Chemicals in Food

Children's diets are an important pathway for exposure to some environmental chemicals and other contaminants. Children may be at a greater risk for exposures to contaminants because they consume more food relative to their body weight than do adults. Additionally, children's dietary patterns are often less varied than those of adults, suggesting that there are greater opportunities for continuous exposure to a foodborne contaminant than in adults.[1]

Food contamination can come from multiple sources, including antibiotics and hormones in meat and dairy products, as well as microbial contamination that can lead to illness. An estimated 48 million Americans suffer from foodborne illnesses each year,[2] and children under age five have the highest incidence of most of these infections.[3] Microbial contamination of food is monitored and regulated by a number of federal agencies, including the Department of Agriculture and the Food and Drug Administration.[i] In addition, a wide variety of chemicals from man-made sources may be found in or on foods, typically at low levels. Chemicals in foods may come from application of pesticides to crops, from transport of industrial chemicals in the environment, or from chemicals used in food packaging products. A number of persistent environmental contaminants tend to accumulate in all types of animals, and are frequently found in meat, poultry, fish, and dairy products. Other chemicals, such as perchlorate and a variety of pesticides, are often found in fruits, vegetables, and other agricultural commodities. Some chemicals in food, such as mercury and perchlorate, have naturally occurring as well as man-made sources. The health risks from chemicals in food are dependent on both the actual level of a chemical in the food as well as the amount of the food consumed by individuals.

Following this text, an indicator is presented for organophosphate pesticides in selected foods. Many chemicals of concern in food lack sufficient data to generate reliable, nationally representative indicators, particularly for children. Selected chemicals of concern for children's health that are frequently found in foods are summarized below. Further details can be found in the Biomonitoring section of this report for several of these chemicals, including methylmercury, polychlorinated biphenyls (PCBs), polybrominated diphenyl ethers (PBDEs), phthalates, perfluorochemicals (PFCs), and perchlorate.

Methylmercury

Mercury is a naturally occurring element that is released to the environment from a variety of sources, including the combustion of coal, the use of mercury in industrial processes, and from breakage of products such as mercury thermometers and fluorescent lighting, as well as from natural sources such as volcanoes. Mercury may enter water bodies through direct release or

[i] More information on microbial contaminants in food is available at http://www.fda.gov/Food/ResourcesForYou/Consumers/ucm103263.htm, http://fsrio.nal.usda.gov/pathogen-detection-and-monitoring, and http://www.fsis.usda.gov/fact_sheets/Foodborne_Illness_&_Disease_Fact_Sheets/index.asp.

through emissions to the atmosphere that are subsequently deposited to surface waters. Bacteria in water bodies convert the deposited mercury into methylmercury.[4] Methylmercury can be absorbed by small aquatic organisms that then are consumed by predators, including fish.[5] As each organism builds up methylmercury in its own tissues, and as smaller fish are eaten by larger fish, concentrations of methylmercury can accumulate, particularly in large fish with longer lifespans[6-8] such as sharks and swordfish.[9]

EPA has determined that methylmercury is known to have neurotoxic and developmental effects in humans.[10] This conclusion is based on severe adverse effects observed in exposed populations in two high-dose mercury poisoning events in Japan and Iraq. Some other studies of populations with prenatal exposure to methylmercury through regular consumption of fish have reported more subtle adverse effects on childhood neurological development.[11-15] Although ingestion of methylmercury in fish may be harmful, other compounds naturally present in many fish (such as high quality protein and other essential nutrients) are extremely beneficial.

In particular, fish are an excellent source of omega-3 fatty acids, which are nutrients that contribute to the healthy development of infants and children.[16] Pregnant women are advised to seek dietary sources of these fatty acids, including many species of fish. However, the levels of both methylmercury and omega-3 fatty acids can vary considerably by fish species. Thus, the type of fish, as well as portion sizes and frequency of consumption are all important considerations for health benefits of fish and the extent of methylmercury exposure.[16] For this reason, EPA and the U.S. Food and Drug Administration (FDA) provide advisory information on fish consumption to females who are pregnant, breastfeeding, or of childbearing age, and to young children. The advisory encourages consumption of up to 12 ounces per week of a variety of fish and shellfish that are lower in mercury, or, in the absence of a local advisory, consumption of up to 6 ounces per week of fish caught from local waters and no other fish that week. EPA and FDA also recommend that these categories of women and young children avoid consuming shark, swordfish, tile fish, or king mackerel, because these species may contain high levels of methylmercury.[17] Fish that are high in omega-3 fatty acids and low in mercury are expected to offer the greatest health benefits.[9,16,18] EPA and FDA are currently working to update the fish consumption advisory to incorporate the most current science regarding the health benefits of fish consumption and the risks from methylmercury in fish. In 2011, the Departments of Agriculture and Health and Human Services jointly released the 2010 Dietary Guidelines for Americans, which recommended that pregnant or breastfeeding women should consume 8–12 ounces of seafood per week, but avoid consumption of the same high-mercury-containing fish identified in EPA and FDA's advisory.[19] More information regarding current fish advisories, and links to lists of fish and shellfish typically containing lower levels of mercury, can be found at http://water.epa.gov/scitech/swguidance/fishshellfish/fishadvisories/general.cfm#tabs-4. Tribal and state-specific fish advisories can be found at http://fishadvisoryonline.epa.gov/General.aspx.

Polychlorinated biphenyls

Polychlorinated biphenyls (PCBs) are a group of persistent chemicals used in electrical transformers and capacitors for insulating purposes, in gas pipeline systems as a lubricant, and in caulks and other building materials. The manufacture, sale, and use of PCBs were generally banned by law in 1979, although EPA regulations have authorized their continued use in certain existing electrical equipment. Due to their persistent nature, large reservoirs of previously released PCBs remain in the environment. PCBs accumulate in fat tissue, so they are commonly found in foods derived from animals. Consumption of fish is a common source of PCB exposure, but other foods with lower PCB levels that are consumed more frequently, including meat, dairy, and poultry products, also contribute to PCB exposure.[20,21] A study by the U.S. Department of Agriculture found that levels of certain PCBs in beef and chicken declined between 2002 and 2008, while levels in turkey and pork remained relatively constant during the same years.[22] Exposure to PCBs remains widespread;[23,24] however, declining environmental levels of PCBs suggest that children today are exposed to lower levels of PCBs compared with children in previous generations.[20,25-28]

Prenatal exposure to PCBs has been associated with adverse effects on children's neurological development and impaired immune response, primarily through studies of populations that consume fish regularly.[29-31] Although there is some inconsistency in the epidemiological literature, several reviews of the literature have found that the overall evidence supports a concern for effects of PCBs on children's neurological development.[29,30,32-34] The Agency for Toxic Substances and Disease Registry has determined that "Substantial data suggest that PCBs play a role in neurobehavioral alterations observed in newborns and young children of women with PCB burdens near background levels."[20] Some studies have also detected associations between childhood exposure and adverse health effects.[30,35-37] In addition to PCBs, many other organochlorine chemicals, including dioxins, dibenzofurans, and organochlorine pesticides, are persistent and bioaccumulative and are frequently found in foods derived from animals.[38]

Polybrominated diphenyl ethers

Polybrominated diphenyl ethers (PBDEs) are a class of flame retardants used in many applications, including furniture foam, small appliances, and electronic products. PBDEs are intended to slow the ignition and rate of fire growth. Of three forms of PBDEs once used in the United States (pentaBDE, octaBDE, and decaBDE), only the decaBDE form, used primarily in televisions, personal computers, and other electrical appliances, is still in production. Manufacturers of decaBDE have agreed to phase out all uses of the chemical by the end of 2013.[39] However, products manufactured prior to the elimination of the pentaBDE and octaBDE forms in 2004, and products manufactured prior to the phaseout of decaBDE in 2013, can remain in use and contribute to the presence of PBDEs in the environment.

Like PCBs, PBDEs are persistent in the environment, accumulate in fat tissue, and have been found in a variety of foods, including fish, meat, poultry, and dairy products as well as breast milk.[40-48] Exposure studies have concluded that the presence of PBDEs in house dust and in foods are both important contributors to PBDE exposures for people of all ages, and that exposures from house dust are generally greater than those from food.[46,47,49-54] PBDE toxicity to the developing nervous system as well as endocrine disruption have been identified as areas of potential concern.[40,55-59]

Bisphenol A

Bisphenol A (BPA) is an industrial chemical used in the production of epoxy resins used as inner liners of metallic food and drink containers to prevent corrosion. BPA is also used in the production of polycarbonate plastics that may be used in food and drink containers. The primary route of human exposure to BPA is through diet, when BPA migrates from food and drink containers, particularly when a container is heated.[60-62]

Much of the scientific interest in BPA is related to published research suggesting that BPA may be an endocrine disrupting chemical.[63,64] Endocrine disruptors act by interfering with the biosynthesis, secretion, action, or metabolism of naturally occurring hormones.[63-65] BPA has demonstrated developmental effects in laboratory animals at high doses, though the effects of lower doses similar to typical human exposure levels are the subject of scientific debate.[61,66-70] Based on a critical review of the existing scientific literature, in 2008 the National Toxicology Program (NTP) determined that there was “some concern” (the midpoint on a five-level scale ranging from “negligible” to “serious”)[ii] for effects of BPA on the brain, behavior, and prostate gland in fetuses, infants, and children.[61] Although there is uncertainty regarding the effects in humans of BPA at typical exposure levels, several retailers and manufacturers have begun phasing out baby products such as bottles and sippy cups that contain BPA. Several states have also introduced legislation to ban or limit BPA in food containers and consumer products. Additional studies by both government and non-government entities are being conducted to provide additional information and address uncertainties about the safety of BPA.

Phthalates

Phthalates are a class of chemicals commonly used to increase the flexibility of plastics in a wide array of consumer products, and have been used in food packaging.[71-74] Some phthalates have been found at higher levels in fatty foods such as dairy products, fish, seafood, and oils, which are most likely to absorb phthalates.[74] Phthalates in a mother’s body can enter her breast milk. Ingestion of that breast milk and infant formula containing phthalates may also contribute to infant phthalate exposure.[75] Certain phthalates are suspected endocrine disruptors, and have shown a number of reproductive and developmental effects in laboratory animal studies[76-85] as well as some reported associations in human epidemiological studies.[86-89]

[ii] More information on NTP concern levels is available at http://www.niehs.nih.gov/news/media/questions/sya-bpa.cfm.

Perfluorochemicals

Perfluorochemicals (PFCs) are a group of chemicals used in a variety of consumer products, including food packaging, and in the production on nonstick coatings on cookware.[90,91] Long-chain PFCs, including perfluorooctane sulfonic acid (PFOS) and perfluorooctanoic acid (PFOA), have already been or will be phased out by the chemical industry by 2015, although the persistence of these chemicals means that they will remain in the environment for several years despite reductions in emissions. While the routes of human exposure to PFCs are not fully understood, two recent studies have identified food consumption as the primary exposure pathway.[92,93] PFC-treated food-contact packaging, such as microwave popcorn bags, may be a source of PFC exposure.[94,95] Heating these materials may cause PFCs to migrate into food, or into the air where they may be inhaled.[iii] Meats may also be contaminated with PFCs due to exposure of source animals to air, water, and feed contaminated with PFCs.[95-97] PFCs have also been detected in some plant-based foods.[93] Studies in laboratory animals have demonstrated reproductive and developmental toxicity of PFCs.[98,99] Some human health studies have reported associations between prenatal exposure to PFCs and a number of adverse birth outcomes,[100-103] while other studies have not.[104,105]

Perchlorate

Perchlorate is a naturally occurring and man-made chemical that has been detected in surface water and groundwater in the United States.[106-109] Perchlorate is used in the manufacture of fireworks, explosives, flares, and rocket propellant.[107,109] Perchlorate has been detected in human breast milk, dairy products, as well as in leafy vegetables and other produce.[108,110-115] Infant formulas have been found to contain perchlorate, and the perchlorate content of the formula is increased if it is prepared with perchlorate-contaminated water.[116-118]

Exposure to high doses of perchlorate has been shown to inhibit iodide uptake into the thyroid gland, thus possibly disrupting the function of the thyroid and potentially leading to a reduction in the production of thyroid hormone.[107,119,120] Thyroid hormones are particularly important for growth and development of the central nervous system in fetuses and infants.[121] Due to the sensitivities of the developing fetus, perchlorate exposures among pregnant women, especially those with preexisting thyroid disorders or iodide deficiency, carry the potential for risk of adverse health effects.

[iii] The U.S. Food and Drug Administration recently worked with several manufacturers to remove grease-proofing agents containing C8 perfluorinated compounds from the marketplace. These manufacturers volunteered to stop distributing products containing these compounds in interstate commerce for food-contact purposes as of October 1, 2011. For more information, see http://www.fda.gov/Food/FoodIngredientsPackaging/FoodContactSubstancesFCS/ucm308462.htm.

Organophosphate Pesticides

Agricultural crops are frequently treated with pesticides to control insects and other pests that may affect crop growth. Some of the most prevalent classes of pesticides used in growing food crops are the carbamates, pyrethroids, and the organophosphates. After crops are harvested, they may retain residues of these pesticides. Apples, corn, oranges, rice, and wheat are among the agricultural commodities consumed in large amounts by children.

Organophosphates are one class of pesticides that are of concern for children's health. Examples of organophosphate pesticides include chlorpyrifos, azinphos methyl, methyl parathion, and phosmet. These pesticides are frequently applied to many of the foods important in children's diets, and certain organophosphate pesticide residues can be detected in small quantities on these foods. Organophosphates can interfere with the proper function of the nervous system when exposure is sufficiently high.[1,122] Childhood is a period of increased vulnerability, because many children may have low capacity to detoxify organophosphate pesticides through age 7 years.[123] Recent studies have reported an association between prenatal organophosphate exposure and childhood attention deficit/hyperactivity disorder (ADHD) in U.S. communities with relatively high exposures to organophosphate pesticides,[124] as well as with exposures found within the general US population.[125] Other recent studies have reported associations between prenatal organophosphate pesticide exposures and a variety of neurodevelopmental deficits in childhood, including reduced IQ, perceptual reasoning, and memory.[126-128] Since 1999, EPA has imposed restrictions on the use of the organophosphate pesticides azinphos methyl, chlorpyrifos, and methyl parathion on certain food crops and around the home, due largely to concerns about potential exposures of children.[129-131]

The 1996 Food Quality Protection Act required EPA to identify and assess the extent of dietary pesticide exposure in the United States, and to determine whether there was a "reasonable certainty of no harm" to vulnerable populations including infants and children.[132] The U.S. Department of Agriculture's Pesticide Data Program (PDP) provides data annually on pesticide residues in food, with a specific focus on foods often consumed by children.[133] Other researchers have supplemented the PDP with their own analyses. A recent study measured pesticide residues in 24-hour duplicate food samples of fruits, vegetables, and juices served to children, and found that 14% of the samples contained at least one organophosphate pesticide.[134] Additional pesticide residue data are available from FDA's pesticide residue monitoring program.[135] A number of pesticide residues, along with a variety of other chemicals in food, are also measured in FDA's Total Diet Study.[136] When pesticide residue data are combined with dietary consumption surveys, it can be possible to estimate pesticide exposure from dietary intake.

Indicator E9 presents the percentage of samples of two fruits and two vegetables analyzed by the USDA PDP that have detectable residues of organophosphate pesticides. This indicator allows for a general comparison of the frequency of organophosphate detection over time for four foods typically consumed by children, although data are not available on each fruit or vegetable for every year. This indicator has been revised since the publication of *America's Children and the Environment, Third Edition* (January 2013) to include data for 2010–2017.

Indicator E9: Percentage of sampled apples, carrots, grapes, and tomatoes with detectable residues of organophosphate pesticides, 1998–2017

About the Indicator: Indicator E9 presents the percentage of sampled apples, carrots, grapes, and tomatoes that were found to contain detectable residues of organophosphate pesticides from 1998–2017. These foods were selected because they are frequent components of children's diets, and because data for these foods were available for multiple years. The data are from an analysis of pesticide residues in foods conducted annually by the U.S. Department of Agriculture.

Pesticide Data Program

The U.S. Department of Agriculture (USDA) collects data on pesticide residues in food annually. USDA's Pesticide Data Program (PDP), initiated in 1991, focuses on measuring pesticide residues in foods that are important parts of children's diets, including apples, apple juice, bananas, carrots, grapes, green beans, orange juice, peaches, pears, potatoes, and tomatoes.

Samples are collected from food distribution centers in 10 states across the country.[137] The PDP has a statistical design in which food samples are randomly selected from the national food distribution system and reflect what is typically available to the consumer, including both domestic and imported foods.[137] Different foods are sampled each year. In its history up to 2009, the PDP tested for more than 440 different pesticides.[133] In 2017, the PDP analyzed fruit and vegetables for 512 pesticides and related chemicals. Prior to analysis, the PDP processes samples by following the preparations an average individual would use before consuming an item. This includes washing fruits and vegetables, as well as removing inedible portions of a food item. For example, tomatoes and grapes are washed with the stems and other materials removed, while apples are washed and the stems and cores are removed.

Data Presented in the Indicator

Indicator E9 displays the percentage of apple, grape, carrot, and tomato samples with detectable organophosphate pesticide residues reported by the PDP from 1998–2017. These four foods were selected as those that were sampled by the PDP in at least five years from 1998–2017 and are among the 20 most-consumed foods identified in an analysis by EPA.[138] Other foods not shown here may have either greater or lesser frequencies of organophosphate pesticide residue detection than the four foods presented in this indicator.

The 42 organophosphates that were sampled in every one of the years 1998–2017 are included in calculation of the indicator; 95 other organophosphates that were added to or dropped from the program in these years are excluded so that the chart represents a consistent set of pesticides for all years shown. Some aspects of trends in organophosphate residues could be missed by the indicator if any organophosphates other than the 42 considered in the indicator had substantial changes in use on the four selected foods during the years 1998–2017. For example, a decrease in the percentage of detections of organophosphate residues may reflect an actual decrease in the use of organophosphate pesticides, but can definitively represent only a decrease in the residues of the 42 OPs included in the indicator; it does not account for potential substitution with other organophosphates or other types of pesticides.

The indicator also defines "detectable" based on the ability to measure residues in the PDP in 1998, so that introduction of more sensitive measurement techniques over time does not affect the indicator and allows for direct comparison of data collected in previous years with those collected today. This means that some produce samples analyzed in recent years with improved detection technology would, for purposes of indicator calculation, be considered to have non-detectable organophosphate residues based on comparison with the older, higher limit of detection.[iv]

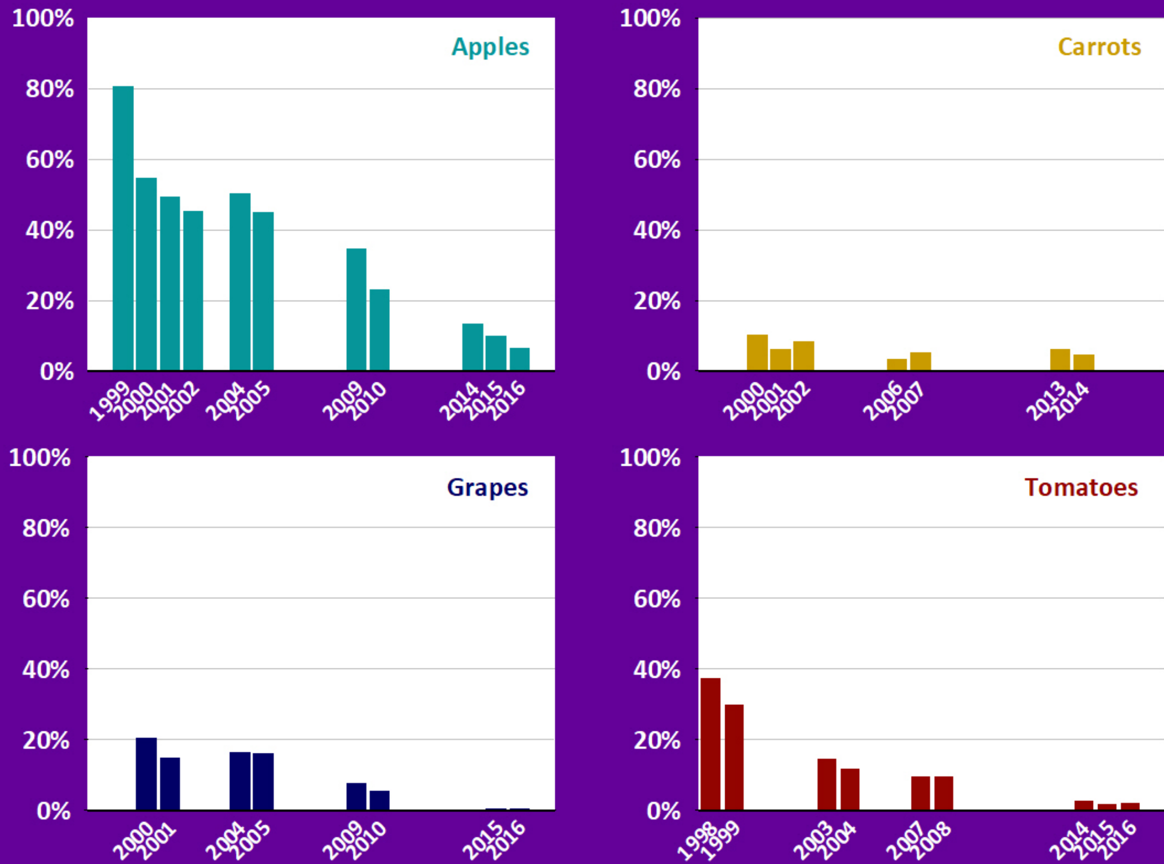
The fruits and vegetables shown in this indicator were each sampled in seven to eleven years between 1998 and 2017. Gaps in the percentage of residue detections from year to year thus represent missing information, rather than an absence of organophosphate residues.

This indicator is a surrogate for children's exposure to pesticides in foods: If the frequency of detectable levels of pesticides in foods decreases, it is likely that exposures will decrease. However, the indicator does not account for many additional factors that affect the risk to children. For example, some organophosphates pose greater risks to children than others do, and residues on some foods may pose greater risks than residues on other foods due to differences in amounts consumed. The indicator also does not distinguish between residue levels that are barely detectable and those that are much higher, which would pose a greater concern for children's health. Finally, exposures to organophosphate pesticides may also occur by pathways other than the diet, such as ingestion of pesticides present in house dust and drinking water.

[iv] An alternate analysis of the data that considered all detectable residues, without holding the limit of detection constant at 1998 levels, resulted in percentages of food samples with detectable organophosphate pesticide residues very similar to those shown in the indicator.

Indicator E9

Percentage of sampled apples, carrots, grapes, and tomatoes with detectable residues of organophosphate pesticides, 1998-2017



Data: U.S. Department of Agriculture, Pesticide Data Program

Note: Pesticide residues were measured only in selected years for each food.
Years without data represent missing values, rather than absence of residues.

America's Children and the Environment, Third Edition, Updated August 2019

Data characterization

- Data for this indicator are obtained from a U.S. Department of Agriculture program that measures pesticide residues in food samples collected from 10 states.
- Food samples are randomly selected from the national food distribution system and reflect what is typically available to the consumer.
- The types of foods sampled change over time; so, for example, data for pesticide residues on apples are not available every year.
- The indicator is calculated using the measurement sensitivity as of 1998 for each year shown; more sensitive measurement techniques have been incorporated over time.

- In 1999, 81% of sampled apples had detectable organophosphate pesticide residues. In 2016, 6% had detectable residues.

- In 2000, 10% of sampled carrots had detectable organophosphate pesticide residues. In 2014, 5% had detectable residues.
- In 2000, 21% of sampled grapes had detectable organophosphate pesticide residues. In 2016, less than 1% had detectable residues.
- In 1998, 37% of sampled tomatoes had detectable organophosphate pesticide residues. In 2016, 2% had detectable residues.

References

1. National Research Council. 1993. *Pesticides in the Diets of Infants and Children*. Washington, DC: National Academy Press. http://www.nap.edu/catalog/2126.html?se_side.

2. Scallan, E., P.M. Griffin, F.J. Angulo, R.V. Tauxe, and R.M. Hoekstra. 2011. Foodborne illness acquired in the United States--unspecified agents. *Emerging Infectious Diseases* 17 (1):16-22.

3. Centers for Disease Control and Prevention. 2011. Vital Signs: Incidence and trends of infection with pathogens transmitted commonly through food--foodborne diseases active surveillance network, 10 U.S. sites, 1996-2010. *Morbidity and Mortality Weekly Report* 60 (22):749-755.

4. Guimaraes, J.R.D., J. Ikingura, and H. Akagi. 2000. Methyl mercury production and distribution in river water-sediment systems investigated through radiochemical techniques. *Water, Air, and Soil Pollution* 124 (1-2):113-124.

5. Chen, C.Y., R.S. Stemberger, B. Klaue, J.D. Blum, P.C. Pickhardt, and C.L. Folt. 2000. Accumulation of heavy metals in food web components across a gradient of lakes. *Limnology and Oceanography* 45 (7):1525-1536.

6. Dietz, R., F. Riget, M. Cleemann, A. Aarkrog, P. Johansen, and J.C. Hansen. 2000. Comparison of contaminants from different trophic levels and ecosystems. *Science of the Total Environment* 245 (1-3):221-231.

7. Gilmour, C.C., and G.S. Riedel. 2000. A survey of size-specific mercury concentrations in game fish from Maryland fresh and estuarine waters. *Archives of Environmental Contamination and Toxicology* 39 (1):53-59.

8. Mason, R.P., J.R. Reinfelder, and F.M.M. Morel. 1995. Bioaccumulation of mercury and methylmercury. *Water, Air, and Soil Pollution* 80:915-921.

9. Mahaffey, K.R. 2004. Fish and shellfish as dietary sources of methylmercury and the omega-3 fatty acids, eicosahexaenoic acid and docosahexaenoic acid: risks and benefits. *Environmental Research* 95 (3):414-28.

10. U.S. Environmental Protection Agency. 1997. *Mercury Study Report to Congress Volumes I to VII*. Washington DC: U.S. Environmental Protection Agency Office of Air Quality Planning and Standards and Office of Research and Development. EPA-452/R-97-003. http://www.epa.gov/hg/report.htm.

11. Karagas, M.R., A.L. Choi, E. Oken, M. Horvat, R. Schoeny, E. Kamai, W. Cowell, P. Grandjean, and S. Korrick. 2012. Evidence on the human health effects of low-level methylmercury exposure. *Environmental Health Perspectives* 120 (6):799-806.

12. Lederman, S.A., R.L. Jones, K.L. Caldwell, V. Rauh, S.E. Sheets, D. Tang, S. Viswanathan, M. Becker, J.L. Stein, R.Y. Wang, et al. 2008. Relation between cord blood mercury levels and early child development in a World Trade Center cohort. *Environmental Health Perspectives* 116 (8):1085-91.

13. Lynch, M.L., L.S. Huang, C. Cox, J.J. Strain, G.J. Myers, M.P. Bonham, C.F. Shamlaye, A. Stokes-Riner, J.M. Wallace, E.M. Duffy, et al. 2011. Varying coefficient function models to explore interactions between maternal nutritional status and prenatal methylmercury toxicity in the Seychelles Child Development Nutrition Study. *Environmental Research* 111 (1):75-80.

14. National Research Council. 2000. *Toxicological Effects of Methylmercury*. Washington, DC: National Academy Press.

15. Oken, E., J.S. Radesky, R.O. Wright, D.C. Bellinger, C.J. Amarasiriwardena, K.P. Kleinman, H. Hu, and M.W. Gillman. 2008. Maternal fish intake during pregnancy, blood mercury levels, and child cognition at age 3 years in a US cohort. *American Journal of Epidemiology* 167 (10):1171-81.

16. Institute of Medicine. 2006. *Seafood Choices: Balancing Benefits and Risks*. Washington, DC: Committee on Nutrient Relationships in Seafood: Selections to Balance Benefits and Risks. Food and Nutrition Board. Institute of Medicine. http://iom.edu/Reports/2006/Seafood-Choices-Balancing-Benefits-and-Risks.aspx.

17. U.S. Environmental Protection Agency, and U.S. Food and Drug Administration. 2004. *What You Need to Know About Mercury in Fish and Shellfish. Advice for Women who Might Become Pregnant, Women who are*

Pregnant, Nursing Mothers and Children. Washington, DC: U.S. Environmental Protection Agency and U.S. Food and Drug Administration. EPA-823-F-04-009. http://www.epa.gov/waterscience/fish/files/MethylmercuryBrochure.pdf.

18. Ginsberg, G.L., and B.F. Toal. 2009. Quantitative approach for incorporating methylmercury risks and omega-3 fatty acid benefits in developing species-specific fish consumption advice. *Environmental Health Perspectives* 117 (2):267-75.

19. U.S. Department of Agriculture, and U.S. Department of Health and Human Services. 2010. *Dietary Guidelines for Americans, 2010*. Washington, DC: U.S. Government Printing Office. http://www.cnpp.usda.gov/Publications/DietaryGuidelines/2010/PolicyDoc/PolicyDoc.pdf.

20. Agency for Toxic Substances and Disease Registry (ATSDR). 2000. *Toxicological Profile for Polychlorinated Biphenyls (PCBs)*. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. http://www.atsdr.cdc.gov/toxprofiles/tp.asp?id=142&tid=26.

21. Choi, A.L., J.I. Levy, D.W. Dockery, L.M. Ryan, P.E. Tolbert, L.M. Altshul, and S.A. Korrick. 2006. Does living near a Superfund site contribute to higher polychlorinated biphenyl (PCB) exposure? *Environmental Health Perspectives* 114 (7):1092-8.

22. U.S. Department of Agriculture. 2009. *DIOXIN 08 Survey: Dioxin and Dioxin-Like Compounds in the U.S. Domestic Meat and Poultry Supply*. Washington, DC: USDA Food Safety and Inspection Service, Office of Public Health Science, Risk Assessment Division. http://www.fsis.usda.gov/PDF/Dioxin_Report_1009.pdf.

23. Axelrad, D.A., S. Goodman, and T.J. Woodruff. 2009. PCB body burdens in US women of childbearing age 2001-2002: An evaluation of alternate summary metrics of NHANES data. *Environmental Research* 109 (4):368-78.

24. Patterson, D.G., Jr., L.Y. Wong, W.E. Turner, S.P. Caudill, E.S. Dipietro, P.C. McClure, T.P. Cash, J.D. Osterloh, J.L. Pirkle, E.J. Sampson, et al. 2009. Levels in the U.S. population of those persistent organic pollutants (2003-2004) included in the Stockholm Convention or in other long range transboundary air pollution agreements. *Environmental Science & Technology* 43 (4):1211-8.

25. Hickey, J.P., S.A. Batterman, and S.M. Chernyak. 2006. Trends of chlorinated organic contaminants in great lakes trout and walleye from 1970 to 1998. *Archives of Environmental Contamination and Toxicology* 50 (1):97-110.

26. Schecter, A., O. Papke, K.C. Tung, J. Joseph, T.R. Harris, and J. Dahlgren. 2005. Polybrominated diphenyl ether flame retardants in the U.S. population: current levels, temporal trends, and comparison with dioxins, dibenzofurans, and polychlorinated biphenyls. *Journal of Occupational and Environmental Medicine* 47 (3):199-211.

27. Sjodin, A., R.S. Jones, J.F. Focant, C. Lapeza, R.Y. Wang, E.E. McGahee, 3rd, Y. Zhang, W.E. Turner, B. Slazyk, L.L. Needham, et al. 2004. Retrospective time-trend study of polybrominated diphenyl ether and polybrominated and polychlorinated biphenyl levels in human serum from the United States. *Environmental Health Perspectives* 112 (6):654-8.

28. Sun, P., I. Basu, P. Blanchard, K.A. Brice, and R.A. Hites. 2007. Temporal and spatial trends of atmospheric polychlorinated biphenyl concentrations near the Great Lakes. *Environmental Science and Technology* 41 (4):1131-6.

29. Schantz, S.L., J.C. Gardiner, D.M. Gasior, R.J. McCaffrey, A.M. Sweeney, and H.E.B. Humphrey. 2004. Much ado about something: the weight of evidence for PCB effects on neuropsychological function. *Psychology in the Schools* 41 (6):669-679.

30. Schantz, S.L., J.J. Widholm, and D.C. Rice. 2003. Effects of PCB exposure on neuropsychological function in children. *Environmental Health Perspectives* 111 (3):357-376.

31. Selgrade, M.K. 2007. Immunotoxicity: the risk is real. *Toxicological Sciences* 100 (2):328-32.

32. Boucher, O., G. Muckle, and C.H. Bastien. 2009. Prenatal exposure to polychlorinated biphenyls: a neuropsychologic analysis. *Environmental Health Perspectives* 117 (1):7-16.

33. Ribas-Fito, N., M. Sala, M. Kogevinas, and J. Sunyer. 2001. Polychlorinated biphenyls (PCBs) and neurological development in children: a systematic review. *Journal of Epidemiology and Community Health* 55 (8):537-46.

34. Wigle, D.T., T.E. Arbuckle, M.C. Turner, A. Berube, Q. Yang, S. Liu, and D. Krewski. 2008. Epidemiologic evidence of relationships between reproductive and child health outcomes and environmental chemical contaminants. *Journal of Toxicology and Environmental Health B Critical Reviews* 11 (5-6):373-517.

35. Jacobson, J.L., S.W. Jacobson, and H.E. Humphrey. 1990. Effects of exposure to PCBs and related compounds on growth and activity in children. *Neurotoxicology and Teratology* 12 (4):319-26.

36. Vreugdenhil, H.J., P.G. Mulder, H.H. Emmen, and N. Weisglas-Kuperus. 2004. Effects of perinatal exposure to PCBs on neuropsychological functions in the Rotterdam cohort at 9 years of age. *Neuropsychology* 18 (1):185-93.

37. Walkowiak, J., J.A. Wiener, A. Fastabend, B. Heinzow, U. Kramer, E. Schmidt, H.J. Steingruber, S. Wundram, and G. Winneke. 2001. Environmental exposure to polychlorinated biphenyls and quality of the home environment: effects on psychodevelopment in early childhood. *Lancet* 358 (9293):1602-7.

38. Institute of Medicine. 2003. *Dioxins and Dioxin-like Compounds in the Food Supply*. Washington, DC: National Academy Press. http://books.nap.edu/openbook.php?record_id=10763&page=R1.

39. U.S. Environmental Protection Agency. 2010. *DecaBDE Phase-out Initiative*. U.S. Environmental Protection Agency. Retrieved February 26, 2010 from http://www.epa.gov/oppt/existingchemicals/pubs/actionplans/deccadbe.html.

40. Costa, L.G., and G. Giordano. 2007. Developmental neurotoxicity of polybrominated diphenyl ether (PBDE) flame retardants. *Neurotoxicology* 28 (6):1047-1067.

41. Frederiksen, M., K. Vorkamp, M. Thomsen, and L.E. Knudsen. 2009. Human internal and external exposure to PBDEs--a review of levels and sources. *International Journal of Hygiene and Environmental Health* 212 (2):109-34.

42. Huwe, J.K., and G.L. Larsen. 2005. Polychlorinated dioxins, furans, and biphenyls, and polybrominated diphenyl ethers in a U.S. meat market basket and estimates of dietary intake. *Environmental Science and Technology* 39 (15):5606-11.

43. Rose, M., D.H. Bennett, A. Bergman, B. Fangstrom, I.N. Pessah, and I. Hertz-Picciotto. 2010. PBDEs in 2-5 year-old children from California and associations with diet and indoor environment. *Environmental Science and Technology* 44 (7):2648-53.

44. Schecter, A., J. Colacino, K. Patel, K. Kannan, S.H. Yun, D. Haffner, T.R. Harris, and L. Birnbaum. 2010. Polybrominated diphenyl ether levels in foodstuffs collected from three locations from the United States. *Toxicology and Applied Pharmacology* 243 (2):217-24.

45. Schecter, A., D. Haffner, J. Colacino, K. Patel, O. Papke, M. Opel, and L. Birnbaum. 2010. Polybrominated diphenyl ethers (PBDEs) and hexabromocyclodecane (HBCD) in composite U.S. food samples. *Environmental Health Perspectives* 118 (3):357-62.

46. Schecter, A., O. Papke, T.R. Harris, K.C. Tung, A. Musumba, J. Olson, and L. Birnbaum. 2006. Polybrominated diphenyl ether (PBDE) levels in an expanded market basket survey of U.S. food and estimated PBDE dietary intake by age and sex. *Environmental Health Perspectives* 114 (10):1515-20.

47. Wu, N., T. Herrmann, O. Paepke, J. Tickner, R. Hale, L.E. Harvey, M. La Guardia, M.D. McClean, and T.F. Webster. 2007. Human exposure to PBDEs: associations of PBDE body burdens with food consumption and house dust concentrations. *Environmental Science & Technology* 41 (5):1584-9.

48. Yogui, G.T., and J.L. Sericano. 2009. Polybrominated diphenyl ether flame retardants in the U.S. marine environment: a review. *Environment International* 35 (3):655-66.

49. Fraser, A.J., T.F. Webster, and M.D. McClean. 2009. Diet contributes significantly to the body burden of PBDEs in the general U.S. population. *Environmental Health Perspectives* 117 (10):1520-5.

50. Johnson-Restrepo, B., and K. Kannan. 2009. An assessment of sources and pathways of human exposure to polybrominated diphenyl ethers in the United States. *Chemosphere* 76 (4):542-8.

51. Lorber, M. 2008. Exposure of Americans to polybrominated diphenyl ethers. *Journal of Exposure Science and Environmental Epidemiology* 18 (1):2-19.

52. Stapleton, H.M., S.M. Kelly, J.G. Allen, M.D. McClean, and T.F. Webster. 2008. Measurement of polybrominated diphenyl ethers on hand wipes: estimating exposure from hand-to-mouth contact. *Environmental Science and Technology* 42 (9):3329-34.

53. U.S. Environmental Protection Agency. 2010. *An Exposure Assessment of Polybrominated Diphenyl Ethers*. Washington, DC: U.S. EPA, National Center for Environmental Assessment. EPA/600/R-08/086F. http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=210404.

54. Wei, H., M. Turyk, S. Cali, S. Dorevitch, S. Erdal, and A. Li. 2009. Particle size fractionation and human exposure of polybrominated diphenyl ethers in indoor dust from Chicago. *Journal of Environmental Science and Health, Part A: Toxic/Hazardous Substances and Environmental Engineering* 44 (13):1353-61.

55. Birnbaum, L.S., and D.F. Staskal. 2004. Brominated flame retardants: cause for concern? *Environmental Health Perspectives* 112 (1):9-17.

56. Branchi, I., F. Capone, E. Alleva, and L.G. Costa. 2003. Polybrominated diphenyl ethers: neurobehavioral effects following developmental exposure. *Neurotoxicology* 24 (3):449-62.

57. Costa, L.G., G. Giordano, S. Tagliaferri, A. Caglieri, and A. Mutti. 2008. Polybrominated diphenyl ether (PBDE) flame retardants: Environmental contamination, human body burden and potential adverse health effects. *Acta Biomedica* 79 (3):172-183.

58. Herbstman, J.B., A. Sjodin, M. Kurzon, S.A. Lederman, R.S. Jones, V. Rauh, L.L. Needham, D. Tang, M. Niedzwiecki, R.Y. Wang, et al. 2010. Prenatal exposure to PBDEs and neurodevelopment. *Environmental Health Perspectives* 118 (5):712-9.

59. McDonald, T.A. 2005. Polybrominated diphenylether levels among United States residents: daily intake and risk of harm to the developing brain and reproductive organs. *Integrated Environmental Assessment and Management* 1 (4):343-54.

60. Le, H.H., E.M. Carlson, J.P. Chua, and S.M. Belcher. 2008. Bisphenol A is released from polycarbonate drinking bottles and mimics the neurotoxic actions of estrogen in developing cerebellar neurons. *Toxicology Letters* 176 (2):149-56.

61. National Toxicology Program. 2008. *NTP-CERHR Monograph on the Potential Human Reproductive and Developmental Effects of Bisphenol A*. Research Triangle Park, NC: National Institute of Environmental Health Sciences, National Toxicology Program. http://ntp.niehs.nih.gov/ntp/ohat/bisphenol/bisphenol.pdf.

62. Vandenberg, L.N., R. Hauser, M. Marcus, N. Olea, and W.V. Welshons. 2007. Human exposure to bisphenol A (BPA). *Reproductive Toxicology* 24 (2):139-77.

63. Diamanti-Kandarakis, E., J.P. Bourguignon, L.C. Giudice, R. Hauser, G.S. Prins, A.M. Soto, R.T. Zoeller, and A.C. Gore. 2009. Endocrine-disrupting chemicals: an Endocrine Society scientific statement. *Endocrine Reviews* 30 (4):293-342.

64. vom Saal, F.S., B.T. Akingbemi, S.M. Belcher, L.S. Birnbaum, D.A. Crain, M. Eriksen, F. Farabollini, L.J. Guillette, Jr., R. Hauser, J.J. Heindel, et al. 2007. Chapel Hill bisphenol A expert panel consensus statement: integration of mechanisms, effects in animals and potential to impact human health at current levels of exposure. *Reproductive Toxicology* 24 (2):131-8.

65. Kavlock, R.J., G.P. Daston, C. DeRosa, P. Fenner-Crisp, L.E. Gray, S. Kaattari, G. Lucier, M. Luster, M.J. Mac, C. Maczka, et al. 1996. Research needs for the risk assessment of health and environmental effects of endocrine

disruptors: a report of the U.S. EPA-sponsored workshop. *Environmental Health Perspectives* 104 (Suppl 4):715-40.

66. Howdeshell, K.L., J. Furr, C.R. Lambright, V.S. Wilson, B.C. Ryan, and L.E. Gray, Jr. 2008. Gestational and lactational exposure to ethinyl estradiol, but not bisphenol A, decreases androgen-dependent reproductive organ weights and epididymal sperm abundance in the male long evans hooded rat. *Toxicological Sciences* 102 (2):371-82.

67. Palanza, P.L., K.L. Howdeshell, S. Parmigiani, and F.S. vom Saal. 2002. Exposure to a low dose of bisphenol A during fetal life or in adulthood alters maternal behavior in mice. *Environmental Health Perspectives* 110 (Suppl 3):415-22.

68. Sharpe, R.M. 2010. Is it time to end concerns over the estrogenic effects of Bisphenol A? *Toxicological Sciences* 114 (1):1-4.

69. Vandenberg, L.N., M.V. Maffini, C. Sonnenschein, B.S. Rubin, and A.M. Soto. 2009. Bisphenol-A and the great divide: a review of controversies in the field of endocrine disruption. *Endocrine Reviews* 30 (1):75-95.

70. U.S. Food and Drug Administration. 2012. *BPA*. USFDA. Retrieved July 20, 2012 from http://www.fda.gov/Food/FoodIngredientsPackaging/ucm166145.htm.

71. Agency for Toxic Substances and Disease Registry (ATSDR). 1995. *Toxicological profile for diethyl phthalate*. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.

72. Agency for Toxic Substances and Disease Registry (ATSDR). 1997. *Toxicological profile for di-n-octylphthalate (DNOP)*. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.

73. Agency for Toxic Substances and Disease Registry (ATSDR). 2001. *Toxicological profile for Di-n-butyl Phthalate. Update*. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. .

74. Agency for Toxic Substances and Disease Registry (ATSDR). 2002. *Toxicological profile for Di(2-ethylhexyl)phthalate (DEHP)*. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.

75. Mortensen, G.K., K.M. Main, A.M. Andersson, H. Leffers, and N.E. Skakkebaek. 2005. Determination of phthalate monoesters in human milk, consumer milk, and infant formula by tandem mass spectrometry (LC-MS-MS). *Analytical and Bioanalytical Chemistry* 382 (4):1084-92.

76. Andrade, A.J., S.W. Grande, C.E. Talsness, K. Grote, A. Golombiewski, A. Sterner-Kock, and I. Chahoud. 2006. A dose-response study following in utero and lactational exposure to di-(2-ethylhexyl) phthalate (DEHP): effects on androgenic status, developmental landmarks and testicular histology in male offspring rats. *Toxicology* 225 (1):64-74.

77. Barlow, N.J., B.S. McIntyre, and P.M. Foster. 2004. Male reproductive tract lesions at 6, 12, and 18 months of age following in utero exposure to di(n-butyl) phthalate. *Toxicologic Pathology* 32 (1):79-90.

78. Christiansen, S., M. Scholze, M. Axelstad, J. Boberg, A. Kortenkamp, and U. Hass. 2008. Combined exposure to anti-androgens causes markedly increased frequencies of hypospadias in the rat. *International Journal of Andrology* 31 (2):241-8.

79. Gray, L.E., Jr., J. Ostby, J. Furr, M. Price, D.N. Veeramachaneni, and L. Parks. 2000. Perinatal exposure to the phthalates DEHP, BBP, and DINP, but not DEP, DMP, or DOTP, alters sexual differentiation of the male rat. *Toxicological Sciences* 58 (2):350-65.

80. Howdeshell, K.L., V.S. Wilson, J. Furr, C.R. Lambright, C.V. Rider, C.R. Blystone, A.K. Hotchkiss, and L.E. Gray, Jr. 2008. A mixture of five phthalate esters inhibits fetal testicular testosterone production in the sprague-dawley rat in a cumulative, dose-additive manner. *Toxicological Sciences* 105 (1):153-65.

81. Lehmann, K.P., S. Phillips, M. Sar, P.M. Foster, and K.W. Gaido. 2004. Dose-dependent alterations in gene expression and testosterone synthesis in the fetal testes of male rats exposed to di (n-butyl) phthalate. *Toxicological Sciences* 81 (1):60-8.

82. Metzdorff, S.B., M. Dalgaard, S. Christiansen, M. Axelstad, U. Hass, M.K. Kiersgaard, M. Scholze, A. Kortenkamp, and A.M. Vinggaard. 2007. Dysgenesis and histological changes of genitals and perturbations of gene expression in male rats after in utero exposure to antiandrogen mixtures. *Toxicological Sciences* 98 (1):87-98.

83. Mylchreest, E., D.G. Wallace, R.C. Cattley, and P.M. Foster. 2000. Dose-dependent alterations in androgen-regulated male reproductive development in rats exposed to Di(n-butyl) phthalate during late gestation. *Toxicological Sciences* 55 (1):143-51.

84. National Research Council. 2008. *Phthalates and Cumulative Risk Assessment: The Tasks Ahead*. Washington, DC: The National Academies Press. http://www.nap.edu/catalog.php?record_id=12528.

85. Sharpe, R.M. 2008. "Additional" effects of phthalate mixtures on fetal testosterone production. *Toxicological Sciences* 105 (1):1-4.

86. Main, K.M., G.K. Mortensen, M.M. Kaleva, K.A. Boisen, I.N. Damgaard, M. Chellakooty, I.M. Schmidt, A.M. Suomi, H.E. Virtanen, D.V. Petersen, et al. 2006. Human breast milk contamination with phthalates and alterations of endogenous reproductive hormones in infants three months of age. *Environmental Health Perspectives* 114 (2):270-6.

87. Nassar, N., P. Abeywardana, A. Barker, and C. Bower. 2009. Parental occupational exposure to potential endocrine disrupting chemicals and risk of hypospadias in infants. *Occupational and Environmental Medicine* 67 (9):585-9.

88. Swan, S.H. 2008. Environmental phthalate exposure in relation to reproductive outcomes and other health endpoints in humans. *Environmental Research* 108 (2):177-84.

89. Swan, S.H., K.M. Main, F. Liu, S.L. Stewart, R.L. Kruse, A.M. Calafat, C.S. Mao, J.B. Redmon, C.L. Ternand, S. Sullivan, et al. 2005. Decrease in anogenital distance among male infants with prenatal phthalate exposure. *Environmental Health Perspectives* 113 (8):1056-61.

90. Agency for Toxic Substances and Disease Registry (ATSDR). 2009. *Toxicological profile for Perfluoroalkyls. (Draft for Public Comment)*. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. http://www.atsdr.cdc.gov/toxprofiles/tp200.pdf.

91. Calafat, A.M., L.Y. Wong, Z. Kuklenyik, J.A. Reidy, and L.L. Needham. 2007. Polyfluoroalkyl chemicals in the U.S. population: data from the National Health and Nutrition Examination Survey (NHANES) 2003-2004 and comparisons with NHANES 1999-2000. *Environmental Health Perspectives* 115 (11):1596-602.

92. Egeghy, P.P., and M. Lorber. 2010. An assessment of the exposure of Americans to perfluorooctane sulfonate: A comparison of estimated intake with values inferred from NHANES data. *Journal of Exposure Science and Environmental Epidemiology* Epub Date 2010/02/11.

93. Trudel, D., L. Horowitz, M. Wormuth, M. Scheringer, I.T. Cousins, and K. Hungerbuhler. 2008. Estimating consumer exposure to PFOS and PFOA. *Risk Analysis* 28 (2):251-69.

94. Begley, T.H., K. White, P. Honigfort, M.L. Twaroski, R. Neches, and R.A. Walker. 2005. Perfluorochemicals: potential sources of and migration from food packaging. *Food Additives and Contaminants* 22 (10):1023-31.

95. Tittlemier, S.A., K. Pepper, C. Seymour, J. Moisey, R. Bronson, X.L. Cao, and R.W. Dabeka. 2007. Dietary exposure of Canadians to perfluorinated carboxylates and perfluorooctane sulfonate via consumption of meat, fish, fast foods, and food items prepared in their packaging. *Journal of Agricultural and Food Chemistry* 55 (8):3203-10.

96. Ericson, I., R. Marti-Cid, M. Nadal, B. Van Bavel, G. Lindstrom, and J.L. Domingo. 2008. Human exposure to perfluorinated chemicals through the diet: intake of perfluorinated compounds in foods from the Catalan (Spain) market. *Journal of Agricultural and Food Chemistry* 56 (5):1787-94.

97. Schecter, A., J. Colacino, D. Haffner, K. Patel, M. Opel, O. Papke, and L. Birnbaum. 2010. Perfluorinated compounds, polychlorinated biphenyl, and organochlorine pesticide contamination in composite food samples from Dallas, Texas. *Environmental Health Perspectives* 118:796-802.

98. Era, S., K.H. Harada, M. Toyoshima, K. Inoue, M. Minata, N. Saito, T. Takigawa, K. Shiota, and A. Koizumi. 2009. Cleft palate caused by perfluorooctane sulfonate is caused mainly by extrinsic factors. *Toxicology* 256 (1-2):42-7.

99. Lau, C., J.R. Thibodeaux, R.G. Hanson, J.M. Rogers, B.E. Grey, M.E. Stanton, J.L. Butenhoff, and L.A. Stevenson. 2003. Exposure to perfluorooctane sulfonate during pregnancy in rat and mouse. II: postnatal evaluation. *Toxicological Sciences* 74 (2):382-92.

100. Apelberg, B.J., F.R. Witter, J.B. Herbstman, A.M. Calafat, R.U. Halden, L.L. Needham, and L.R. Goldman. 2007. Cord serum concentrations of perfluorooctane sulfonate (PFOS) and perfluorooctanoate (PFOA) in relation to weight and size at birth. *Environmental Health Perspectives* 115 (11):1670-6.

101. Fei, C., J.K. McLaughlin, R.E. Tarone, and J. Olsen. 2007. Perfluorinated chemicals and fetal growth: a study within the Danish National Birth Cohort. *Environmental Health Perspectives* 115 (11):1677-82.

102. Fei, C., J.K. McLaughlin, R.E. Tarone, and J. Olsen. 2008. Fetal growth indicators and perfluorinated chemicals: a study in the Danish National Birth Cohort. *American Journal of Epidemiology* 168 (1):66-72.

103. Washino, N., Y. Saijo, S. Sasaki, S. Kato, S. Ban, K. Konishi, R. Ito, A. Nakata, Y. Iwasaki, K. Saito, et al. 2009. Correlations between prenatal exposure to perfluorinated chemicals and reduced fetal growth. *Environmental Health Perspectives* 117 (4):660-7.

104. Hamm, M.P., N.M. Cherry, E. Chan, J.W. Martin, and I. Burstyn. 2010. Maternal exposure to perfluorinated acids and fetal growth. *Journal of Exposure Science and Environmental Epidemiology* 20:589-597.

105. Monroy, R., K. Morrison, K. Teo, S. Atkinson, C. Kubwabo, B. Stewart, and W.G. Foster. 2008. Serum levels of perfluoroalkyl compounds in human maternal and umbilical cord blood samples. *Environmental Research* 108 (1):56-62.

106. Centers for Disease Control and Prevention. *Perchlorate in Baby Formula Fact Sheet*. Retrieved August 13, 2009 from http://cdc.gov/nceh/features/perchlorate_factsheet.htm.

107. National Research Council. 2005. *Health Implications of Perchlorate Ingestion*. Washington, DC: National Academy Press. http://www.nap.edu/catalog.php?record_id=11202.

108. Sanchez, C.A., L.M. Barraj, B.C. Blount, C.G. Scrafford, L. Valentin-Blasini, K.M. Smith, and R.I. Krieger. 2009. Perchlorate exposure from food crops produced in the lower Colorado River region. *Journal of Exposure Science and Environmental Epidemiology* 19 (4):359-68.

109. U.S. Environmental Protection Agency. *Perchlorate*. Retrieved August 13, 2009 from http://www.epa.gov/safewater/contaminants/unregulated/perchlorate.html.

110. Dasgupta, P.K., A.B. Kirk, J.V. Dyke, and S. Ohira. 2008. Intake of iodine and perchlorate and excretion in human milk. *Environmental Science & Technology* 42 (21):8115-21.

111. Kirk, A.B., J.V. Dyke, C.F. Martin, and P.K. Dasgupta. 2007. Temporal patterns in perchlorate, thiocyanate, and iodide excretion in human milk. *Environmental Health Perspectives* 115 (2):182-6.

112. Kirk, A.B., P.K. Martinelango, K. Tian, A. Dutta, E.E. Smith, and P.K. Dasgupta. 2005. Perchlorate and iodide in dairy and breast milk. *Environmental Science & Technology* 39 (7):2011-7.

113. Murray, C.W., S.K. Egan, H. Kim, N. Beru, and P.M. Bolger. 2008. US Food and Drug Administration's Total Diet Study: dietary intake of perchlorate and iodine. *Journal of Exposure Science and Environmental Epidemiology* 18 (6):571-80.

114. Sanchez, C.A., K.S. Crump, R.I. Krieger, N.R. Khandaker, and J.P. Gibbs. 2005. Perchlorate and nitrate in leafy vegetables of North America. *Environmental Science & Technology* 39 (24):9391-7.

115. U.S. Food and Drug Administration. 2007. *2004-2005 Exploratory Survey Data on Perchlorate in Food*. U.S. FDA. Retrieved January 18, 2012 from http://www.fda.gov/Food/FoodSafety/FoodContaminantsAdulteration/ChemicalContaminants/Perchlorate/ucm077685.htm.

116. Blount, B.C., and L. Valentin-Blasini. 2007. Biomonitoring as a method for assessing exposure to perchlorate. *Thyroid* 17 (9):837-41.

117. Pearce, E.N., A.M. Leung, B.C. Blount, H.R. Bazrafshan, X. He, S. Pino, L. Valentin-Blasini, and L.E. Braverman. 2007. Breast milk iodine and perchlorate concentrations in lactating Boston-area women. *The Journal of Clinical Endocrinology and Metabolism* 92 (5):1673-7.

118. Schier, J.G., A.F. Wolkin, L. Valentin-Blasini, M.G. Belson, S.M. Kieszak, C.S. Rubin, and B.C. Blount. 2009. Perchlorate exposure from infant formula and comparisons with the perchlorate reference dose. *Journal of Exposure Science and Environmental Epidemiology* 20 (3):281-7.

119. Greer, M.A., G. Goodman, R.C. Pleus, and S.E. Greer. 2002. Health effects assessment for environmental perchlorate contamination: the dose response for inhibition of thyroidal radioiodine uptake in humans. *Environmental Health Perspectives* 110 (9):927-37.

120. U.S. Food and Drug Administration. *Perchlorate Questions and Answers*. Retrieved August 13, 2009 from http://www.fda.gov/Food/FoodSafety/FoodContaminantsAdulteration/ChemicalContaminants/Perchlorate/ucm077572.htm#effects.

121. Morreale de Escobar, G., M.J. Obregon, and F. Escobar del Rey. 2000. Is Neuropsychological Development Related to Maternal Hypothyroidism or to Maternal Hypothyroxinemia? *The Journal of Clinical Endocrinology and Metabolism* 85 (11):3975-87.

122. Eskenazi, B., A. Bradman, and R. Castorina. 1999. Exposures of children to organophosphate pesticides and their potential adverse health effects. *Environmental Health Perspectives* 107 (Suppl. 3):409-19.

123. Huen, K., Harley, K., Brooks, J., Hubbard, A., Bradman, A., Eskenazi, B., Holland, N. 2009. Developmental changes in PON1 enzyme activity in young children and effects of PON1 polymorphisms. *Environmental Health Perspectives* 117 (10):1632-8.

124. Marks, A.R., K. Harley, A. Bradman, K. Kogut, D.B. Barr, C. Johnson, N. Calderon, and B. Eskenazi. 2010. Organophosphate pesticide exposure and attention in young Mexican-American children: the CHAMACOS study. *Environmental Health Perspectives* 118 (12):1768-74.

125. Bouchard, M.F., D.C. Bellinger, R.O. Wright, and M.G. Weisskopf. 2010. Attention-deficit/hyperactivity disorder and urinary metabolites of organophosphate pesticides. *Pediatrics* 125 (6):e1270-7.

126. Bouchard, M.F., J. Chevrier, K.G. Harley, K. Kogut, M. Vedar, N. Calderon, C. Trujillo, C. Johnson, A. Bradman, D.B. Barr, et al. 2011. Prenatal exposure to organophosphate pesticides and IQ in 7-year old children. *Environmental Health Perspectives* 119 (8):1189-95.

127. Engel, S.M., J. Wetmur, J. Chen, C. Zhu, D.B. Barr, R.L. Canfield, and M.S. Wolff. 2011. Prenatal exposure to organophosphates, Paraoxonase 1, and cognitive development in childhood. *Environmental Health Perspectives* 119 (8):1182-8.

128. Rauh, V., S. Arunajadai, M. Horton, F. Perera, L. Hoepner, D.B. Barr, and R. Whyatt. 2011. 7-year neurodevelopmental scores and prenatal exposure to Chlorpyrifos, a common agricultural pesticide. *Environmental Health Perspectives* 119 (8):1196-201.

129. U.S. Environmental Protection Agency. 2002. *Interim Reregistration Eligibility Decision for Chlorpyrifos*. Washington, DC: U.S. EPA, Office of Prevention, Pesticides, and Toxic Substances. EPA 738-R-01-007. http://www.epa.gov/oppsrrd1/REDs/chlorpyrifos_ired.pdf.

130. U.S. Environmental Protection Agency. 2008. *Azinphos-Methyl (AZM) Registration Review Status*. Washington, DC: U.S. EPA, Office of Pesticide Programs. EPA-HQ-OPP-2005-0061. http://www.epa.gov/oppsrrd1/registration_review/azm/azm-status.pdf.

131. U.S. Environmental Protection Agency. 2006. *Interim Reregistration Eligibility Decision for Methyl Parathion*. Washington, DC: U.S. EPA, Office of Pesticide Programs. EPA-HQ-OPP-2006-0618. http://www.epa.gov/oppsrrd1/REDs/methylparathion_ired.pdf.

132. U.S. Environmental Protection Agency. 2010. *Food Quality Protection Act (FQPA) of 1996*. U.S. EPA, Office of Pesticide Programs. Retrieved December 28, 2010 from http://www.epa.gov/pesticides/regulating/laws/fqpa/.

133. U.S. Department of Agriculture. 2010. *Pesticide Data Program*. U.S. Department of Agriculture, Agricultural Marketing Service. Retrieved December 28, 2010 from http://www.ams.usda.gov/AMSv1.0/pdp.

134. Lu, C., F.J. Schenck, M.A. Pearson, and J.W. Wong. 2010. Assessing children's dietary pesticide exposure - direct measurement of pesticide residues in 24-hour duplicate food samples. *Environmental Health Perspectives* 118 (11):1625-30.

135. U.S. Food and Drug Administration. 2010. *FDA Pesticide Program Residue Monitoring: 1993-2008*. U.S. FDA. Retrieved January 18, 2012 from http://www.fda.gov/Food/FoodSafety/FoodContaminantsAdulteration/Pesticides/ResidueMonitoringReports/default.htm.

136. U.S. Food and Drug Administration. 2012. *Total Diet Study*. USFDA. Retrieved July 20, 2012 from http://www.fda.gov/Food/FoodSafety/FoodContaminantsAdulteration/TotalDietStudy/default.htm.

137. U.S. Department of Agriculture. 2011. *Pesticide Data Program Annual Summary, Calendar Year 2009*. Washington, DC: USDA Marketing and Regulatory Programs, Agricultural Marketing Service. http://www.ams.usda.gov/AMSv1.0/getfile?dDocName=STELPRDC5091055.

138. U.S. Environmental Protection Agency. 2002. Endocrine Disruptor Screening Program, proposed chemical selection approach for initial round of screening; request for comment. *Federal Register* 67 (250):79611-29.