps? They are NOT known to be a friend of the vulnerable EJ populations here or anywhere!

Full Name (First and Last): Joanie Steinhaus
Name of Organization or Community: Turtle Island Restoration Network
City and State: Galveston, Texas
Brief description about the concern: Communities of color have not received the same protection from the existing environmental laws meant to assure clean air, water and a livable environment. Protection should be distributed equally and not based on wealth, race or simply because the majority of the population does not live near these existing or proposed refineries, chemical and plastic plants.

What do you want the WHEJAC to advise the White House Council on Environmental Quality to do?: The EPA is outlining a series of broad policy actions and Turtle Island Restoration Network fully supports these initiatives to directly address the concerns of communities historically and disproportionately impacted by pollution. •Committing EPA to aggressively use its authority to conduct unannounced inspections of suspected non-compliant facilities, as needed to protect public health. When facilities are found to be non-compliant, EPA should use all available tools to hold them accountable. • Deploying a new program to expand air monitoring capacity, utilizing assets such as the ASPECT airplane, GMAP mobile air monitoring vehicle, and additional air pollution inspectors to enhance enforcement. •Mobilizing agency resources to invest in community air monitoring to better protect people and public health in vulnerable areas. •Pressing state and local elected officials to take urgent action to better protect the most overburdened communities. •Holding companies more accountable for their actions in overburdened communities with increased monitoring and oversight of polluting facilities. •Applying best available science to agency policy making to safeguard public health and protect the environment. We applauded the programs to improve communication, especially when it comes to information on environmental risk and enforcement, developing local strategies to address air toxins in communities, providing better transparency regarding enforcement, and improving access to risk management plans.

Full Name (First and Last): Cemelli de Aztlan
Name of Organization or Community: Centro Fronterizo del Obrero (dba) La Mujer Obrera
City and State: El Paso
Brief description about the concern: EPA finally designated El Paso County as an ozone nonattainment area in Nov. 2021. This is a major victory for air quality and environmental justice in our community, to hold polluting industries accountable. The EPA, Texas Commission on Environmental Quality (TCEQ), and others will be making decisions over the next year that will determine whether this victory is transformative in terms of reducing pollution in our community, or not. TCEQ has already stated, on record, that it disagrees with the EPA's designation, and they are seeking to reverse this victory and return to the status-quo. El Paso-Las Cruces is currently classified as a "marginal" nonattainment area. That means it is subject to the lowest level of regulation. In marginal areas, new industrial sources are required to install additional emission controls and purchase offsets, and infrastructure projects that receive federal funding must undergo additional environmental review, but there are no requirements for existing industrial sources. TCEQ is not required to develop a plan to reduce emissions from existing

industrial sources unless El Paso-Las Cruces is reclassified as a “moderate” nonattainment area. Under the Clean Air Act, an area is ordinarily reclassified as moderate if it fails to attain the ozone standard within 3 years. Though, the complication is that Section 179B of the Clean Air Act provides that an area is not reclassified from marginal to moderate if a state can show that it would attain the standard “but for emissions emanating from outside of the United States.” Section 179B was actually written specifically with El Paso in mind, which is the largest binational community in the world and has the largest concentration of maquiladoras along the border, a majority of them (63%) are US based Fortune 500 companies producing pollution and highly toxic waste. We ask that the EPA reject the 179b exemption, which is based on a dangerous and exploitive loophole. The reality is: US industries have been and are currently violating the ozone standard even if emissions from Mexico are excluded. Recent data modeling showed Mexico contributing about 7 parts per billion to ozone levels in our community, which is reporting an ozone value of 78 parts per billion. If you subtract the 7 parts per billion attributable to Mexico, you still have an ozone value of 71 ppb, which is above the standard. We ask the EPA to reclassify the El Paso/Las Cruces area from “marginal” to “moderate,” which would result in regulation of existing sources.

What do you want the WHEJAC to advise the White House Council on Environmental Quality to do?:
We needs EPA to have regulations and restrictions requiring accountability and ethics to protect the public health of our most vulnerable populations. Depending on TCEQ air monitors has proven to be faulty and detrimental to people's health.

Comment to WHEJAC:
This letter in support of NM Senate Memorial 27 encompasses some of the reasons New Mexicans keep fighting to remove gas and oil development away from Chaco Culture NHP and the surrounding communities. _________________________________Dear Honorable Members of the Senate, In 2017, Representative Lente worked with members across the Navajo Chapters around the Chaco Culture National Historical Park to craft a House Memorial to inform New Mexicans as to the immense significance of preserving the integrity of this ancient, scientific and indigenous place. It passed the House but was filibustered in the Senate. The significance of many of the bills being heard today are of short duration and quickly fade as money is spent and programs succeed or fail. The preservation of cultural history and places of scientific discovery of time, seasons, cycles of planting crops, and observation of the rotation and orbits of the earth and planets during the day and the night - this is everlasting and the essence of human survival. Please read this short Memorial of human impacts on the permanence of the cultural landscape long before there was a New Mexico, when people walked every mile, prayed to ancestors and rulers for guidance, and lived in a glorified center at Chaco until drought, famine and violence brought it to a close. These are the natural laws and lessons our generations must learn. Please hear the wisdom of this memorial and support it. Sincerely, Teresa Seamster, MS EdS Member, Counselor Health Impact Assessment Committee (2015-present)

SENATE MEMORIAL 27
55TH LEGISLATURE - STATE OF NEW MEXICO - SECOND SESSION, 2022
INTRODUCED BY
Benny Shendo, Jr. And Shannon D. Pinto and
Antoinette Sedillo Lopez
A MEMORIAL
AFFIRMING NEW MEXICO'S COMMITMENT TO ADDRESSING CULTURAL, ENVIRONMENTAL AND HEALTH IMPACTS IN THE GREATER CHACO CANYON LANDSCAPE.

WHEREAS, the greater Chaco canyon landscape, located in the San Juan basin in northwestern New Mexico, is an important ancestral landscape of the twenty pueblos of New Mexico and Texas and is an important ancestral landscape and current place of residence of the Navajo Nation; and
WHEREAS, portions of the greater Chaco canyon landscape are considered in the United States department of the interior bureau of land management's and bureau of Indian affairs' decision area for the Farmington Mancos-Gallup resource management plan amendment and its associated environmental impact statement and National Historic Preservation Act Section 106 process; and
WHEREAS, the Farmington Mancos-Gallup resource management plan amendment is being prepared due to changing oil and gas development patterns, including horizontal drilling technology and multistage hydraulic fracturing, resulting in more oil and gas wells and surface disturbances and impacts than were analyzed in the 2003 resource management plan; and
WHEREAS, one million eight hundred thousand acres of land have already been leased for oil and gas development within the Farmington Mancos-Gallup resource management plan amendment's decision area; and
WHEREAS, as of February 8, 2020, the draft Farmington Mancos-Gallup resource management plan amendment counts an existing twenty-seven thousand seventy-four vertical and horizontal wells, with an additional estimated two thousand two hundred wells projected over the life of the resource management plan amendment; and
WHEREAS, the potential impacts to the environment, human health, air quality, water quality and traditional cultural ways of life from oil and gas leasing and development are experienced by Indian nations, tribes and pueblos that interact with the greater Chaco canyon landscape by virtue of their residence, ancestry, customs, traditions and cultural practices; and
WHEREAS, the Navajo Nation chapter residents of Counselor, Torreon and Ojo Encino who reside in the greater Chaco canyon landscape and in the Farmington Mancos-Gallup resource management plan amendment's decision area have been impacted by ongoing health and environmental effects as well as cultural impacts caused by extensive oil and gas development; and
WHEREAS, the Counselor health impact assessment committee's July 20, 2021 report, titled A Cultural, Spiritual, and Health Impact Assessment of Oil Drilling Operations in the Navajo Nation area of Counselor, Torreon, and Ojo Encino Chapters, determined that significant increases in emissions from over four hundred oil and gas wells contributed significantly to respiratory health impacts of residents; and
WHEREAS, the all pueblo council of governors, representing the twenty governors of the sovereign nations of New Mexico and Texas, in comments provided on the draft Farmington Mancos-Gallup resource management plan amendment and its environmental impact statement, said that oil and gas development impacts on cultural resources and traditional practices directly impact the physical and mental health of pueblo members; and
WHEREAS, the New Mexico legislature has recognized and called for the protection and preservation of the greater Chaco canyon landscape's cultural sites and resources through House Memorial 70, titled "a memorial reaffirming New Mexico's commitment to protecting and preserving tribal, cultural and historical sites and resources in the greater Chaco canyon landscape", passed during the 2017 regular legislative session; and
WHEREAS, many federal laws, including the federal National Environmental Policy Act of 1969 and the federal Clean Air Act, are designed to require consideration of the environmental and health effects of federal actions; and
WHEREAS, Article 20, Section 21 of the constitution of New Mexico declares "protection of the state's healthful environment" to be of "fundamental importance to the public interest, health, safety and the general welfare"; and

WHEREAS, President Joe Biden's Executive Order 14008 formalized the president's and vice president's commitment to ensuring that all federal agencies develop programs, policies and activities to address the disproportionately high and adverse health, environmental, climate and other cumulative impacts on communities that are marginalized, underserved and overburdened by pollution; and
WHEREAS, the department of environment's environmental justice policy promotes fair treatment as meaning no group of people should bear a disproportionate share of the negative environmental consequences resulting from industrial, governmental or commercial operations or policies; and
WHEREAS, greater action is needed in New Mexico from the United States department of the interior to better regulate oil and gas leasing and development to ensure environmental and health impacts to New Mexico's Indian nations, tribes and pueblos are minimized in the greater Chaco canyon landscape;
NOW, THEREFORE, BE IT RESOLVED BY THE SENATE OF THE STATE OF NEW MEXICO that New Mexico's commitment to addressing cultural, environmental and health impacts in the greater Chaco canyon landscape be affirmed; and
BE IT FURTHER RESOLVED that the United States department of the interior's bureau of land management and the oil, gas and minerals division of the state land office be requested to comply with federal law by fully considering and addressing the effects of oil and gas development on the environment and health concerns of frontline communities described in the Farmington Mancos-Gallup resource management plan amendment and the accompanying environmental impact statement; and
BE IT FURTHER RESOLVED that the United States department of the interior bureau of land management and the oil, gas and minerals division of the state land office be requested to refrain from any oil and gas leasing, issuance of drilling permits or development without adequate environmental and human health protections in place that are created in consultation with potentially impacted communities, including the Navajo Nation chapters of Torreon, Counselor and Ojo Encino; and
BE IT FURTHER RESOLVED that copies of this memorial be transmitted to the United States department of the interior bureau of land management and the bureau of Indian affairs, the secretary of cultural affairs, the secretary of Indian affairs, the secretary of environment, the governor and the New Mexico congressional delegation.

West -5

Colorado, Wyoming, Montana, Idaho, Washington, Oregon, Utah, Nevada, California, Alaska, Hawaii

Full Name (First and Last): Jared Hanley
Name of Organization or Community: NatureQuant
City and State: Eugene, OR
Brief description about the concern: Given the clear connections between nature and health, and the existing inequities in nature access in the U.S., Environmental Justice frameworks must include a measurement for nearby-nature.

What do you want the WHEJAC to advise the White House Council on Environmental Quality to do?: Please consider using a calculation for beneficial nature (like NatureScore) in the EJ Scorecard.

Full Name (First and Last): Pamela L. Perez, PhD
Name of Organization or Community: California State University, Northridge

City and State: Tarzana, California
Brief description about the concern: The Tree as a Cyclical and Natural Water Producing Agent Trees have two primordial functions, which benefit the health of our planet. 1) Trees produce oxygen, while absorbing toxic gases from the air. 2) Trees collect water. They produce water. The function, which is of interest in this commentary is the latter. The natural simplicity of the trees' water producing function is as follows: In following with the perfection of its own natural design, the large stature of the tree allows its leaves to collect rainwater and moisture from the air in general. This water, collected by the leaves, is transported through the tree's vascular system to its trunk.. From this depository, the water reaches the ground, where it is stored in its roots. Through osmosis, the water found in the tree's roots is absorbed into the surrounding ground. This ground water, originally sourced from the tree, provides for the double terrestrial benefit, of preventing land erosion. The additional positive aspects of using trees, as a water producing instrument, is that their maintenance is extremely low cost, which is beneficial to low income communities. Also, in being a natural and organic element, and resource, the trees' usage to such an end, leaves no toxic chemical residues. In order to take full advantage of the trees' water producing ability, a cultivation of this natural organism must be done on a systematic, as well as on both a very extensive and intensive basis. Planting must be done in strategic areas, such as along roadsides, where contaminants occur from the accumulation of fuel of passing vehicles, as well as from the concentration of rubber particles released by the friction of the automobile tires moving on the pavement. Also, an intensive and extensive planting of trees must be realized in urban centers, with special attention devoted to their industrial nuclei, with their consequential release of contaminents. Using an organic element such as the tree, in order to solve environmental problems, such as the exposure of human beings to the toxic component, lead, dates back to the philosophies of the traditional environmentalists of the 19th Century, and even before. Their ecological belief system was founded on the faith these naturalists placed upon the superb capacity of self correction our planet has always exerted upon itself. The Earth's ability to self-regulate, and correct itself, proved to exist even before man appeared as an inhabitant of our planet's surface. To ignore Earth's innate capacity to heal and regulate itself, would be akin to denying the validity of antibodies, as a component of the human anatomy, as natural and efficient combatants, in warding off human disease and illness.

What do you want the WHEJAC to advise the White House Council on Environmental Quality to do?:
I would, please, advise and recommend the White House Council on Environmental Quality to engaging in the planting and cultivation of trees on a massive intensive and extensive level, that is to say locally, nationally and from a global policy perspective. Thank you for your most kind attention in this important matter.

Full Name (First and Last): John Price
Name of Organization or Community: Richland, WA
City and State: Richland, WA
Brief description about the concern: US Department of Energy, Office of Legacy Management has a web page for EJ. USDOE Office of Environmental Management has no web page information regarding EJ.

What do you want the WHEJAC to advise the White House Council on Environmental Quality to do?:
direct USDOE, Office of Environmental Management to add web pages that describe their implementation plan for Environmental Justice.

Full Name (First and Last): Irene Svete
Name of Organization or Community: Member of AARP and Audubon Society

City and State: Seattle, WA
Brief description about the concern: Noise and air pollution from aircraft, freeways and construction should be considered as an environmental justice issue. I live in one of the few affordable multifamily communities in the Capitol Hill neighborhood of Seattle. We are sandwiched between Interstate 5 and the flight approach to SeaTac Airport. In addition, we are buffeted by noise from construction and float-plane flights. This becomes ever worse in the summer as, like many buildings in the Pacific Northwest, we do not have air conditioning and the cost of retrofitting the building is prohibitive. Recently, Washington Department of Transportation applied for a waiver for the freeway intersection north of us to exceed noise ordinances at night for four years. In the summer, I have to dust my floors and furniture daily to keep up with the black particulate matter that drifts in the windows. To the south of me, just above I-5, are two public housing projects for seniors and at least two low-income buildings operated by nonprofits. Long-term exposure to traffic noise has been linked to heart disease. According to Harvard Health Publishing, "every 5-decibel increase in the average 24-hour noise level was associated with a 34% increase in heart attacks, strokes, and other serious heart-related problems." Heart problems are already a major issue for seniors.

What do you want the WHEJAC to advise the White House Council on Environmental Quality to do?: Noise pollution needs to be included as an environmental justice issue and should be considered in community impact statements for future airport and highway expansions. Further, there need to be efforts made to mitigate the existing impacts. Thank you!

International Comments

Full Name (First and Last): John Oladejo Oluwaleye
Name of Organization or Community: Gender Based Violence as a Public Health Issue
City and State: Mowe / Ogun
Brief description about the concern: The percentage of criminality is approximately 45% especially in Asia and Africa countries while corruption can be rated as 75% within their Governments politicians and ordinary civilians Government must not man over justice system justice must be independent throughout the whole world they must declare freedom for justice in Asia and west Africa power of the justice in Asia and west Africa power of the justice must not be undermine by Governments Council must be implemented to system of justice they need the council must be set up in local and international level to monitor justice system to audit them for accountability the council need to published information to regional so they can comments

What do you want the WHEJAC to advise the White House Council on Environmental Quality to do?: There must be council in justice system most especially in Asia and Africa countries, these council must be set up both locally and international level selected from different countries the council implemented the roles of monitoring , auditing and accountability to justice system to eradicate corruption in justice the council will published their information's to regionals to comments Please take notes security personnel needed to be assigned to regionals The council need to screening and monitoring The council must on the roles of auditing monitoring and accountability the justice need to submit their report to council there must be must be council there must be council, the council must be selected from local level and international level from different countries their roles is to monitor audit and do accountability for justice system security personnel needed to be assign for regional

Dockett Comments

White House Environmental Justice Advisory Council Virtual Public Meeting
Comments of Ian Zabarte, Secretary of the Native Community Action Council and Principal Man of the Western Bands of the Shoshone Nation of Indians (18 Statute 689). These comments characterize the abuse suffered by the Shoshone people as a result of violation of peremptory norms, environmental racism and racial bias by other Americans in the discharge of official duties authorized by the President and Congress and advise creation of the treaty reservation by the President as a remedy for the safety, protection and benefit of all the Western Bands of the Shoshone Nation. Reminding the United States of the 1848 Treaty of Guadalupe Hidalgo Article 11, "…special care shall then be taken not to place its Indian occupants under the necessity of seeking new homes…". Enforcing the obligation to protect and defend the United States Constitution, Article 6 Section 2, treaty supremacy clause, "…all treaties made, or which shall be made, under the authority of the United States, shall be the supreme law of the land; and the judges in every state shall be bound thereby, anything in the Constitution or laws of any State to the contrary notwithstanding." Restraining Americans by the Nevada Enabling Act by, "Provided, further, That nothing in this act contained shall be construed to impair the rights of person or property now pertaining to the Indians in said Territory, so long as such rights shall remain unextinguished by treaty between the United States and such Indians, or to include any territory which, by treaty with any Indian tribe, is not, without the consent of said tribe, to be included within the territorial limits or jurisdiction of any State or Territory; but all such territory shall be excepted out of the boundaries and constitute no part of the Territory of Nevada." Acting on the alliance between the Western Bands of the Shoshone Nation and the United States guaranteed by Article 6 of the Treaty of Ruby Valley (18 Statute 689), I herein request the creation of a treaty homeland for the safety and protection of the Western Shoshone people and to settle current off-reservation occupancy by American nationals trespassing in Western Shoshone Indian Country defined by the Treaty of Ruby Valley, Article 5, the Doty Map, and the Indian Claims Commission Judicially Established Indian Lands 1978 that did not complete the statutorily defined process and did, in fact, find that no taking had occurred, according the ICC 1978 Final Report to Congress. There has been no explicit act of Congress to diminish or extinguish Indian title to the Western Bands of the Shoshone Nation defined by the Treaty of Ruby Valley or survey the extent of the treaty defined lands of the Western Shoshone people. Therefore, without claims by the United Stated made pursuant to the Treaty of Ruby Valley American nationals are trespassing in doing their job administering acts of Congress within the homelands of the Western Bands of the Shoshone Nation.
American colonialism exploits the homelands of the Western Bands of the Shoshone Nation for mineral development, nuclear weapons development and disposal of high-level nuclear waste

without regard for the safety or protection of the Shoshone people and homelands. Those activity manifest abuse by taking resources from Western Shoshone property by taxation without representation, an abuse in the case of the states of California, Nevada, Utah and Idaho where those tax funds are then distributed to all levels of local government except the Indians. We possess the only ratified treaty in California and Nevada. We seek equity and environmental justice to protect the land and people of the Western Bands of the Shoshone Nation.

Nuclear development poses the most tragic consequences upon the people and land of the Western Bands of the Shoshone Nation because it is done in secret. It is my family, my tribe and my property that are being destroyed systematically. Killing Indians in secret will not stand. Nuclear weapons testing and nuclear material disposal inflict conditions intended to bring about the destruction of the Shoshone Nation, a peremptory norm in International Law that the United States has acceded under the Proxmire Act in 1988 (18 USC 1091) and the 2009 Human Rights Enforcement Act. They include over 900 above and below ground nuclear tests; development of Yucca Mountain as a high-level nuclear waste repository; negotiation by Nevada with the Department of Energy for disposal of nuclear material within the Treaty of Ruby Valley boundaries without regard for the safety of the Shoshone people. It is the culture of secrecy that manifest intent to violate the aforementioned law(s).

We advised the Department of Energy the Shoshone people are vulnerably and that lifestyle differences are important. We advised the Environmental Protection Agency of the disproportionate burden of risk the Shoshone people are to be exposed in siting the Yucca Mountain nuclear waste repository that is currently in licensing by the Nuclear Regulatory Commission. We entered the Atomic Safety Licensing Board proceedings at the Nuclear Regulatory Commission Docket 63-001 with three contentions of Ownership of land, ownership of water and disproportionate burden of exposure risk based on lifestyle differences. Yucca Mountain is Western Shoshone property and even with the Bureau of Land management Master Title Plats the Department of Energy cannot prove ownership according to 10 CFR 60.121 OWNERSHIP. Origin is important and Shoshone individuals need to be followed for health consequences known to be plausible from exposure to radiation in fallout from nuclear weapons testing. The Shoshone people need funding for collaborative research to monitoring, surveillance and registries of Shoshone down-winders affected by nuclear weapons. We need radiation exposure compensation and the creation of a treaty homeland for the safety and protection of all Western Shoshone people. Further protection of the Shoshone Nation can happen if all federal actions are required to prove ownership free of significant encumbrances and the creation of a reservation under the Treaty of Ruby Valley Article 6 for all my people.

Our experience shows that all Americans need universal healthcare because of past exposure to radioactive fallout from testing nuclear weapons of mass destruction. Silence on the situation of the Western Shoshone is systemic racism and structured racism. The Shoshone Nation will continue to protect and defend the Shoshone people and land, the Treaty of Ruby Valley and the Constitution of the United States of American without fail. We seek as much of other Americas. We can protect our environment, our Mother Earth by ending our nuclear obsession.

Thank you. Ian Zabarte, Secretary Native Community Action Council Principal Man Ian Zabarte

Western Bands of the Shoshone Nation of Indians Treaty of Ruby Valley (Consolidated Treaty Series Vol. 127 1863)

Dear WHEJAC, I have really enjoyed your presentation over the last few days. I commend and respect your efforts towards solving an important and challenging issue. I would like to suggest again using "nature access" as a core component of the "EJ Scorecard." As seen here, there are powerful links between proximity to nature and health outcomes (as well as the mitigation of hazards - air pollution, urban heat islands, waste management, water quality, etc.). Further, there are dramatic inequities in terms of nature across the US. My organization would like to provide ***free national data*** (by census block group or census tract) to help identify communities that are nature deficient. Please let me know how we can support your efforts. Thanks - Jared H.

When this started in our community we organized a petition and gathered over 1,000 signatures and over 700 comments. I have attached that petition. Since then it's only gotten worse as they have slowly lowered the altitude from over 5,000 ft to over 3,000 ft. This, despite having been sued by the city and lost, received the petition and ongoing complaints. They just don't care. Please make them care.

TO: White House Environmental Justice Advisory Council

RE: Public Comment

FROM: Maryann Aberg, Founder of Grassroots Logan Aircraft Noise Working Group

One decade ago, the Federal Aviation Administration introduced NextGen RNAV aviation policies in the Boston area, gradually concentrating flights over a few, narrow residential corridors to geographically isolate environmental hazards and increase carrier profits without raising the concern of most citizens. Touted as a means of promoting “efficiency” and “safety,” this deeply cynical strategy exposes hundreds of thousands of residents to a disproportionate share of noise and particulate pollution without acknowledging and addressing the effects of its operations on a minority of “sacrificial” neighborhoods. Since 2011, the situation has deteriorated into a crisis—disrupting lives, decreasing property values, and placing residents’ health and welfare at risk. Enabled by passage of the flawed Airport Noise and Capacity Act of 1990 and a deliberate misapplication of the National Environmental Policy Act (NEPA), FAA shrewdly calculated its standards for environmental assessment to return findings of “no significant impact.” Yet results from the recent, scientifically rigorous Neighborhood Environmental Survey (NES) show that a much greater proportion of people are highly annoyed by aircraft noise across all levels of DNL than was previously thought. Earlier studies underestimated aircraft annoyance because they included all forms of transportation—including noise generated by road, rail, and air—and utilized a mix of less robust methodologies. In contrast, NES focuses exclusively on aircraft-noise annoyance and utilizes state-of-the-art methodologies in both design and modeling. Not surprisingly, NES results refute the efficacy of the long-standing Schultz-curve and FICON standards, which have been the foundation of existing aviation noise policy for years. Logan Aircraft Noise Working Group and other grassroots organizations throughout the nation do not believe it is morally defensible to expose a relatively small segment of citizens—especially children, elders, racial minorities, and the economically disadvantaged—to a disproportionate burden of jet traffic departing from a single airport. Although the adverse effects of some aviation noise and pollution are necessary to meet the transportation needs of everyone in the United States, environmental justice demands that these harms be equally distributed among all populations rather than unfairly burdening those with less education, income, and political clout. As a matter of public policy, procedures developed by a public institution to regulate aircraft noise and particulate emissions are manifestly “unjust” if they disenfranchise the right of one segment of citizens to quiet enjoyment of their homes in favor of another segment, defined in both cases by their proximity to an arbitrary RNAV flight path. The coast-to-coast groundswell of opposition to RNAV policies in the United States over the past decade provides prima facie evidence of FAA’s “unfair, significant, and systematic” obstruction of citizens’ basic right to quiet enjoyment of their homes. As the primary regulating body for aviation policy in the United States, FAA has an ethical obligation to replace procedures that (1) are detrimental to a segment of the public, (2) were implemented without residents’ consent, and (3) are under its authority. These corrections do not require new legislation. To fulfill this mandate, FAA must provide a timely roadmap for changing current noise regulations and utilize NES results as the new basis for evaluation of community impacts (including FAA’s Environmental Review Process and Part 150): •FAA must use the additional, existing N-Above metric, which counts the number of aviation-noise events over a certain location and decibel level, to better reflect noise impacts on communities. New noise metrics need not be developed or researched before noise policy is changed.•FAA must immediately begin to report N-Above in addition to DNL. Communities should not be forced to wait for new FAA regulations when such data can be reported now.•FAA must appoint an independent Blue-Ribbon Commission of multi-disciplinary experts (e.g., environmental scientists, public and medical health professionals, engineers) to identify metrics and thresholds that will define “significant impact” based on the NES results as well as the actual experiences of people, local noise environments, nighttime noise, and current scientific knowledge.•FAA must commission the National Academies to form (1) an independent committee within the Division of Medicine to produce a consensus report on the health effects of noise and pollution and (2) an independent committee within the Division of Sciences to produce a consensus report on ultrafine particles. Both committees would use existing scientific studies and knowledge.Along with hundreds of thousands of other citizens throughout the country, members of Logan Aircraft Working Group urge the White House Environmental Justice Advisory Council to acknowledge and remedy this serious national crisis immediately.

DATE: JANUARY 31, 2022
TO: WHITE HOUSE ENVIRONMENTAL JUSTICE ADVISORY COUNCIL
RE: PUBLIC COMMENT ABOUT ENVIRONMENTAL JUSTICE: THE PROBLEM OF TRANSPORTATION NOISE

FROM: DANIEL FINK MD MBA
BOARD CHAIR
THE QUIET COALITION
LINCOLN MA 01773
A program of Quiet Communities, Inc.

As stated in the American Public Health Association policy statement on noise, released earlier this month [1], noise is harmful and/or unwanted sound.[2] As Garret Keizer wrote in 2010, noise is “a ‘weak’ issue because it affects ‘the weak’ ”. [3] Keizer goes on to write, “Make a list of the people most likely to be affected by loud noises…either because of their greater vulnerability to the effects of loud sound or because of their greater likelihood of being exposed to it, and you come up with a set of members whose only common features are their humanity and their lack of clout.” He goes on to list children, the elderly, the physically ill, racial minorities, the poor, laborers, and prisoners. Keizer’s remarks are especially true for transportation noise (road traffic noise, railroad noise, and most notably aircraft noise) which affects poor and minority populations disproportionately. [4] Additionally, transportation noise, from the internal combustion engine, diesel engines, and jet engines, is inextricably linked with particulate matter, including PM10 and PM2.5, and gaseous pollutants. One might say that transportation noise is the musical accompaniment to air pollution from vehicles, trains, and aircraft. It can be difficult to separate the adverse health effects of transportation noise from those of the associated air pollution, but this can be done with sophisticated statistical techniques. [5] Furthermore, noise is stressful [6], and stress has been shown to cause inflammation of the blood vessel lining, increased cardiovascular disease, and death. [7] The link between noise stress and cardiovascular disease has been best made for aircraft noise by Munzel et al. in Germany. [8, 9 among many others] Transportation noise also has adverse effects on animals, including fish and marine mammals. Ocean noise does not affect humans directly, but its effects on those living in the waters are indisputable. We urge you to take action to reduce transportation noise in and adjacent to poor and minority communities. This will also lead to cleaner air for those populations to breathe. A quieter world will be a cleaner world and a healthier world for all. Please do not hesitate to contact me if you have any questions or need more information about the adverse non-auditory effects of noise on health. Most doctors are still not aware of this important body of information. DISCLOSURE: I serve as an Expert Consultant to the World Health Organization on its Make Listening Safe Program, and as a subject matter expert on noise and the public for the National Center for Environmental Health at the Centers for Disease Control and Prevention. I have no financial conflicts or interests to disclose. 1. APHA policy statementhttps://apha.org/Policies-and-Advocacy/Public-Health-Policy-Statements/Policy-Database/2022/01/07/Noise-as-a-Public-Health-Hazard 2. New definition of noiseA new definition of noise: noise is unwanted and/or harmful sound. Noise is the new 'secondhand smoke'. 3. Keizer, G. The Unwanted Sound of Everything We Want: A Book About Noise. New York: Public Affairs 2010 4. Article by GA CaseyRace/Ethnicity, Socioeconomic Status, Residential ... - NCBIhttps://www.ncbi.nlm.nih.gov › articles › PMC5744659 5. Halonen article about road traffic noise and cardiovascular disease and death in LondonRoad traffic noise is associated with increased cardiovascular ..https://pubmed.ncbi.nlm.nih.gov › …6. Babisch statement about noise being stressful is in this articleA meta-analysis Babisch W - Noise Healthhttps://www.noiseandhealth.org › article › issn=1463-174…7. Tawakol article stress and the heartRelation between resting amygdalar activity and ... - The Lancethttps://www.thelancet.com › lancet › article › full text 8.Environmental Noise and the Cardiovascular System - JACC ...https://www.jacc.org › doi › abs › j.jacc.2017.12.015 9.Adverse Cardiovascular Effects of Traffic Noise with a Focus ...https://www.annualreviews.org › doi › annurev-publhealt...

They are turning a small mom and pop airport into a monster. Terrible noise and environmental issues. Please help

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People who live in airport impacted communities are subjected to constant levels of noise and emissions from aircraft and attendant traffic that EPA knows causes health effects and short to long term damage. These impacts include but are not limited to heart, lung, brain, cognition, sleep loss, hypertension and COPD. A recent survey of health impacts in a 10 mile radius around Sea-Tac Airport found that this area of 670,000 residents have a disproportionate number of these effects including increased hospitalizations, premature births, lung illness, educational impacts, lower life expectancy, higher deaths from disease to name some, when compared to people living outside the radius. These effects are more pronounced the closer to the airport. The majority of these residents are eligible for EJ consideration and are some of the poorest residents of the county. People have been talking about the need to pay attention to this area and these disproportionate impacts for 20 years and so far, nothing has been done while the problems worsened. Its time for action. And while the effects are evident and the pollution load of noise and emissions attributable to Sea-Tac Airport exceeds that of all other industry in the region that EPA governs, both for noise and emissions, the airport is moving forward with expansion plans that will worsen the situation. While regulators are aware of this problem, (I have kept Region X EPA updated on all the new information as it became available), they continue to ignore the problems caused by this unregulated, unmonitored and unmitigated polluter. It is already too much pollution, people are already exhibiting negative health effects and the sources are known. You cannot add polluters in this environment and expect things to get better. I hope you will consider doing some or all of the following: Restore NEPA to its former condition prior to the re-write under the Trump Administration. Include consideration for airports as a source in any new proposed legislation. Require the polluter to prove their mitigation plans work. Consider noise walls around major roadways that connect airports (the wealthier areas have them but the airport communities do not) EPA needs to be a voice for EJ communities and a mediator between communities and the polluter. EJ residents are more unable to lobby for their own protection due to language barriers and other social detriments. It is incumbent upon the EPA to make sure people are subject to equal protection and not wait for legal action or community groups to form before safeguarding the environment.

WHITE HOUSE 2 - ENVIRONMENTAL JUSTICE ADVISORY COUNCIL
Testimony January 26, 2022, 3:00 PM, Catherine Skopic
Renewable Energy Only - Protect Hudson River - Reject CHPE

CODE RED - that's what we're in, as proclaimed by the Intergovernmental Panel on Climate Change, the IPCC. We're in CLIMATE EMERGENCY that calls for ACTION!

I'm here to ask for the following ACTIONS1. **Rescind the Presidential Permit** allowing energy cables to cross our border with Canada; 2. **Recognize** the racial, environmental and economic injustice of CHPE and 3. **Reject** the CHPE project.

My name is Catherine Skopic, I live in New York, am an artist, activist and educator. New York State has ambitious climate legislation in the 2019 Community Leadership and Climate Protection Act - CLCPA. We have active development of off and on-shore wind, solar, heat pumps, geothermal and other renewable energy projects, all good.

However, there is one energy project - NOT New York State initiated - that is **not** good as it produces **methane, CO2, causes racial, environmental and economic injustice -** the Champlain-Hudson Power Express - CHPE, or "Chippie." initiated by the privately-owned company, Blackstone with over a decade of trying to get it realized.

Hydro Quebec builds mega-dams resulting in monstrously-sized reservoirs that give off huge amounts of **methane and CO2.** Their non -renewable energy would travel by marine and land cable to New York City.These mega-dams are built without permission on Indigenous lands, destroying their centuries-old means of survival - hunting, fishing, trapping, and they cause methyl-mercury, a poison, formed by the submerging of vegetation. One native called it "Slow Genocide."
As fine as Canada is in other ways, we can't have more methane & CO2.

Cables would be laid under Lake Champlain and the Hudson River, the dredging process ruining marine life and the drinking water of the Hudson 7- people of the 7 communities along the Hudson River who get their drinking water from the Hudson, as the PCBs and toxins stirred up would render their water undrinkable. These cables also give off electromagnetic frequencies that disrupt fish productivity and can harm people, as well.

NYS would be exporting its jobs and energy dollars to Canada - leaving NYS rate payers responsible for the estimated $3.6 **B. c**ost, causing economic hardship for some.

Rescind, Recognize, Reject the Champlain-Hudson Power Express! Thank you!
PEACE, Catherine -Thank you Pres. Biden & Administration, Advisory Council

To: whejac@epa.gov

Subject: NEJAC Public Meeting January 5, 2022; Public Comment

To NEJAC:

I am Teresa Wilkinson, RN, OMD, and I have held professional positions in personal health for 42 years. I learned from a patient of mine that you are accepting comments about environmental justice issues. That client is highly educated as a professional water utility engineer who has been tracking the science and history of water fluoridation for more years than he is willing to admit. He has indicated that the US Public Health Service has conclusive data showing exposure to fluoride disproportionately harms Blacks and other vulnerable subgroups – an environmental *injustice* - and that the visible evidence can be seen in their teeth as dental fluorosis. Dental fluorosis is seen as the paper-white blotches embedded in the otherwise uniform and slightly translucent tooth enamel, which are actually from incomplete mineralization of tooth structure due to systemic fluoride exposure during pre-eruptive tooth development, and that's from ingesting fluoride from toothpaste and drinking water.

Evidently the dental profession and supporting U.S. federal agencies have been pulling the wool over our eyes and giving us a false sense of security by ignoring the fact that the fluoride added to public drinking water is not purified in any way. One only must google for some truth (even from the CDC itself, but only after digging deep into its website) to learn that it comes from the contaminated toxic waste in pollution control systems at fertilizer factories. Having learned that fact, I am totally amazed, even ashamed, that we and our children (OMG!!) have been and are still consuming what has been an industrial hazardous waste, as a form of medicine to prevent cavities. Shame on all who have been promoting and giving the okay to this insidious practice!

As a trained acupuncturist and massage therapist, I have treated many clients for various musculoskeletal conditions, using acupuncture and other natural remedies. I have encountered numerous cases of arthritis, which I have also learned may not be arthritis at all, but progressively debilitating forms of skeletal fluorosis resulting from a lifetime of daily fluoride exposure.

I have also learned that recent studies have shown fluoride to be just as toxic to early brain development as lead, as from prenatal, neonatal, and early childhood exposure. This is truly shocking! That the pregnant mother's body does not protect the fetus from toxic fluoride.

My dentists have always promoted fluoride treatments in the dentist chair and touted the benefits of fluoride added to our drinking water. They never told me that the fluoride added to water is not pharmaceutical grade but is actually from that toxic waste, not even purified to remove other harmful substances, like arsenic and lead. Maybe these dentists don't know it either!

If the NEJAC can do anything about this deceptive practice, please do so.

Thank you in advance for giving this issue your long overdue attention.

To: whejac@epa.gov

Subject: WHEJAC Public Meeting January 26-27, 2022; Public Comment

To WHEJAC:

I am Teresa Wilkinson, RN, OMD, and I have held professional positions in personal health for 42 years. I learned from a patient of mine that you are accepting comments about environmental justice issues. That client is highly educated as a professional water utility engineer who has been tracking the science and history of water fluoridation for more years than he is willing to admit. He has indicated that the US Public Health Service has conclusive data showing exposure to fluoride disproportionately harms Blacks and other vulnerable subgroups – an environmental *injustice* - and that the visible evidence can be seen in their teeth as dental fluorosis. Dental fluorosis is seen as the paper-white blotches embedded in the otherwise uniform and slightly translucent tooth enamel, which are actually from incomplete mineralization of tooth structure due to systemic fluoride exposure during pre-eruptive tooth development, and that's from ingesting fluoride from toothpaste and drinking water.

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If the WHEJAC can do anything about this deceptive practice, please do so.

Thank you in advance for giving this issue your long overdue attention.

26 January 2022

White House Environmental Justice Advisory Council (WHEJAC)
Docket Number EPA-HQ-OA-2021-0683
whejac@epa.gov

Re: Comments to the WHEJAC, January 2022

Thank you for the continued opportunities to offer comments to the WHEJAC. I speak as a scientist working on environmental justice and climate justice with Healthy Gulf[1], a Gulf of Mexico coastal conservation non-profit. I speak in defense of the marginalized communities of people of color and low incomes on the Gulf Coast. The time has come for action. I urge the WHEJAC to make recommendations now, so that those suffering the most from pollution, climate change and COVID can receive the help they've been asking for.

In Lake Charles, Louisiana this morning (Jan 25th, 2022), there was an explosion of ethylene dichloride, a liquid hydrocarbon that is a precursor to PVC plastic. The plume of smoke ballooning from the Westlake Chemical plant could be seen for miles around. Community members heard a loud boom and one person reported that they thought a car had crashed into their house because of the noise and they felt their house shaking. Can you imagine that? Feeling afraid because you don't know what's happening, with explosion sounds and your house shaking? Now think of that happening multiple times a year. Every couple of months, even weeks, there's another emergency, another shelter in place order, another evacuation order until plant workers can safely return. There is a siren system in greater Lake Charles, like a tornado siren say, that goes off when there's a fossil fuel or petrochemical emergency. If there are enough emergencies to warrant a siren system, that is too many emergencies.

Greater Lake Charles is in Calcasieu Parish. Calcasieu Parish has a population of around 217,000, of which 26% is Black (as of the 2020 Census). Lake Charles, where the vast majority of the fossil fuel industrial complex is located, is 48% Black. Many of you have heard the story of Mossville, a town of the descendants of formerly enslaved people. Mossville is an egregious example of environmental injustice, and suffering that should have never come about at the hands of corporate greed and corporate welfare. And now there are hundreds of thousands more people of color in the metro area that are also systematically discriminated against and bear an outsized pollution burden. This cannot be allowed to continue.

At the moment, each fossil fuel chemical complex is permitted separately for air pollutants, despite being adjacent with other complexes also emitting hazardous pollutants. In some cases, several air pollution permits are authorized for different

[1] Healthy Gulf's purpose is to collaborate with and serve communities who love the Gulf of Mexico by providing research, communications and coalition-building tools needed to reverse the long-pattern of over exploitation of the Gulf's natural resources.

components of the same complex. How will the public ever be fully informed of even the pollution legally permitted, if different overlapping facilities receive permits separately? Isn't that the point of some of these permits - that there should be a cap on how much pollution is allowable? And forget about water pollution and hazardous waste - trying to identify what is being released into the water and buried in the sand is like finding a needle in a haystack already because of the mountains of technical paperwork within which this information is hidden away.

As if this situation weren't enough to warrant action, there's more. Southwest Louisiana (Calcasieu and Cameron Parish) is the site of the largest liquified fossil gas ("LNG") export terminal expansion in the country. If these terminals all get built, southwest Louisiana will export larger volumes of LNG than any country in the world. Nevermind the extensive pipelines that will also be constructed through people's homes. These massive terminals give off the greenhouse gases of four power plants per year - millions of metric tons of carbon dioxide equivalent.

So on top of the environmental injustice from pollution (and pipelines) of the fossil fuel chemical complexes, now southwest Louisiana is also told that they will be better off having greenhouse gas belching LNG export terminals. These same terminals will fuel the runaway train of climate change, which wreaked havoc in the region in 2020 from Hurricanes Laura and Delta. Thousands of people are still displaced from those storms. Similar stories are unfolding as we speak in southeast Louisiana following Hurricane Ida. The list of needs on the Gulf Coast for Disaster Justice is too long to be enumerated and include safe housing and evacuation, food security and what i'm calling "universal basic disaster income".

In sum, i want to call for:

1. WHEJAC should demand that the EPA (or other agencies) and the corporations ensure the human rights of clean water and clean air, by: a) telling people what's in their water and b) telling people what's in their air.
2. WHEJAC should recommend that the agencies and the White House revoke and/or deny permits for those facilities in the fossil fuel industrial complex that add pollution or climate injustice to environmental justice communities.
3. WHEJAC should insist that every environmental analysis of each project involve a rigorous cumulative impacts analysis.
4. All of the other recommendations i have made and submitted to the record in the past, such as:
    - WHEJAC should demand that the EPA (and other agencies) require the corporations to ensure the human rights of clean water and clean air, by: a) telling people what's in their water and b) telling people what's in their air
        - Require states and counties/parishes to track cases of cancer and other illnesses (including COVID-19) that can be exacerbated by pollution by location or locations where the person lives (as opposed to the hospital they are treated at)
        - Make this data is anonymized but publicly and readily available

    - Require agencies like USCG, EPA, FEMA and NOAA to openly coordinate after a disaster especially in industrial zones, and to share with the public what's in the air and water
- Immediate implementation of a Gulf Coast investigation group as Advisor McCarthy agreed to at the WHEJAC meeting on 11/17/21
- Require that greenhouse gas (GHG) emissions be taken into account in NEPA
- Ensure that rigorous Environmental Justice review (including cumulative impacts) is conducted as a part of NEPA
    - Set out clear methodology for designation of an EJ area, and local revisions/petitions to be incorporated
    - WHEJAC should create methods for EJ analysis and community identification that are much more rigorous than what is currently used by the US Army Corps of Engineers, the Federal Energy Regulatory Commission and other agencies
- Establish Just Transition plans for the Gulf Coast (and the nation) informed by regional Green New Deal goals

Polluting industries need to be held accountable, and new massive fossil fuel complexes must be allowed to go forward as though the Gulf is a "sacrifice zone". The WHEJAC must recommend and agitate for the change we wish to see in these communities, and others like them throughout the Gulf South. There's no time to waste.

On behalf of Healthy Gulf and our thousands of members across the Gulf south, on behalf of the undersigned and everyone they represent, on behalf of everyone who loves the Gulf of Mexico, on behalf of the land and the rivers and the watersheds, on behalf of the ecosystems and the global climate crisis, we urge the WHEJAC to institute the changes we need to dismantle systemic environmental and climate racism and injustice.

I look forward to a swift response.
(submitted via Docket at regulations.gov and email)

Best regards,

Naomi Yoder, Staff Scientist
Healthy Gulf
PO Box 66226
Houston, TX 77266
naomi@healthygulf.org