BEFORE THE ADMINISTRATOR
UNITED STATES ENVIRONMENTAL PROTECTION AGENCY

In the Matter of:	)
	)
Bowen Steam-Electric Generating Plant Title V Operating Permit Amendment 4911-015-0011-V-01-3	)
	)
McDononough/Atkinson Steam-Electric Generating Plant Title V Operating Permit Amendment 4911-067-0003-V-01-3	)
	)
Yates Steam-Electric Generating Plant Title V Operating Permit Amendment 4911-077-0001-V-01-3	)
	) Petition No: 04-02-_____
Hammond Steam-Electric Generating Plant Title V Operating Permit Amendment 4911-115-003-V-01-3	)
	)
Wansley Steam-Electric Generating Plant Title V Operating Permit Amendment 4911-149-0001-V-01-6	)
	)
Scherer Steam-Electric Generating Plant Title V Operating Permit Amendment 4911-207-0008-V-01-3	)
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Branch Steam-Electric Generating Plant Title V Operating Permit Amendment 4911-237-0008-V-01-3	)
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Issued by the Georgia Environmental Protection Division	)

SIERRA CLUB AND GEORGIA PIRG'S PETITION TO HAVE THE ADMINISTRATOR OBJECT TO SEVEN GEORGIA POWER COAL FIRED POWER PLANTS TITLE V OPERATING PERMIT AMENDMENTS

I. INTRODUCTION

A recent scientific study claims that air pollution from power plants shortens the lives of over 1,600 people in Georgia each year.[1] 2,581,516 Georgians live in areas that the United States Environmental Protection Agency (EPA) has designated as failing to meet the health based ambient air quality standard for ground level ozone.[2] Ozone is a powerful lung irritant that can cause shortness of breath, coughing, burning eyes, chest pain, asthma attacks and other respiratory problems as well as a lessened ability to fight off disease and infection.[3] The 13 county Metro Atlanta area has exceeded EPA's health based National Ambient Air Quality Standard (NAAQS) for over two decades. The fact that Metro-Atlanta had almost twice as many violations of the ozone NAAQS in 2002 as it did in 2001 is not a positive trend. While some may try to blame this on the weather, it ignores the fact that we could control emissions of ozone forming chemicals to avoid dangerous levels of ozone under any weather conditions. However, as the ambient air monitoring data shows, we are not doing that.

There are also significant economic consequences of air pollution. For example, the US EPA has concluded that the direct benefits from the Clean Air Act from 1970 to 1990 had a central tendency estimate of $22.2 trillion dollars. During the same period, implementing the Clean Air Act had a direct cost of $523 billion. This means that the economic benefit of the Clean Air Act outweighed the costs by more than a factor of 42.[4] Georgia's air pollution

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[1] Death, Disease and Dirty Power, Clean Air Task Force, October 2000, at 22 available at http://www.cleartheair.org/fact/mortality/mortalitystudy.vtml?PROACTIVE_ID=cecfcfcecfccc6cdccc5cecfcfcfc5cecfc9cbcccac6c6c7c9c5cf.

[2] Smog Watch 2000, Clean Air Network, June, 2000 at 11 available at http://www.cleartheair.org/fact/SmogWatch2000.pdf?PROACTIVE_ID=cecfcfcfcacacac8c6c5cecfcfcfc5cecfcacfc9c6c8cecec9c5cf.

[3] *Id.* at 16.

[4] EPA, The Benefit and Costs of the Clean Air Act: 1970 to 1990 EPA Report to Congress, EPA-410-R-97-002, Oct. 1997 at Abstract.

problems have reached such levels as to catch the attention of the media including major local newspapers. *See e.g.* May 1, 2001 Atlanta Journal, "Bad air days: Atlanta ranks sixth in pollution."

Interposed between Georgians and the air pollution is the Clean Air Act. In simple terms, the Clean Air Act requires the EPA to set standards for safe ambient air and then requires air pollution control agencies to issue permits to major stationary sources of air pollution as well as implement regulations to control pollution from mobile sources. The permits for major stationary sources are designed to ensure that aggregate air pollution does not exceed ambient air quality standards.

This petition concerns Georgia Environmental Protection Division's (EPD) failure to take the legally mandated steps necessary to end the two decade long public health tragedy of excess ozone levels in the Metro Atlanta area. One of the key tools in our fight to end unsafe levels of ozone is the requirement that before new facilities are constructed that will add more pollution to the Metro Atlanta area, the owner of the new facility must obtain reductions in pollutants from existing facilities that are greater than the levels of pollution the new facility will emit. These reductions are known as offsets. Offsets must be real, permanent, enforceable and surplus. Georgia EPD has issued amendments to the Title V permits for seven coal fired power plants that are in or contribute pollution to the Metro Atlanta nonattainment area. The Title V permit amendments claim to create offsets. However, the offsets are bogus because they are not surplus. Rather other regulatory provisions already require the reductions in the Title V permit amendments. In other words, Georgia EPD has engaged in Enron-style accounting of double counting.

It is now up to the EPA Administrator. She can object to the seven Title V permit amendments and thus move in the direction of protecting the health and economic well-being of the millions of inhabitants of the Metro Atlanta area or she can allow business as usual to go on, thereby watching as the decades of needless loss and suffering from unsafe ozone levels continue.

II. PARTIES

The Sierra Club, a non-profit corporation, is one of the nation's oldest and largest environmental organizations. Founded in 1892, the Sierra Club has long been involved in air pollution issues in Georgia and throughout the nation. The Georgia Chapter of the Sierra Club has over 14,000 members. Sierra Club members and staff live, work, farm, recreate, grow food, own land and structures, and obtain spiritual and aesthetic pleasure from locations that are, and will continue to be adversely affected by the air pollution from the facilities in this petition as well as the air pollution from the facilities that will use these bogus offsets.

Plaintiff Georgia Public Interest Research Group ("Georgia PIRG") is a nonprofit corporation organized under the laws of the State of Georgia with its primary offices in Atlanta, Georgia. It has over 3,000 members across the State. Georgia PIRG is a non-profit, nonpartisan consumer, environmental, and "good government" watchdog organization. Georgia PIRG is part of the national network of state PIRGs that have a long history of working to protect the environment, and has been actively involved in national, state and local clean air policy debates since the early 1970s. Georgia PIRG members and staff live, work, farm, recreate, grow food, own land and structures, and obtain spiritual and aesthetic pleasure from locations that are, and will continue to be adversely affected by the air

pollution from the facilities in this petition as well as the air pollution from the facilities that will use these bogus offsets.

III. PREVIOUS PROCEEDINGS

The EPA granted final approval of the Georgia Title V operating permit program on June 8, 2000. 65 FR 36398 (June 8, 2000). The Georgia Environmental Protection Division (Georgia EPD) of the Georgia Department of Natural Resources is the agency responsible for issuing Title V operating permits in Georgia. O.C.G.A. §§12-9-3(12), 12-9-4, 12-9-6(b)(3).

Georgia EPD issued draft Title V operating permit amendments for the Bowen, McDonough/Atkinson, Yates, Hammond, Wansley, Scherer and Branch Steam-Electric Generating Plants [hereinafter collectively "Facilities"). Georgia EPD granted the public a thirty-day period to comment on the draft permit amendments. Sierra Club and Georgia PIRG submitted written comments to Georgia EPD during the public comment period. See Ex. 1. Georgia EPD then proposed the seven permit amendments to EPA. EPA did not object. See http://www.epa.gov/region4/air/permits/index.htm. Thus, the public's 60-day period in which to petition the EPA for an objection expires on November 18, 2002. See Ex. 2.

IV. FACTS

The Metro Atlanta area has been in nonattainment for ground level ozone for nearly a quarter of a century. This means that for over two decades, millions of people have been exposed to ozone that can cause babies to be born with heart valve defects, children to develop asthma if they play outside and many other adverse health effects. See Ex. 4 (ozone

causes birth defects); Ex. 5 (ozone causes asthma in children who play outside). See generally 1000 Friends of Maryland v. Browner, 265 F.3d 216, 220, n.2 (4th Cir. 2001)(ground level ozone is very harmful to human health). Elevated levels of ozone also have significant economic effects beyond just increased health care costs and lost wages. These economic effects include decreases in the productivity of agriculture such as tree farms, which is one of Georgia's largest industries.

In order to combat the evils of unsafe levels of ozone, the Georgia EPD created, and the EPA approved a Metro Atlanta ozone nonattainment State Implementation Plan (SIP). The Metro Atlanta SIP contains a requirement that before a new facility begins to emit nitrogen oxides (NOx) in or into the nonattainment area, it must obtain a reduction of NOx emissions from an existing source in an amount 1.1 times greater than will be emitted from the new facility. Georgia Rules for Air Quality Control (Rule) 391-3-1-.03(c)15. These emissions reductions are know as "offsets" because the new facility is suppose to be offsetting its new pollution with reductions in existing pollution. Offsets must be real, permanent, quantifiable, enforceable, and surplus. Rule 391-3-1-.03(13)(b)(1).

Georgia EPD attempted to create NOx offsets by amending the Title V permit for seven Georgia Power power plants. Specifically, Plant Yates' Conditions 3.2.3, 3.2.4 and 3.2.5, Plant Bowen's Conditions 3.2.6, 3.2.7 and 3.2.8, Plant Branch's Conditions 3.2.3, 3.2.4, and 3.2.5, Plant Hammond's Conditions 3.2.3, 3.2.4 and 3.2.5, Plant McDonough's Conditions 3.2.5, 3.2.6, and Plant Scherer's Conditions 3.2.6, 3.2.7, and 3.2.8 of the above referenced Title V permit amendments claim to create offsets for the 2003, 2004 and 2005 ozone season.[5]

[5] The Permit Amendments are available at http://www.air.dnr.state.ga.us/sspp/titlev/issued.html

However, the reductions created by these conditions area also required by other regulatory provisions. For example, the reductions contained in these Title V permit amendments will also be required by the EPA's NOx SIP Call which EPA has stated it will issue a final rule for in early 2003 and require compliance by May 1, 2005. See 67 FR 8395, 8396 (Feb. 22, 2002). The reductions are also required for Plants Bowen and Scherer by the New Source Review (NSR) and New Source Performance Standards (NSPS) of the Clean Air Act. See *United States v. Georgia Power*, 99CV2859-JEC (N.D.Ga. 1999) and related Notices of Violation.

Other regulatory provisions may also require the reductions contained in the Title V permit amendments. For example, the Regional Haze SIP, the Metro Atlanta severe ozone nonattainment SIP, the Georgia 2.5 micron particulate matter (PM fine) SIP and the 8-hour ozone SIPs are all also likely to require the reductions found in the challenged Title V permit amendments and more.

V. SUMMARY OF THE ARGUMENT

The offsets created by the Facilities' Title V amendment are not surplus because they are already required by the NOx SIP Call, as well as NSR and NSPS.

VI. ARGUMENT

A. LEGAL BACKGROUND AND STANDARD OF REVIEW

The Clean Air Act is "Congress's response to well-documented scientific and social concerns about the quality of the air that sustains life on earth and protects it from . . . degradation and pollution caused by modern industrial society." *Delaware Valley Citizens*

Council for Clean Air v. Davis, 932 F.2d 256, 260 (3rd Cir. 1991). A key component of achieving the Clean Air Act's goal of protecting our precious air is the Title V operating permit program. Title V permits are supposed to consolidate all of the requirements for a facility into a single permit and provide for adequate monitoring and reporting to ensure the regulatory agencies and the public that the permittee is complying with its permit. *See generally* S. Rep. No. 101-228 at 346-47; *see also In re: Roosevelt Regional Landfill*, (EPA Administrator May 11, 1999) at 64 FR 25336.

When a state or local air quality permitting authority issues a Title V operating permit, the EPA will object if the permit is not in compliance with any applicable requirement or requirements under 40 CFR Part 70. 40 CFR § 70.8(c). However, if the EPA does not object, then "any person may petition the Administrator within 60 days after the expiration of the Administrator's 45-day review period to make such objection." 40 CFR § 70.8(d); 42 U.S.C. § 7661d(b)(2)(CAAA § 505(b)(2)). "To justify exercise of an objection by EPA to a [T]itle V permit pursuant to Section 505(b)(2), a petitioner must demonstrate that the permit is not in compliance with applicable requirements of the Act, including the requirements of Part 70. [40 CFR] § 70.8(d)." *In re: Pacificorp's Jim Bridger and Naughton Plants*, VIII-00-1 (EPA Administrator Nov. 16, 2000) at 4.

B. THE TITLE V PERMIT AMENDMENTS ARE NOT IN COMPLIANCE WITH THE GEORGIA SIP REQUIREMENT THAT OFFSETS BE SURPLUS.

1. THE REDUCTIONS ARE NOT SURPLUS BECAUSE THE NOx SIP CALL REQUIRES THEM.[6]

The EPA must object to a Title V permit amendment when it is not in compliance with an applicable requirement. *In re: Pacificorp's Jim Bridger and Naughton Plants*, VIII-00-1 (EPA Administrator Nov. 16, 2000) at 4. Requirements in SIPs are applicable requirements. 40 CFR § 70.2 applicable requirement (1). The Georgia SIP requires that:

> (iv)For purposes of satisfying the emission offset requirements of this subsection, the ratio of total emission reductions of nitrogen oxides to total increased emissions of such pollutant from the new or modified electrical generating units shall be at least 1.1 to 1 for emission offsets external or internal to the contiguous area under common control at which the proposed new or modified major stationary source is located. . . .

Rule 391-3-1-.03(c)15.

These offsets must be real, permanent, quantifiable, enforceable, and surplus. Rule 391-3-1-.03(13)(b)(1). Surplus means:

> not required by any local, state, or federal law, regulation, order, or requirement and in excess of reductions used by the Division in issuing any other permit or to demonstrate attainment of federal ambient air quality standards or reasonable further progress towards achieving attainment of federal ambient air quality standards. For the purpose of determining the amount of surplus emission reductions, any seasonal emission limitation or standard shall be assumed to apply throughout the year. Emission reductions which have previously been used to avoid New Source Review through a netting demonstration are not considered surplus.

[6] Petitioners raised this issue in their Comment 1 at pages 2-3, attached as Ex. 1. Therefore, Petitioners satisfied the requirement of 40 CFR § 70.8(d) that the petition points were raised with reasonable specificity during the public comment period.

The offsets created by the seven Title V permit amendments do not comply with the Georgia SIP because they are not surplus. To begin with, the reductions are not surplus because they will be required under the NOx SIP Call which is both a state and federal regulation. The Title V permit amendments require the seven power plants to reduce their NOx emissions to 32, 335.8 tons per ozone season during the 2005 ozone season. See e.g. Plant Bowen Title V permit amendment at Condition 3.2.8. The NOx SIP Call, however, will place a limit on all Electric Generating Units (EGUs) in the top two-thirds of Georgia, including Georgia Power's seven plants, of no more than 29,416 tons of NOx per ozone season. Even if Georgia created a NOx SIP rule that allocated all of its allowance to Georgia Power's seven plants, the seven plants would still be required by the NOx SIP call to reduce its pollution to 29,416 tons per ozone season or less. See Ex. 6 at 2. Therefore, the reductions contained in the Title V permit amendments are already required by the NOx SIP Call, thus making them not surplus. EPA should object to the seven Title V permit amendments on these grounds.

EPA may respond that it need only object to the permit condition that requires reductions in 2005, because the seven power plants will not have to comply with the NOx SIP call in 2003 and 2004. The problem with that approach is that in addition to offsets having to be surplus, they also have to be permanent. Rule 391-3-1-.03(13)(b)(1). It would be arbitrary and capricious to claim that offsets that only last for two years are permanent.

Georgia EPD responded to this comment by stating that since the NOx SIP Call rule for Georgia was not final at the time Georgia Power Company submitted its applications and still is not final, the NOx SIP Call cannot be considered in setting the baseline from which the offsets are calculated. If one's goal is to protect the environment, then Georgia EPD's

approach does not make sense. The fact that the Georgia rule is not final because industry has engaged EPA in a protracted legal battle delaying the final rule is not relevant. We do know that the seven power plants will have to comply with the NOx SIP Call by May 1, 2005. 67 FR 8395, 8396 (Feb. 22, 2002). We also know that the NOx SIP Call will limit NOx emissions from the seven power plants to below the 32,335.8 tons limit contained in the Title V permit conditions. Thus, we currently know that the reductions required by the Title V permit conditions are not surplus. Despite this knowledge, to determine that the NOx SIP Call cannot be considered because it is not final is irrational.

It is worth noting that Georgia EPD's approach would also be an invitation to industry to challenge all rules that impose any emission reductions. Industry would benefit from the delays caused by litigation, because they would get to take credit for reductions that will ultimately be required by the delayed litigation.

2. THE REDUCTIONS FROM PLANTS BOWEN AND SCHERER ARE NOT SURPLUS BECAUSE THEY ARE REQUIRED BY NSR AND NSPS.[7]

The reductions achieved at Plants Bowen and Scherer are not surplus because these reductions are also required by the New Source Review (NSR) and New Source Performance Standards (NSPS) requirements of the Clean Air Act. EPA has determined that NSR's PSD is applicable to at least Unit 2 at Plant Bowen and PSD and NSPS are applicable to Unit 3 and Unit 4 at Plant Scherer. *See United States v. Georgia Power*, 99CV2859-JEC (N.D.Ga. 1999). See also Notice of Violation EPA-CAA-2000-04-0006 (Nov. 2, 1999). Attorney

[7] Petitioners raised this issue in their Comment 1 at pages 2-3, attached as Ex. 1. Therefore, Petitioners satisfied the requirement of 40 CFR § 70.8(d) that the petition points were raised with reasonable specificity during the public comment period.

General Ashcroft has recently re-affirmed the validity of this and other enforcement actions. NSR and NSPS would require Plant Scherer to reduce its emission rate to no more than 0.15 lbs /MMBtu over a three hour average year round. This is substantially below what Plant Scherer is currently permitted. Although Plant Bowen is permitted at 0.07 lbs/MMBtu, NSR would still require reductions. To begin with, Plant Bowen is permitted to emit at 0.07 lbs/MMBtu over a thirty day average. EPA Region 4 has stated, in the context of the PSD permit for the Duke Murray Facility and other places, that a 30 day averaging time for a NOx BACT limit is not acceptable. Thus, Plant Bowen would have to adopt a shorter BACT limit. Moreover, Georgia EPD has indicated that BACT for coal fired plants is at least four times the 0.07 lbs/MMBtu limit. See Ex. 7 at 2. These substantial results in the NOx emissions rates would result in substantial results in the NOx mass emissions. Therefore, the emissions "reductions" created by Conditions 3.2.6, 3.2.7 and 3.2.8 of Plant Bowen's and Plant Scherer's Title V permit amendments are not surplus as they are required by NSR and NSPS. EPA should therefore object to the permit because it does not comply with the applicable requirement that offsets be surplus.

Georgia EPD responded to this comment by stating that the claims that NSR and NSPS are applicable to Plants Bowen and Scherer are mere allegations. While this may be true for Georgia EPD because it has refused to investigate this matter, EPA has determined that NSR and NSPS are applicable. This determination is the basis upon which EPA could issue its Notice of Violations and file its complaint. To the extent that the permitting staff is not familiar with this information, Petitioners hereby incorporate by reference all of the information upon which EPA based its decision to file the Notice of Violations and complaint as well as information uncovered in discovery in the on going lawsuit. EPA would

need to articulate a rational basis for taking a position in responding to this petition contrary to the position EPA took when it issued the Notice of Violations and filed its complaint. However, since there is no rational basis to take a contrary position, EPA should object to the Title V permit amendments.

VI. CONCLUSION

For the reasons explained above, Petitioners request that EPA object to the Title V Amendments for the seven facilities.

Respectfully Submitted,

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Dated: November 18, 2002

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EXHIBIT 1

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VIA CERTIFIED MAIL AND E-MAIL

June 17, 2002

Mr. James P. Johnston, P.E.
Program Manager
Stationary Source Permitting Program
Air Protection Branch / Environmental Protection Division
Georgia Department of Natural Resources
4244 International Parkway, Suite 120
Atlanta, GA 30354

RE: Bowen Steam-Electric Generating Plant
Title V Permit Amendment 4911-015-0011-V-01-3

Branch Steam-Electric Generating Plant
4911-237-0008-V-01-3

Hammond Steam-Electric Generating Plant
4911-115-0003-V-01-3

McDonough-Atkinson Steam-Electric Generating Plant
4911-067-0003-V-01-3

Scherer Steam-Electric Generating Plant
4911-207-0008-V-01-2

Wansley Steam-Electric Generating Plant
4911-149-0001-V-01-6

Yates Steam-Electric Generating Plant
4911-077-0001-V-01-3

Dear Mr. Johnston:

On behalf of the Sierra Club and the Georgia Public Interest Research Group, I am writing to submit comments on the above referenced permit amendments. These comments will specifically address Plant Bowen's permit amendment but are intended to apply to all seven plants, unless otherwise specifically noted.

As you should be aware, the Metro-Atlanta area has not been in compliance with the health based national ambient air quality standard for ground level ozone (smog) for over two decades. This year, we have already suffered more violations of the one-hour ozone standard than we suffered last year. This means that millions of Georgians are subjected to air that puts them at risk of developing birth defects, asthma and other respiratory illnesses and people with existing respiratory illnesses are consistently subjected to air that can exacerbate these illnesses such as by triggering asthma attacks for people who already have asthma. The Georgia Board of Natural Resources has worked hard to try to address the smog problem by creating new regulations that are part of the Metro-Atlanta Attainment State Implementation Plan (SIP). However, these regulations only work if properly implemented by the Georgia Environmental Protection Division (EPD). The proposed permit amendments contain several easily corrected flaws that allow Georgia Power's coal fired power plants to continue to endanger the health and well being of the people of the 13 county Metro-Atlanta nonattainment area as well as risk additional counties being designated as nonattainment. We ask you to fix these permit shortcomings so that the people of Georgia, and especially the children and elders, can breath easy. Specifically, our concerns are:

1) THE EMISSIONS REDUCTIONS CREATED IN CONDITIONS 3.2.6, 3.2.7, AND 3.2.8 ARE NOT SURPLUS

Conditions 3.2.6, 3.2.7 and 3.2.8 claim to create an emissions cap that will create offsets that can be used to meet the offset requirement for new units. This is not so for several reasons.

To begin with, these reductions will be required under the NOx SIP Call. Emission reductions cannot be considered surplus and used for emissions offset credits if they are required by some other regulation or law. The NOx SIP Call will place a limit on all EGUs, including Georgia Power's seven plants, of 29,416 tons per ozone season. However, Georgia Power is claiming that a reduction from 35,362 tons per ozone season is a surplus reduction. Even if Georgia created a NOx SIP rule that allocated all of its allowance to Georgia Power's seven plants, the seven plants would still be required by the NOx SIP call to reduce its pollution to less than 29,416 tons per ozone season. Therefore, in order for reductions to be considered surplus, they must be reductions below 29,416. In other words, the baseline can be no more than 29,416 if Georgia Power wants to consider these reductions as surplus.

Furthermore, the reductions achieved at Plants Bowen and Scherer are not surplus because the current emissions are in violation of the New Source Review (NSR) and New Source Performance Standards (NSPS) requirements of the Clean Air Act. *See United States v. Georgia Power*, 99CV2859-JEC (N.D.Ga. 1999). Again, in order for emissions to be surplus, they must not be required by other regulations or laws. Therefore, the emissions "reductions" created by Conditions 3.2.6, 3.2.7 and 3.2.8 are not surplus as they are required by NSR and NSPS.

Finally, the narrative notes that the EPD used the seven plant averaging plan rather than the more stringent five plant averaging plan to determine the baseline. EPD should use the more stringent five plant averaging plan for determining the baseline for those 5 plants and then the seven plant averaging plan for the other two plants.[1]

2) CONDITION 3.4.11 AND 3.4.12 DO NOT MAKE IT CLEAR WHAT TRIGGERS THE ALTERNATIVE EMISSION LIMIT.

From reviewing Conditions 3.4.11 and 3.4.12, it is not clear if one violation of the alternative emission limits in 3.4.7 - 3.4.10 during any ozone season requires an analysis of all affected units under the SIP emission limit for the rest of the ozone season or just one 30 day reporting period. In order for this Conditions 3.4.11 and 3.4.12 to be enforceable as a practical matter, EPD should clarify whether one violation of Conditions 3.4.7 - 3.4.10 triggers the SIP emission limit for the whole ozone season or just one 30 day period.

3) THE PERMIT DOES NOT CONTAIN MONITORING AND REPORTING FOR THE ALTERNATIVE EMISSION LIMIT

The permit does not contain monitoring and reporting for the SIP emission limit contained in 3.4.11 and 3.4.12. While the permit does contain a require for CEMS for NOx, the permit needs to have the permittee monitor the heat input as well as other factors to record the emissions in pounds per MMBtu. Further, the permit needs to require that the permittee report the heat input and emissions in pounds per MMBtu for all seven plants in the seven plant averaging plan in the biannual report.

4) THE EMISSION LIMIT ON THE CTs AT PLANT BOWEN NEEDS TO CONTAIN AN AVERAGING TIME.

Condition 3.4.13 contains a limit of 50 ppm NOx for the combustion turbines at Plant Bowen. However, this condition does not contain an averaging time. Without an averaging time, the 50 ppm limit is meaningless. We would suggestion that EPD consider a one hour averaging time but in no event should the averaging time be greater than three hours.

5) THE PERMIT NEEDS TO INCLUDE A REQUIREMENT TO USE CEMS TO MONITOR FOR THE NOx LIMIT IN CONDITION 3.4.13.

Condition 4.2.2 requires a one time performance test of the combustion turbines at Plant Bowen to demonstrate compliance with the NOx limit in Condition 3.4.13. However, this performance test will not demonstrate compliance because it does not

[1] In the event that EPD does issue the permit, it should clearly state that Georgia Power cannot count the reductions that it makes to comply with the permit as early reduction credits for the NOx SIP Call.

require testing a various loads. NOx emissions tend to be significantly higher in combustion turbines at lower loads.

In addition, the monitoring of average temperate, as required by Condition 5.2.3(j), is not an adequate indication of continuous compliance with the NOx limit. NOx emissions can be influenced by a variety of factors beyond average temperature at the inlet, such as weather conditions, fuel being used and load. Rather than requiring an one time performance test, the permit should require a CEMS for monitoring NOx emissions from the CTs. In addition, the permit should require the permittee to report all CEMS data in its biannual reports.

6) THE PERMIT SHOULD REQUIRE GEORGIA POWER TO RECORD MASS EMISSIONS MONITORED BY THE NOx CEMS

In order to properly monitor and report mass emission limits, Condition 5.2.1.b should include a requirement that the CEMS record the NOx emissions in mass (pounds) as well as ppm.

7) THE PERMIT MUST REQUIRE THE PERMITTEE TO SUBMIT ALL MONITORING INFORMATION TO EPD.

According to 40 CFR § 70.6(a)(3)(iii)(A) and 42 U.S.C. § 7661(c)(a), permits issued by state agencies must require the permittee to submit reports containing all monitoring data at least every 6 months. Although this permit may require the reporting of all excess emissions, exceedances, and excursions, this reporting of such deviations is in fact required by § 70.6(a)(3)(iii)(**B**), whereas § 70.6(a)(3)(iii)(**A**) requires submitting records from *all* monitoring. However, there is other monitoring such as monitoring for NOx required by Conditions 5.3.10 and 5.3.11 for which there is no requirement that the permittee report the monitoring information.

The fact that the permittee has to report violations is not enough. The public needs to be able to review the "raw data," as often times regulated entities make mistakes in calculating violations. The Clean Water Act's NPDES program provides a good example. Under the NPDES program, permittees are required to report the raw data as well as violations. Often times, permittees submit monitoring reports that do not have the violations "box' checked. However, a review of the raw data shows that there is indeed a violation. This problem is equally likely to appear under the Title V program. Therefore, consistent with the plain language of the regulation and law, the permit should require Georgia Power to report all monitoring including, but not limited to, the monitoring found in Conditions 5.3.10 and 5.3.11.

8) THE PERMIT CANNOT LIMIT THE USE OF CREDIBLE EVIDENCE IN AN ENFORCEMENT ACTION.

As emphasized by the United States Environmental Protection Agency's (EPA) Credible Evidence Rule, 62 FR 8314 (Feb. 24, 1997), the Clean Air Act (CAA) allows the public,

EPD, EPA, and the regulated facility to rely upon any credible evidence to demonstrate violations of or compliance with the terms and conditions of a Title V operating permit. Specifically, EPA revised 40 CFR § 51.212, 51.12. 52.30, 60.11 and 61.12 to "make clear that enforcement authorities can prosecute actions based exclusively on any credible evidence, without the need to rely on any data from a particular reference test" [62 FR at 8316]. EPD must ensure that no permit purports to limit the use of credible evidence. Moreover, the permit should include standard language stating that all credible evidence may be used.

A. EPD Must Modify Statements that Purport to Limit Credible Evidence.

EPD must ensure that its Title V permits contain no language that could be interpreted to limit credible evidence. For example, condition 4.1.3 in the Facility's permit states that "[t]he methods for the determination of compliance with emission limits listed under Sections 3.2, 3.3, 3.4 and 3.5 which pertain to the emission units listed in Section 3.1 are as follows." One could read this provision to stand for the proposition that when a government agency or member of the public takes an enforcement action for a permittee violating its permit, the enforcer can only rely on information from the methods of determination listed in the permit. This position is directly contrary to the Clean Air Act requirements in CAA §§ 113(a), 113(e)(1) and 40 CFR § 51.212, 51.12. 52.30, 60.11 and 61.12, which allow anyone taking an enforcement action to rely on any credible evidence. Therefore, Section 4.1.3 should be modified to allow for the determination of noncompliance by any credible evidence.

B. EPD Should Include Standard Language in the Permit that Explicitly States that Anyone Can Use Any Credible Evidence

The permit does not affirmatively state that any credible evidence may be used in an enforcement action. EPA supports the inclusion of credible evidence language in all Title V permits. As explained by the Acting Chief of US EPA's Air Programs branch [letter from Cheryl L. Newton, Acting Chief, Air Programs Branch, EPA, to Robert F. Hodanbosi, Chief, Division of Air Pollution Control, Ohio Environmental Protection Agency, dated October 30, 1998]:

> It is the United States Environmental Protections Agency's position that the general language addressing the use of credible evidence is necessary to make it clear that despite any other language contained in the permit, credible evidence can be used to show compliance or noncompliance with applicable requirements. . . . [A] regulated entity could construe the language to mean that the methods for demonstrating compliance specified in the permit are the only methods admissible to demonstrate violation of the permit terms. It is important that Title V permits not lend themselves to this improper construction.

In fact, EPA apparently sent a letter in May 1998 specifically directing EPD to amend its SIP to include language clarifying that any credible evidence may be used. Nevertheless, while more than three and a half years have elapsed since EPA's request, the permit does not contain the necessary language.

While anyone may rely on *all* credible evidence regardless of whether this condition appears in the permit, EPD should include credible evidence language in the permits and permit template to make the point clear. Specifically, EPA has recommended that the following language be included in all Title V permits [letter from Stephen Rothblatt, Acting Director, Air and Radiation Division, US EPA, to Paul Deubenetzky, Indiana Department of Environmental Management, dated July 28, 1998]:

> Notwithstanding the conditions of this permit that state specific methods that may be used to assess compliance or noncompliance with applicable requirements, other credible evidence may be used to demonstrate compliance or noncompliance.

We request that EPD include this provision in the permit to clarify the acceptability of any credible evidence to demonstrate noncompliance with permit requirements.

9) THE PERMIT AMENDMENTS SHOULD CONTAIN A SHORTER AVERAGING TIME FOR ALL THE NOx LIMITS.

Conditions 3.4.7 through 3.4.12 all contain 30-day rolling averages. While this 30-day rolling average comes from the SIP, EPD should use its power under Rule 391-3-1-.02(2)(a)3(i) and (ii) to lower the averaging time to three hours. As noted above, the Metro-Atlanta area has been in noncompliance with the one hour ozone NAAQS for over two decades. Macon and Athens also are in violation of this NAAQS. Moreover, most of North Georgia is out of compliance with the eight hour ozone NAAQS. This means that the Director has failed and continues to fail to safeguard the public health, safety and welfare of the people of the State of Georgia from ozone. It is unlikely that a NOx emissions limit with a thirty day averaging time will bring Georgia back into compliance with the NAAQS and thus safeguard the public health, safety and welfare of the people of the State of Georgia. Rather, more likely is Georgia Power will continue to emit high levels of NOx on hot, sunny days when our ground level ozone problem is worse and emit lower levels on other days when emission reductions are not as critical. EPD can reverse its absymal record of failing the people of the State of Georgia by reducing the averaging time so that NOx emissions are reduced on days when it is most important; hot and sunny days.

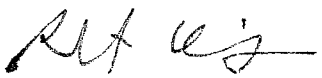
10) HOURS WHEN THE FACILIITES ARE NOT OPERATING SHOULD NOT COUNT TOWARDS THE AVERAGES.

Conditions 5.3.12.b and 5.3.12.e, as currently written, can be interpreted to allow Georgia Power to include hours that it is not operating into its averages to determine

compliance with concentration based emission standards. EPD should re-write these conditions to make clear that hours when a particular unit is not operating are not valid hours and thus should not be included in the average used to determine compliance with the concentration based emission standards.

In conclusion, we thank you for the opportunity to comment and sincerely hope that EPD will take the steps it needs to finally protect the people of Georgia from harmful air pollution.

Sincerely,



Robert Ukeiley
Counsel for Sierra Club and Georgia PIRG

Cc: Ronald Methier

EXHIBIT 2

U.S. Environmental Protection Agency

EPA Region 4 Proposed Title V Permits

Serving Alabama, Florida, Georgia, Kentucky, Mississippi, North Carolina, South Carolina, Tennessee

Georgia Proposed Title V Permits

Air Permitting Issues
ALABAMA
FLORIDA
GEORGIA
KENTUCKY
MISSISSIPPI
NORTH CAROLINA
SOUTH CAROLINA
TENNESSEE

Title V Permits

State	Source Name	PA Permit Number	Proposed Permit Received	Petition Deadline
GA	Golden Foundry & Machine Company	3321-215-0002-V-01-0	07/20/2002	11/02/2002
GA	Georgia Power Company - Plant Bowen	4911-015-0011-V-01-3	08/05/2002	11/18/2002
GA	Georgia Power Company - Plant McDonough/Atkinson	4911-067-0003-V-01-3	08/05/2002	11/18/2002
GA	Georgia Power Company - Plant Yates	4911-077-0001-V-01-3	08/05/2002	11/18/2002
GA	Georgia Power Company - Plant Hammond	4911-115-0003-V-01-3	08/05/2002	11/18/2002
GA	Georgia Power Company - Plant Wansley	4911-149-0001-V-01-6	08/05/2002	11/18/2002
GA	Georgia Power Company - Plant Scherer	4911-207-0008-V-01-2	08/05/2002	11/18/2002
GA	Georgia Power Company - Plant Branch	4911-237-0008-V-01-3	08/05/2002	11/18/2002
GA	Sewell Creek Energy Facility	4911-233-0042-V-01-0	08/13/2002	11/26/2002
GA	Georgia Pacific Corp. - Monticello Panelboard	2439-159-0011-V-01-0	08/23/2002	12/06/2002
GA	YKK National Manufacturing Center - Chestney Site	3965-021-0176-V-01-0	09/06/2002	12/20/2002
GA	Inland Paperboard/Packaging, Inc. - Rome Mill	2631-115-021-V-01-1	09/09/2002	12/23/2002
GA	WesTek, Inc. - Martha Mills	2296-293-0003-V-01-	09/13/2002	12/27/2002

		1		
GA	Weyerhaeuser - Flint River Operations	2631-193-0013-V-01-0	09/13/2002	12/27/2002
GA	Cessna Aircraft Company	3728-215-0174-V-03-0	09/23/2002	01/06/2003
GA	Dan River, Inc. - Bibb Engineered Products Div.	2296-217-0018-V-02-1	09/28/2002	01/11/2003
GA	Shaw Industries - Plant No. 81	2273-313-0001-V-01-1	10/06/2002	01/19/2003
GA	SP Newsprint Company	2621-175-0004-V-01-0	10/08/2002	01/21/2003
GA	Pratt Industries, Inc. - Visy Paper	2631-247-0037-V-01-0	10/08/2002	01/21/2003
GA	Pratt Industries, Inc. - Jett Corr	2679-247-0047-V-02-0	10/08/2002	01/21/2003
GA	Hercules Incorporated - Brunswick	2861-127-0002-V-02-0	10/10/2002	01/23/2003
GA	Southern Natural Gas Company - Thomaston Station	4922-293-0025-V-01-3	10/10/2002	01/23/2003
GA	Hollingsworth & Vose Company	2621-235-0008-V-01-1	10/11/2002	01/24/2003
GA	Georgia Pacific Corp. - Monticello Plywood	2436-159-0009-V-01-0	10/17/2002	01/30/2003
GA	Georgia Pacific Corporation - Monticello MDF	2439-159-0012-V-01-0	10/18/2002	01/31/2003
GA	ALCAN Aluminum Corporation	3341-133-0001-V-01-0	10/18/2002	01/31/2003
GA	West Georgia Generating Facility L.L.C.	4911-293-0027-V-01-0	10/22/2002	02/04/2003
GA	YKK National Mfg. Center - Ocumulgee Sit	3965-021-0078-V-01-0	10/25/2002	02/07/2003
GA	J.M. Huber - Marble Hill Plant	1455-277-0011-V-01-0	10/30/2002	02/12/2003

For information about the contents of this page please contact Art Hofmeister.

Last updated on Tuesday, November 5th, 2002
URL: http://www.epa.gov/region4/air/permits/Georgia.htm

EXHIBIT 3

Department of Natural Resources
ENVIRONMENTAL PROTECTION DIVISION
270 WASHINGTON STREET, S.W.
ATLANTA, GEORGIA 30334

JOE D. TANNER
Commissioner

J. LEONARD LEDBETTER
Division Director

January 16, 1979

Mr. John C. White
Regional Administrator
Environmental Protection Agency
Region IV
345 Courtland Street
Atlanta, Georgia 30308

Dear John:

This letter is to transmit ten copies of the revisions to Georgia's State Implementation Plan to attain and maintain compliance with ambient air standards. The Plan was subjected to a public hearing on December 20th, following a 30 day prior notice to the general public as required in Federal Regulations for Implementation Plans. I am submitting the Plan to you under authority granted to me in Section 6 of the Georgia Air Quality Act of 1978.

Due to vehicle related pollution problems in the Atlanta and Columbus areas, we require an extension to 1987 to achieve compliance with the ozone and carbon monoxide air standards in Atlanta and the ozone standard in Columbus. I request that such an extension be granted to us. The need for this extension is indicated in the Plan.

Based upon the analyses and information in the Plan, I request that EPA redefine the attainment status and boundaries of the areas previously designated nonattainment with the suspended particulate air standard in Sandersville and Savannah. The Plan indicates the adjustments deemed appropriate.

The quality of the comments and the procedure EPA elected to use in presenting them during our public hearing on December 20th are of grave concern to me. I considered the EPA comments detrimental to your program and ours. We have been in contact with your staff to determine exactly what is necessary to more fully explain and document the conclusions in the Plan and answer the comments that were provided to us during the hearing. Many of the issues raised were unnecessary and unrealistic. In addition to the SIP, we are enclosing some additional information in response to the EPA comments.

Due to the importance of having our Plan approved and the effects upon federal funding and permitting of major sources in and around nonattainment areas, I

request EPA's prompt review and action on the Plan submittal. If we may assist in this regard in any manner, do not hesitate to contact me.

Sincerely,

Leonard

J. Leonard Ledbetter
Director

JLL:rcr

c: Governor George Busbee

EXHIBIT 4

American Journal of Epidemiology

Vol. 155, No. 1
Printed in U.S.A.

Ambient Air Pollution and Risk of Birth Defects in Southern California

Beate Ritz,[1,2] Fei Yu,[3] Scott Fruin,[4,5] Guadalupe Chapa,[4] Gary M. Shaw,[6] and John A. Harris[6]

The authors evaluated the effect of air pollution on the occurrence of birth defects ascertained by the California Birth Defects Monitoring Program in neonates and fetuses delivered in southern California in 1987–1993. By using measurements from ambient monitoring stations of carbon monoxide (CO), nitrogen dioxide, ozone, and particulate matter <10 μm in aerodynamic diameter, they calculated average monthly exposure estimates for each pregnancy. Conventional, polytomous, and hierarchical logistic regression was used to estimate odds ratios for subgroups of cardiac and orofacial defects. Odds ratios for cardiac ventricular septal defects increased in a dose-response fashion with increasing second-month CO exposure (odds ratio $(OR)_{2nd\ quartile}$ CO = 1.62, 95% confidence interval (CI): 1.05, 2.48; $OR_{3rd\ quartile}$ CO = 2.09, 95% CI: 1.19, 3.67; $OR_{4th\ quartile}$ CO = 2.95, 95% CI: 1.44, 6.05). Similarly, risks for aortic artery and valve defects, pulmonary artery and valve anomalies, and conotruncal defects increased with second-month ozone exposure. The study was inconclusive for other air pollutants. The authors' results are supported by the specificity of the timing of the effect and some evidence from animal data; however, this is the first known study to link ambient air pollution during a vulnerable window of development to human malformations. Confirmation by further studies is needed. *Am J Epidemiol* 2002;155:17–25.

abnormalities; air pollution; carbon monoxide; cleft lip; cleft palate; environment and public health; heart defects, congenital; ozone

Recently, studies conducted in different countries such as China, the Czech Republic, Brazil, Mexico, and the United States related ambient air pollution to adverse birth outcomes, specifically low birth weight, intrauterine growth retardation, preterm birth, and fetal mortality (1–9). Our previous studies indicated that exposure to high concentrations of carbon monoxide during the last trimester of pregnancy may increase the risk of being of low weight for term birth and that exposure to carbon monoxide and particulate matter <10 μm in aerodynamic diameter (PM_{10}) either shortly after conception or before birth may trigger preterm birth (10, 11). Risks of several common birth defects including neural tube defects, oral clefts, and cardiovascular defects may be influenced by exposure to environmental contaminants (12). However, few epidemiologic studies have examined whether ambient air pollutants affect such risks.

Mechanistically, air pollutants could be involved in the etiology of birth defects via hemodynamic, anoxic events; oxidative stress; and toxicity to certain cell populations during development. Ozone and carbon monoxide are toxic in the developing rat and produce skeletal malformations in animals (13–15). Maternal exposure to low levels of nitrogen dioxide has produced deficits in neuromuscular coordination in newborn mice (16); in humans, elevated exposure to oxidized nitrogen has been associated with poor birth outcomes such as low birth weight (17). Components of particulates such as metals or organic compounds could be fetotoxic. For example, PM_{10} has been implicated as a risk factor for infant mortality and preterm birth (7, 9, 11). However, no known animal or human studies have examined the teratogenic potential of urban air particulates.

Since California has both a population-based birth defect registry and an extensive air pollution monitoring network, we investigated whether maternal exposures to air pollution were associated with elevated birth defect risks in a cohort of southern California infants and fetuses delivered between 1987 and 1993. Vehicular traffic is the major source of air pollution in the metropolitan area of southern California and is responsible for producing carbon monoxide, nitrogen dioxide, fine components of PM_{10}, and ozone.

MATERIALS AND METHODS

Subjects and outcome

Data on birth defects were collected by the California Birth Defects Monitoring Program (CBDMP) for four counties and represented births in July 1990–July 1993 for Los Angeles,

Received for publication January 5, 2001, and accepted for publication July 9, 2001.

Abbreviations: CBDMP, California Birth Defects Monitoring Program; CI, confidence interval; OR, odds ratio; PM_{10}, particulate matter <10 μm in aerodynamic diameter.

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[3] Department of Biostatistics, School of Public Health, University of California, Los Angeles, Los Angeles, CA.
[4] Department of Environmental Health Sciences, School of Public Health, University of California, Los Angeles, Los Angeles, CA.
[5] California Air Resources Board, Sacramento, CA.
[6] California Birth Defects Monitoring Program, Oakland, CA.
Correspondence to Dr. Beate Ritz, Department of Epidemiology, School of Public Health, University of California, Los Angeles, P.O. Box 951772, Los Angeles, CA 90095-1772 (e-mail: britz@ucla.edu).

1989 for Riverside, 1988–1989 for San Bernardino, and 1987–1989 for Orange counties. CBDMP staff actively review medical and genetics center records to ascertain cases in these surveillance areas (18). For this study, eligible cases were all liveborn infants and fetal deaths diagnosed between 20 weeks of gestation and 1 year after birth with isolated, multiple, syndromic, or chromosomal cardiac or orofacial cleft defects who 1) could be matched to California birth or fetal death registry data to obtain gestational age and zip code of residency at birth and 2) lived within 10 miles (16 km) of an air monitoring station (84 percent of all cases, of whom 78 percent were born in Los Angeles County).

Originally, we grouped isolated cardiac defects into eight diagnostic and anatomic subcategories, but since we observed too few cases in two categories to allow modeling of pollutant effects (tricuspid and Epstein anomalies (17 with complete data) and hypoplastic right heart and common ventricle anomalies (13 with complete data)), this paper presents results for six groups only: 1) aortic defects; 2) defects of the atrium and atrium septum; 3) endocardial and mitral valve defects; 4) pulmonary artery and valve defects; 5) conotruncal defects including tetralogy of Fallot, transposition of great vessels, truncus arteriosus communis, double outlet right ventricle, and aorticopulmonary window; and 6) ventricular septal defects not included in the conotruncal category. All cardiac defects were confirmed by autopsy or by surgical reports, catheterization, or echocardiogram. We divided orofacial clefts into isolated cleft palate and isolated cleft lip with or without cleft palate and examined separately all malformations attributed to a syndrome, chromosomal defects, and multiple defects, that is, all children diagnosed with more than one major anomaly. In all, we created 11 malformation groups for analyses (table 1).

Infants and fetuses were eligible as controls if 1) they were born during the same period in which the CBDMP was active in each county, they were born within at least 10 miles of an air monitoring station in a zip code area for which at least one eligible case was reported, and none of the gestational age information was missing on their birth or death certificates (86 percent of all eligible controls); and 2) they had not been diagnosed with a birth defect by age 1 year. Of the 754,030 infants and fetuses eligible as controls, we randomly selected 10,649 whose covariate information was complete (9,357 after excluding those for whom information on maternal education not recorded prior to 1989 was missing) to achieve a case-control ratio of approximately 1:10 for defect-specific analyses. Additional cases and controls were excluded from multivariate analyses because first-trimester data for one of the four air pollutants examined was missing. Birth and fetal death certificates were our only sources of risk-factor information other than air pollution measurements.

Exposure assessment

We used ambient air monitoring data for carbon monoxide, nitrogen dioxide, ozone, and PM_{10} collected by the South Coast Air Quality Management District at 30 stations between 1987 and 1993 to estimate exposure during pregnancy, in general relying on the station nearest to the residential zip code reported on birth or fetal death certificates. However, while 22 stations collected carbon monoxide and nitrogen dioxide data, and 27 collected ozone data, only 11 were equipped with PM_{10} samplers. Overall, 23 stations collected data for at least three pollutants, but no more than 10 stations collected data simultaneously for all four pollutants. In general, stations measuring all gaseous pollutants were located predominantly in the western and coastal areas of the Southern California Air Basin, while PM_{10} samplers were concentrated in the eastern and inland areas. Thus, there was little overlap between stations monitoring for the three gaseous pollutants and those monitoring for PM_{10}. Since particulate and gaseous pollutant measures were less often collected simultaneously (e.g., carbon monoxide and PM_{10} overlapped at 11 stations only), we had to rely on stations farther removed from a residence to estimate PM_{10} exposures. A member of our research team (S. F.) manually assigned to each zip code of maternal residence the most relevant monitoring station according to distance, topography, major wind direction, and air flow in the Southern California Air Basin.

By using the recorded birth or death date and gestational age at either date, we averaged air pollution measured at the assigned ambient station over each fetus's first, second, and third month of gestation and, in addition, its second and third trimester and a 3-month period prior to conception. For these calculations, 24 hourly measurements were available for the three gaseous pollutants, but, for PM_{10}, we had to use 24-hour average measurements taken every 6 days. The relevant embryologic period for cardiac defects and orofacial defects is within the first 4–12 weeks of gestation (19, 20).

Statistical methods

The effect of ambient air pollution on birth defects was estimated by logistic regression, and, because we examined several air pollutants and birth defects, a hierarchical (two-level) regression model (a modified version of the SAS-IML program written by Witte et al. (21)) was used to adjust for multiple comparisons, as recommended by Greenland (22). The first stage of this model is a polytomous logistic regression on all 11 outcome categories; the second stage is a linear model for the parameters of the main model (second-stage model: $\beta = Z\pi + \delta$; β is the first-stage coefficient for a pollutant, Z is the matrix of second-stage covariates that predict the first-stage coefficients β, π is the vector of linear effects of the second-stage covariates (Z) on β, and δ is a vector of residual effects arising from interactions among the second-stage covariates or from covariates not in Z). The function of the second stage is to constrain the distribution of β in the first stage, that is, to shrink first-stage coefficient estimates according to some prespecified assumptions. We examined the effect of two different assumptions to define the second-stage covariates. For carbon monoxide and ozone (measured in units of ppm and pphm, respectively, but with comparable exposure ranges and effect sizes), we assumed that within the same gestational period and for all

TABLE 1. Demographic characteristics (%) of eligible children and fetuses delivered alive or dead between 1987 and 1989 in four southern California counties

	Control infants and fetuses (*n* = 10,949)	Aortic defects (*n* = 324)	Pulmonary artery and valve anomalies (*n* = 246)	Conotruncal defects (*n* = 194)	Ventricular septal defects (*n* = 313)	Atrium and atrium septum defects (*n* = 487)	Endocardial and mitral valve defects (*n* = 97)	Isolated cleft palate (*n* = 246)	Isolated cleft lip with or without palate (*n* = 530)	Multiple defects with cardiac and/or cleft defects (*n* = 254)	Chromosomal defects with cardiac and/or clefts defects (*n* = 534)	Syndromic defects with cardiac and/or clefts defects (*n* = 224)
Infant's gender												
Male	51	59	54	66	53	54	57	44	63	45	50	56
Female	49	41	46	34	47	46	43	56	37	55	50	44
No prenatal care	2	1	3	3	2	2	4	2	1	1	3	3
Multiple births	2	4	7	3	6	3	3	4	2	3	2	5
No siblings	40	38	43	40	46	41	44	49	40	39	29	41
Maternal race												
White	25	35	27	29	23	24	32	31	29	25	18	25
Hispanic	56	48	51	47	57	58	55	49	58	58	66	58
Black	10	8	13	11	10	12	6	7	5	10	7	10
Asian	6	4	5	9	7	5	4	4	6	6	5	4
Other	3	4	4	4	4	4	2	9	4	2	4	3
Maternal age (years)												
<20	12	7	15	8	14	12	11	14	9	11	7	10
20–24	27	24	21	26	24	27	26	24	32	28	16	28
25–29	29	31	29	24	27	26	30	24	29	26	20	29
30–34	21	20	22	27	21	23	23	24	20	22	23	21
≥35	11	18	13	14	13	12	10	15	10	13	34	13
Maternal education* (years)												
≤8	22	18	21	20	25	26	31	23	24	26	36	29
9–11	23	19	23	20	19	19	21	22	22	21	17	21
12	27	26	30	28	31	29	24	17	28	30	21	24
13–15	16	20	13	17	19	14	15	21	15	15	16	13
≥16	12	17	13	15	7	10	8	16	11	9	10	14
Born before 1990	21	23	24	30	24	18	23	24	26	19	19	17
Season of conception												
Summer	22	25	22	22	23	24	28	20	22	22	19	26
Fall	30	26	30	30	26	29	22	26	27	32	32	24
Winter	27	29	24	26	27	23	27	28	29	21	23	30
Spring	21	20	24	22	22	23	24	24	22	25	25	19

* Maternal education was not recorded on California birth or fetal death certificates prior to 1989; thus, the total numbers in each outcome category are 9,367 (controls), 282 (aortic), 213 (pulmonary), 156 (conotruncal), 268 (ventricular septal defect), 452 (atrium), 86 (endocardial), 215 (cleft palate), 540 (cleft lip with or without cleft palate), 199 (multiple), 476 (chromosomal), and 234 (syndromic).

outcome categories, 1) each pollutant-specific coefficient β has a common mean for all outcome categories, 2) both pollutant coefficients have the same common mean, and 3) the common mean is (close to) zero (no effect for pollutants). We used semi-Bayesian estimation and set the prior (second-stage) variance to 0.5, which corresponds to a prior that 95 percent of the uncertainty in the odds ratios for the factor effects, the exp (β), is within an $\exp(2(1.96 \times \sqrt{0.5})) = 16$-fold span such as 0.5 to 8.

We used indicator terms for quartiles of pollutant averages based on all subjects included in the analyses by period (month) of gestation, and this paper presents results for single- and multiple-pollutant models. The most influential gestational period of exposure was identified according to the strength and pattern of the observed effects and the width of the confidence intervals.

To allow the hierarchical models to converge in a reasonable amount of time with minimal loss of power, the size of the control group was limited to 3,000 randomly selected from the larger control group (note that polytomous regression point estimates and confidence limits changed minimally when more than 1,000 randomly selected controls were included). We adjusted for risk factors that could potentially confound the relation between outcomes and neighborhood air pollution levels. These factors were maternal age (<20, 20–24, 25–29, 30–34, >34 years), maternal race/ethnicity (White, Hispanic, Black, Asian, other), maternal education (<9, 9–11, 12, 13–15, >15 years), access to prenatal care (none vs. any), infant gender, decade of infant's birth (1980s vs. 1990s), parity (none vs. one or more), birth type (single vs. multiple), time since last pregnancy (>12 months), season of conception (spring, summer, fall, winter), and other air pollutants.

RESULTS

The distribution of demographic factors and potential risk factors for malformations is presented by case and control status in table 1. As expected, chromosomal defects were associated with advanced maternal age (>34 years) and somewhat with low maternal educational level and lack of prenatal care; isolated cleft lip with or without cleft palate affected a higher proportion of males.

Estimates derived from crude and covariate-adjusted models were almost identical; thus, crude effect estimates are not shown in table 1. When exposure quartiles were used, first-month carbon monoxide exposure exhibited some effects on both isolated cleft types but lacked a dose-response pattern for cleft palate, and effects were not observed consistently in single- and multiple-pollutant models (results not shown). No other pollutant showed a consistent effect on isolated orofacial clefts.

Dose-response patterns were observed for the following outcomes and pollutants: 1) second-month carbon monoxide exposure on ventricular septal defects (odds ratio $(OR)_{2nd\ quartile}$ carbon monoxide = 1.62, 95 percent confidence interval (CI): 1.05, 2.48; $OR_{3rd\ quartile}$ carbon monoxide = 2.09, 95 percent CI: 1.19, 3.67; $OR_{4th\ quartile}$ carbon monoxide = 2.95, 95 percent CI: 1.44, 6.05) (table 2) and 2) second-month ozone exposure on aortic artery and valve defects, pulmonary artery and valve anomalies, and conotruncal defects (table 2). Furthermore, the average effect sizes and patterns of second-month ozone exposure were similar for these defects and varied only slightly from single- to multiple-pollutant models or when we adjusted for other potential confounding factors. We did not observe consistently increased risks and dose-response patterns for nitrogen dioxide and PM_{10} after controlling for the effects of carbon monoxide and ozone on these cardiac defects (results not shown).

Adjustment for multiple comparisons using polytomous (table 3) or hierarchical logistic models (results not shown) suggested that the second-month trends for ozone and carbon monoxide for the four cardiac categories displayed in table 2 remained stable no matter which of three assumptions about a common mean was used (e.g., ventricular septal defects: $OR_{2nd\text{-}month\ carbon\ monoxide\ polytomous\ model} = 1.33$; 95 percent CI: 1.00, 1.78; $OR_{2nd\text{-}month\ carbon\ monoxide\ hierarchical\ model} = 1.32$; 95 percent CI: 1.00, 1.75; aortic defects: $OR_{2nd\text{-}month\ ozone\ polytomous\ model} = 1.56$; 95 percent CI: 1.16, 2.09; $OR_{2nd\text{-}month\ ozone\ hierarchical\ model} = 1.53$; 95 percent CI: 1.15, 2.03). A negative dose-response relation for third-month carbon monoxide and ozone exposures was observed for several outcome categories, including aortic and ventricular septal defects, chromosomal defects, and orofacial clefts (table 3). Other than a possible negative effect for first-trimester exposures, carbon monoxide and ozone were not associated with chromosomal, syndromic, or multiple malformations with cardiac or cleft defects (table 3).

Thus, for ozone and carbon monoxide, 1) we found a clear dose-response pattern for aortic septum and valve and ventricular septal defects and possibly for conotruncal and pulmonary artery and valve defects; 2) effects were comparable in size; and 3) increased risks were observed for exposures during the second month of pregnancy.

We found no consistent pattern of effects for any other pregnancy period (results not shown). Stratification according to maternal age or race did not suggest effect modification by these factors, yet the numbers of cases in most substrata were too small to be informative.

DISCUSSION

Although ambient air pollution has recently been linked to several adverse pregnancy outcomes (1–11), our results substantially extend the epidemiologic data on the potential relation between increases in ambient air pollutants during vulnerable pregnancy periods and congenital malformations. Compared with the few previous studies on this topic, our investigation 1) was large, 2) was population based, 3) enabled nearly complete ascertainment of cases, 4) examined vulnerable pregnancy periods, and 5) considered potential confounders. To our knowledge, the only previous epidemiologic information on this topic comes from ecologic studies conducted in Poland, the Czech Republic, and Russia, where communities with high versus low levels of ambient air toxics were found to have increased rates of heart defects (23), new mutations and multiple malformations (24–26), and infant mortality due to congenital mal-

TABLE 2. Odds ratios (95% confidence intervals)* for aortic and pulmonary artery and valve anomalies, ventricular septal defects, and conotruncal defects by average concentration quartile for carbon monoxide and ozone during the first 3 months of pregnancy, southern California, 1987–1993†

	Aortic artery and valve defects		Pulmonary artery and valve anomalies		Ventricular septal defects		Conotruncal defects	
	Single-pollutant model	Multiple-pollutant model	Single-pollutant model	Multiple-pollutant model	Single-pollutant model	Multiple-pollutant model	Single-pollutant model	Multiple-pollutant model
Carbon monoxide (ppm)‡								
No. of cases	276	241	209	187	260	234	152	123
No. of controls	9,106	7,944	9,106	7,944	9,106	7,944	9,106	7,944
1st month								
<1.14	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
1.14–<1.60	0.87 (0.69, 1.27)	0.88 (0.43, 1.07)	1.07 (0.69, 1.65)	1.16 (0.69, 1.96)	0.88 (0.58, 1.34)	1.05 (0.66, 1.68)	0.90 (0.55, 1.47)	1.10 (0.61, 2.00)
1.60–<2.47	0.90 (0.49, 1.29)	0.85 (0.36, 1.20)	1.14 (0.65, 2.01)	1.42 (0.68, 2.98)	0.77 (0.47, 1.27)	1.12 (0.60, 2.12)	0.75 (0.39, 1.45)	1.28 (0.56, 2.95)
≥2.47	0.96 (0.53, 1.73)	0.95 (0.42, 2.14)	0.96 (0.47, 1.94)	1.55 (0.59, 4.11)	0.67 (0.36, 1.23)	1.23 (0.53, 2.82)	0.79 (0.35, 1.78)	2.31 (0.76, 7.01)
2nd month								
<1.14	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
1.14–<1.57	1.10 (0.73, 1.66)	1.37 (0.85, 2.20)	1.09 (0.69, 1.73)	1.13 (0.67, 1.90)	1.52 (1.05, 2.48)	1.93 (1.00, 2.66)	0.79 (0.47, 1.32)	0.80 (0.48, 1.67)
1.57–<2.39	1.25 (0.74, 2.13)	1.58 (0.81, 3.07)	0.92 (0.50, 1.70)	0.97 (0.46, 2.05)	2.09 (1.19, 3.67)	1.97 (1.00, 3.91)	0.73 (0.36, 1.47)	0.69 (0.27, 1.73)
≥2.39	0.93 (0.47, 1.85)	1.03 (0.42, 2.53)	1.00 (0.46, 2.17)	1.09 (0.40, 2.82)	2.95 (1.44, 6.05)	2.64 (1.15, 6.09)	0.85 (0.38, 2.38)	0.86 (0.25, 2.87)
3rd month								
<1.12	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
1.12–<1.51	0.75 (0.50, 1.11)	0.73 (0.46, 1.16)	1.06 (0.66, 1.66)	1.14 (0.68, 1.91)	0.80 (0.54, 1.19)	0.77 (0.49, 1.22)	1.14 (0.70, 1.85)	1.31 (0.71, 2.42)
1.51–<2.27	0.80 (0.56, 1.44)	0.76 (0.40, 1.42)	1.18 (0.69, 2.02)	1.12 (0.56, 2.25)	0.58 (0.34, 0.93)	0.54 (0.29, 1.02)	0.62 (0.31, 1.22)	0.83 (0.34, 2.03)
≥2.27	1.27 (0.71, 2.30)	1.00 (0.45, 2.23)	1.03 (0.52, 2.05)	0.73 (0.29, 1.87)	0.55 (0.30, 1.01)	0.70 (0.31, 1.58)	0.91 (0.41, 2.05)	1.97 (0.62, 6.27)
Ozone (pphm)‡								
No. of cases	274	241	208	187	261	234	151	123
No. of controls	9,049	7,944	8,049	7,944	9,049	7,944	9,049	7,944
1st month								
<1.06	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
1.06–<1.94	0.99 (0.66, 1.49)	1.16 (0.72, 1.85)	1.16 (0.74, 1.83)	1.06 (0.63, 1.78)	1.00 (0.66, 1.52)	0.85 (0.53, 1.36)	0.97 (0.56, 1.70)	0.99 (0.53, 1.86)
1.94–<2.84	1.05 (0.61, 1.82)	1.30 (0.67, 2.51)	0.88 (0.47, 1.65)	0.81 (0.39, 1.68)	1.81 (0.95, 2.74)	1.38 (0.73, 2.59)	1.11 (0.54, 2.28)	1.40 (0.58, 3.32)
≥2.84	0.81 (0.42, 1.58)	1.02 (0.47, 2.23)	0.95 (0.44, 2.02)	0.87 (0.36, 2.12)	1.48 (0.76, 2.87)	1.29 (0.60, 2.78)	1.02 (0.43, 2.45)	1.44 (0.51, 4.10)
2nd month								
<1.07	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
1.07–<1.99	1.19 (0.71, 2.01)	0.88 (0.54, 1.78)	1.35 (0.76, 2.48)	1.48 (0.77, 2.90)	1.21 (0.73, 2.01)	1.23 (0.70, 2.16)	1.63 (0.83, 3.23)	1.83 (0.85, 3.95)
1.99–<2.86	1.68 (0.84, 3.42)	1.27 (0.56, 2.86)	1.42 (0.62, 3.23)	2.10 (0.83, 5.33)	0.94 (0.46, 1.91)	1.06 (0.49, 2.31)	1.98 (0.74, 5.31)	2.00 (0.70, 5.27)
≥2.86	2.08 (1.19, 6.05)	2.51 (0.99, 6.37)	1.59 (0.77, 5.18)	2.94 (1.00, 8.67)	1.13 (0.50, 2.54)	1.32 (0.54, 3.22)	2.50 (0.82, 7.66)	2.63 (0.75, 9.24)
3rd month								
<1.16	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
1.16–<2.05	0.67 (0.44, 1.02)	0.85 (0.49, 1.07)	0.71 (0.44, 1.15)	0.54 (0.30, 0.99)	1.14 (0.75, 1.74)	1.25 (0.75, 2.09)	1.23 (0.70, 2.18)	1.67 (0.82, 3.39)
2.05–<2.91	0.58 (0.34, 0.99)	0.63 (0.33, 1.19)	0.74 (0.40, 1.37)	0.53 (0.25, 1.13)	1.15 (0.67, 1.98)	1.49 (0.75, 2.74)	0.94 (0.45, 1.98)	1.11 (0.44, 2.80)
≥2.91	0.42 (0.22, 0.79)	0.42 (0.19, 0.91)	0.61 (0.30, 1.26)	0.58 (0.23, 1.37)	0.86 (0.44, 1.64)	1.03 (0.47, 2.27)	1.15 (0.49, 2.69)	1.39 (0.48, 4.04)

* Adjusted for the following covariates: born in the 1980s vs. 1990s, infant sex, maternal race (White, Hispanic, Black, Asian, Other), maternal age (<20, 20–24, 25–29, 30–34, >35 years), single vs. multiple birth, parity (none vs. one or more), no prenatal care, maternal education (≤ 8, 9–11, 12, 13–15, >15 years), and season of conception.
† Numbers of cases and controls vary owing to missing data for pollutants at some stations.
‡ Exposures for all 3 months of the first trimester are included in each model.

TABLE 3. Results (adjusted odds ratios (95% confidence intervals))* from polytomous logistic regression models† for carbon monoxide and ozone exposure measured continuously‡ for 11 malformation outcome categories,§ southern California, 1987–1993

	Aortic defects (n = 241)	Pulmonary valve defects (n = 185)	Conotruncal defects (n = 129)	Ventricular septal defects (n = 235)	Atrium and atrium septum defects (n = 365)	Endocardial and mitral valve defects (n = 67)
Carbon monoxide¶						
1st month	1.15 (0.89, 1.49)	1.14 (0.88, 1.53)	1.11 (0.78, 1.57)	0.97 (0.75, 1.26)	0.92 (0.75, 1.13)	0.71 (0.44, 1.15)
2nd month	0.86 (0.65, 1.14)	1.03 (0.74, 1.42)	0.98 (0.67, 1.45)	1.33 (1.00, 1.78)	1.01 (0.81, 1.28)	1.02 (0.60, 1.71)
3rd month	1.01 (0.77, 1.32)	0.94 (0.70, 1.28)	1.00 (0.69, 1.44)	0.80 (0.61, 1.05)	0.86 (0.77, 1.19)	0.91 (0.56, 1.49)
Ozone#						
1st month	0.98 (0.76, 1.27)	1.02 (0.76, 1.35)	1.07 (0.76, 1.50)	1.02 (0.79, 1.32)	1.01 (0.82, 1.24)	1.23 (0.78, 1.94)
2nd month	1.56 (1.16, 2.09)	1.34 (0.96, 1.87)	1.36 (0.91, 2.03)	1.13 (0.84, 1.52)	0.85 (0.67, 1.07)	0.81 (0.49, 1.36)
3rd month	0.70 (0.54, 0.90)	0.90 (0.68, 1.20)	0.98 (0.70, 1.36)	0.92 (0.71, 1.19)	1.01 (0.83, 1.23)	0.89 (0.57, 1.39)

	Multiple malformations with cardiac or cleft defect (n = 180)	Syndromes with cardiac or cleft defect (n = 200)	Chromosomal malformations with cardiac or cleft defect (n = 407)	Isolated cleft palate (n = 189)	Isolated cleft lip with/without palate (n = 450)
Carbon monoxide¶					
1st month	0.79 (0.59, 1.05)	0.92 (0.70, 1.21)	0.88 (0.70, 1.06)	1.12 (0.84, 1.50)	1.09 (0.90, 1.32)
2nd month	1.12 (0.81, 1.55)	1.06 (0.78, 1.43)	0.93 (0.73, 1.17)	1.06 (0.76, 1.45)	0.94 (0.76, 1.17)
3rd month	1.06 (0.78, 1.45)	0.92 (0.69, 1.22)	0.87 (0.70, 1.08)	0.71 (0.52, 0.95)	0.90 (0.74, 1.11)
Ozone#					
1st month	1.16 (0.87, 1.54)	0.76 (0.57, 1.02)	1.02 (0.84, 1.25)	1.09 (0.83, 1.45)	0.89 (0.73, 1.08)
2nd month	0.94 (0.67, 1.31)	0.93 (0.67, 1.30)	1.09 (0.86, 1.38)	0.95 (0.68, 1.32)	1.13 (0.90, 1.40)
3rd month	0.93 (0.70, 1.23)	1.10 (0.84, 1.45)	0.80 (0.66, 0.98)	1.01 (0.76, 1.33)	0.92 (0.76, 1.11)

* Adjusted for the following covariates: maternal ethnicity Hispanic, maternal age (<20, 20–29, 30–34, >35 years), parity (none vs. one or more), season of conception (winter, spring, summer, fall).

† A continuous variable for these log-linear models was created by using mean values for each quartile of exposure.

‡ Per 1 ppm carbon monoxide and per 1 pphm ozone increase.

§ Results are based on 3,000 randomly selected control children.

¶ Carbon monoxide quartile means (ppm), 1st month: 0.80, 1.34, 1.98, 3.35; 2nd month: 0.80, 1.34, 1.94, 3.29; 3rd month: 0.78, 1.31, 1.85, 3.16 (range, 0.09–7.02).

Ozone quartile means (pphm), 1st month: 0.64, 1.52, 2.39, 3.42; 2nd month: 0.64, 1.56, 2.42, 3.49; 3rd month: 0.68, 1.66, 2.48, 3.49 (range, 0.14–9.94).

formations (27). Confounding by other risk factors differentially distributed between these communities and ecologic bias could not be ruled out.

Active and passive smoking may be the exposure most comparable to air pollution in their potential to adversely affect fetal development. Active maternal smoking during pregnancy has been associated with a number of birth defects including ventricular septal defects and orofacial clefts (28–36). Prenatal exposure of the human fetus to tobacco smoke through maternal passive smoking has been linked to low birth weight (37). While teratogenicity of sidestream smoke has not been clearly demonstrated in humans, researchers have reported evidence of an unfavorable osteopathic effect of sidestream smoke on fetal development in rats (38).

Our results suggest that certain fetal heart phenotypes may be susceptible to the adverse effects of two ambient pollutants, carbon monoxide and ozone. One potential etiologic pathway may include the neural crest cell population. Normal migration and differentiation of neural crest cells are important for heart development (20). Furthermore, neural crest cells are particularly sensitive to toxic insults and respond by undergoing apoptosis, in part because they lack antioxidative stress proteins (12, 39, 40). Ozone is a very reactive molecule and a strong oxidizing agent that can generate superoxides, hydrogen peroxide, and hydroxyl radicals (41); that is, it contributes to oxidative stress.

Kavlock et al. (15) found that environmentally high exposure to ozone (>1.26 ppm) during organogenesis was embryocidal in rats, resulting in largely increased resorption of fetuses; high ozone levels also reduced skeletal ossification but showed no other obvious teratogenic effects. At lower exposure levels, ozone was observed to interact synergistically with the teratogen salicylate, enhancing fetotoxic effects in the exposed rats possibly by interfering with detoxification of the teratogen or induction of oxidative stress and vitamin E deficiency in the mother (15). Exposing rats for 1–4 days to ozone at 0.4 ppm lowered their serum retinol concentrations by about 85 percent (42), and vitamin A deprivation during development is known to cause numerous congenital defects (43). Ozone prolonged the elimination time of xenobiotics in the lungs of several animals (44), and, while enzyme levels increased in the lung following ozone exposure, liver antioxidant enzymes (superoxide dismutase and glutathione peroxidase) were concomitantly depressed (45). Thus, action of toxic compounds in the atmosphere coinciding with increased ambient ozone formation could be enhanced.

In experimental systems, carbon monoxide has been demonstrated to 1) decrease metabolization of xenobiotics such as benzo-[a]-pyrene (13); 2) interfere with metabolic and transport functions of the placenta (13); 3) have a toxic effect on the developing nervous system of rats (13); 4) produce minor skeletal malformations in mice and rabbits at relatively high doses (13); and 5) at lower doses, cause a number of malformations in a dose-dependent and synergistic manner in mice deficient in protein intake during pregnancy (46).

We observed an increased risk of several cardiac defects for second-month carbon monoxide and ozone exposures; thus, the timing of exposures is consistent with cardiac development. However, we also found a reduced risk associated with increased exposures in the third month. This observation might suggest a differential loss of certain affected pregnancies not captured by the CBDMP and may be comparable to increased fetal resorption rates observed in animal exposure studies (14). For chromosomal defects, which manifest at conception, we observed a negative association with carbon monoxide for all 3 months of the first trimester, which may suggest that these fetuses are vulnerable and more likely to die when exposed to carbon monoxide. Ascertainment bias due to prenatal diagnosis as well as selective abortion of fetuses with chromosomal defects cannot be ruled out. These speculations cannot be addressed without outcome information on all conceptions.

A large percentage of carbon monoxide, nitrogen dioxide, and the fine components of PM_{10} in the metropolitan area of southern California is produced by the same vehicular sources, and these pollutants accumulate when trapped over the city by inversion layers, especially during the colder seasons. Ozone is a secondary pollutant generated in the troposphere from the precursors nitrogen dioxide and hydrocarbons, and it follows the opposite seasonal pattern. High levels of carbon monoxide during the winter are related to average wind speed affecting dilution and dispersion of emissions, while low temperatures reduce surface vertical mixing and cause near-surface inversions to be stronger and last longer; high levels of ozone during the summer are due to the contributions of sunlight to ozone production (47). Thus, as expected, Pearson's correlation coefficients (r) for monthly air-pollutant averages during the first trimester of pregnancy showed that, for the population studied, carbon monoxide was most strongly correlated with nitrogen dioxide ($r = 0.73$), less strongly with PM_{10} ($r = 0.32$), and negatively correlated with ozone ($r = -0.72$). Furthermore, sharp carbon monoxide gradients can occur near sources such as areas with a high vehicle density, contributing to a nonhomogeneous spatial distribution of carbon monoxide in close proximity to sources such as freeways. Because of prevailing onshore wind patterns, ozone shows a west-east gradient in the Southern California Air Basin, with higher levels in the eastern and inland areas. If variations in exposure levels were mostly attributable to seasonal and not regional differences in air pollution, risk factors would also have to vary seasonally to confound the relation we observed with air pollution. However, while confounding by unmeasured seasonal factors is possible, we found that our effect estimates were stable or even strengthened when our models included a term for season of conception.

We were unable to evaluate several potential risk factors for birth defects, including maternal smoking, occupational exposures, vitamin supplement use, diet, and obesity, because they are not adequately reported on California birth certificates. However, if these factors vary seasonally and/or are correlated with socioeconomic status, we may have indirectly adjusted for them to some extent by including season of conception, maternal education, and race/ethnicity in our models.

Estimating individual average exposures during specific gestational months by relying on the ambient air monitoring station closest to the maternal residence at delivery may have resulted in exposure misclassification. Particulate measuring stations were on average located farther away from residences and may have provided the least accurate surrogate measures for personal exposure. Potential sources of exposure misclassification for all pollutants include the following: 1) residential addresses reported on birth certificates might be more indicative of the last than the first months of pregnancy (48) and 2), additional exposure misclassification might have occurred if mothers spent substantial amounts of time during pregnancy outside their residential air monitoring district, such as while working or in microenvironments with higher or lower concentrations of specific pollutants; one such high-exposure source for carbon monoxide is in a vehicle while commuting (49). In addition, differences between outdoor and indoor pollutant levels, and thus personal exposures, depend on residential air exchange rates, physical activity, and time spent at home and may have further contributed to exposure misclassification. These errors are assumed to be nondifferential with respect to case or control status. Thus, we assume that such errors would have underestimated the effects. In fact, a recent study showed that when area-wide measures of exposure to air pollution, such as those obtained from fixed-site monitoring stations, are used as proxies for personal exposures, estimates of pollutant effects are generally smaller than those based on exposure levels determined by personal sampling (50).

In conclusion, our results suggest that, in southern California, exposure to increased levels of ambient carbon monoxide during pregnancy may contribute to the occurrence of ventricular septal defects and exposure to increased levels of ozone may elevate the risk of aortic artery and valve defects, and possibly also of pulmonary artery and valve anomalies and of conotruncal defects. While our results for cardiac defects are supported by the specificity of the embryologic and exposure timing and some evidence from animal data, these initial findings need to be confirmed by further studies.

ACKNOWLEDGMENTS

This study was supported by a pilot grant (PO H07172) as part of a University of California, Los Angeles–University of Southern California National Institute of Environmental Health Sciences center grant (5P30ES70048) and pilot funds provided by the California Birth Defects Monitoring Program.

The authors thank the members of the South Coast Air Quality Management District, specifically Joe Cassmassi, for their assistance. They also thank Darin Hanson and Michelle Wilhelm (University of California, Los Angeles) for help with the air pollution data and editing.

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EXHIBIT 5

Asthma in exercising children exposed to ozone: a cohort study

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Summary

Background Little is known about the effect of exposure to air pollution during exercise or time spent outdoors on the development of asthma. We investigated the relation between newly-diagnosed asthma and team sports in a cohort of children exposed to different concentrations and mixtures of air pollutants.

Methods 3535 children with no history of asthma were recruited from schools in 12 communities in southern California and were followed up for up to 5 years. 265 children reported a new diagnosis of asthma during follow-up. We assessed risk of asthma in children playing team sports at study entry in six communities with high daytime ozone concentrations, six with lower concentrations, and in communities with high or low concentrations of nitrogen dioxide, particulate matter, and inorganic-acid vapour.

Findings In communities with high ozone concentrations, the relative risk of developing asthma in children playing three or more sports was 3·3 (95% CI 1·9–5·8), compared with children playing no sports. Sports had no effect in areas of low ozone concentration (0·8, 0·4–1·6). Time spent outside was associated with a higher incidence of asthma in areas of high ozone (1·4, 1·0–2·1), but not in areas of low ozone. Exposure to pollutants other than ozone did not alter the effect of team sports.

Interpretation Incidence of new diagnoses of asthma is associated with heavy exercise in communities with high concentrations of ozone, thus, air pollution and outdoor exercise could contribute to the development of asthma in children.

Lancet 2002; **359:** 386–91

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Introduction

Asthma is the most common chronic disease of childhood; asthma prevalence and incidence have been increasing in children in developed countries during the past few decades.[1,2] Causes for this epidemic are unknown, although changes in frequency and severity of early-life infections, diet, and exposure to indoor allergens and to indoor and outdoor air pollutants have all been linked with asthma.

Cross-sectional studies[3–5] have shown that competitive athletes have a high prevalence of asthma and exercise-induced bronchospasm or bronchial hyper-reactivity. Possible mechanisms for this association include increased inhalation of cold air, allergens, or air pollutants, increased response to respiratory infections, and increased parasympathetic tone.[3–6] Various mechanisms could be linked with sports-associated asthma. However, few epidemiological investigations have all been done, and there have been no prospective studies of asthma in competitive athletes or children playing team sports.[5]

Acute exposure to ozone and other outdoor air pollutants exacerbates asthma;[7] the chronic effects of air pollution have been less studied, but combustion-related air pollution is not thought to cause asthma.[8] However, this conclusion is based on studies in which personal exposure was measured with community air pollution monitors. The true dose of outdoor air pollutants to the lung depends on local pollutant concentrations, which may vary within a community, and on personal habits such as time spent outside and physical activity. People exercising outside should receive greater doses of outdoor pollutants to the lung than those who do not, and thus be more susceptible to any chronic effects of air pollution. Because the onset of asthma might cause athletes to reduce their levels of exercise, cross-sectional studies are not an appropriate way to measure the causal relations between exercise, air pollution, and asthma.

We postulated that children engaged in team sports in polluted communities might also be at high risk of asthma. Because the amount of time spent playing sports is an individual factor that affects exposure to ambient pollution, this approach avoids many biases of studies of air pollution that have relied on between-community comparisons of rates of asthma and other illnesses. We assessed the association of playing team sports with subsequent development of asthma during 5 years of follow-up of participants in the Southern California Children's Health Study. Study communities were selected on the basis of concentrations of ambient ozone and other pollutants.[9]

Methods

Participants

We selected 12 communities in southern California for variability of concentrations of ozone, particles with aerodynamic diameter less than 10 μm (PM_{10}), and nitrogen dioxide (NO_2).[9] In 1993, in each of the 12 communities, we recruited around 150 children aged 9–10 years, 75 aged 12–13 years, and 75 aged 15–16 years from schools in areas of the communities with stable, mainly middle-income populations. All children from targeted classrooms were invited to take part; participants completed a baseline questionnaire with help from their parents. In

early 1996, we recruited an additional cohort of around 175 children aged 9–10 years from every community. Children were followed up and interviewed yearly until 1998 (or until 1995, for children aged 15–16 years at entry).

Procedures

Children were excluded if they answered yes to "Has a doctor ever diagnosed this child as having asthma?" on the baseline questionnaire sent home to every child's parents or if a child answered yes to the question "Has a doctor ever said you had asthma?" on a questionnaire administered by an interviewer in 1993 (or in 1996, for the 1996 cohort). We ascertained history of wheezing in the baseline questionnaire with the question "Has your child's chest ever sounded wheezy or whistling, including times when he or she had a cold?" A study interviewer administered a questionnaire yearly to every child. Children were classed as having newly-diagnosed asthma in the year that they first answered yes to the question "Has a doctor ever said you had asthma?"

A question on the baseline questionnaire asked "Has your child been on any sports teams in the past 12 months?" and, if the answer was yes, "what teams?" Children were grouped into those who played no team sports, and those who played one, two, and three or more sports. The question had eight answer options, including sports with high metabolic indices, typically involving six or more times resting work expenditure (basketball, football, soccer, swimming, and tennis), low metabolic indices, typically involving less than six times resting work expenditure (baseball, softball, and volleyball), and other sports.[10] For some analyses, children who played sports were grouped into those who played at least one high activity sport and children who played no high activity sports, but at least one low activity sport.

In the baseline questionnaire we also obtained information on children's sex, age, race and ethnic origin (Hispanic, non-Hispanic white, Asian or Pacific Islander, African American, or other), history of allergies, reported time spent outdoors, current maternal smoking, history of asthma in either parent, membership of a health insurance plan, and family income. We split each cohort (1993 and 1996) into children playing more than the median time outdoors and those playing less. We classed families as having low socioeconomic status if their income was less than US$15 000 (or, if income was not reported, if the responding parent had not completed a secondary school education). We defined high socioeconomic status as family income of $100 000 or more (or, if income was not reported, by responding parent having received postgraduate training). We classed remaining families as having middle socioeconomic status. Body-mass index (BMI) was calculated from children's heights and weights at the time of the first interview of the child, and was used to divide children into quartiles for analysis.

We established air pollution monitoring stations in all 12 communities, and measured pollutant concentrations from 1994 to 1998.[9,11] Every station monitored hourly concentrations of ozone, PM_{10}, and NO_2. $PM_{2.5}$ (particulate mass less than 2·5 μm in diameter) and acid vapour were measured with 2-week integrated samplers. Yearly means were calculated from 24-h mean concentrations of ozone, PM_{10}, and NO_2; from 10:00 h to 18:00 h mean concentrations of ozone ($ozone_{10-18}$); and from a daily maximum 1-h ozone concentration. We also calculated yearly means from 2-week mean concentrations of $PM_{2.5}$ and inorganic hydrochloric and nitric acid vapour. We calculated 4-year mean concentrations (1994–97) in every community for every pollutant. We used 4-year means to rank communities because between-year variation was small,[11] and these means provided more stable estimates of exposure than yearly means. For every pollutant, we grouped the 12 communities into six with high 4-year mean concentrations and six with low concentrations. For some analyses, communities were stratified by tertiles of selected pollutants.

Statistical analyses

Before grouping into high and low pollution communities, Pearson correlation coefficients were calculated to measure the relation between different pollutants in the 12 communities. Relative risks (hazard ratios) of asthma for living in a high or low pollutant community, adjusted for ethnic origin, were evaluated for every pollutant with a multivariate proportional hazards model. We stratified baseline hazards by age and sex. We selected age groups to divide the 9–10-year-old cohort by median age at study entry, and for least overlap of this cohort with other cohorts. Age groups were: younger than 9·70 years, 9·70–11·49 years, and older than 11·49 years. To establish whether ozone had more effect than NO_2 (which was highly correlated with particulate pollutants and acid), the effect of team sports on the risk of new asthma was assessed in every pollution setting. To assess whether type of sport played

	Number playing sports*
Sex (p<0·0001)†	
Girls	837 (46%)
Boys	1097 (67%)
Age (years; p=0·06)	
Younger than 9·70	646 (55%)
9·70–11·49	647 (54%)
Older than 11·49	641 (59%)
Ethnic origin (p=0·0001)†	
Non-Hispanic white	1239 (61%)
Hispanic	481 (50%)
Black	80 (46%)
Asian	82 (45%)
Other	52 (50%)
BMI quartile (p=0·09)†	
1	486 (56%)
2	493 (57%)
3	501 (58%)
4	452 (52%)
Allergies (p=0·01)†	
No	1392 (54%)
Yes	462 (59%)
Asthma in family (p=0·29)†	
No	1560 (57%)
Yes	266 (54%)
Socioeconomic status (p<0·0001)†	
Low	308 (40%)
Medium	1221 (59%)
High	373 (69%)
Maternal smoking (p=0·01)†	
No	1729 (56%)
Yes	185 (50%)
Insurance (p<0·0001)*	
No	226 (43%)
Yes	1675 (58%)
Wheeze (p=0·25)*	
No	1488 (55%)
Yes	446 (58%)
Time outside (p<0·0001)*	
Low	840 (50%)
High	948 (62%)

*Total number of participants varies because of missing values. †p value based on χ^2 test for homogeneity. BMI=body-mass index.

Table 1: **Distribution of baseline characteristics of children by participation in team sports**

	N (incidence)*	RR (95% CI)
Number of sports played		
0	104 (0·022)	1·0
1	90 (0·026)	1·3 (1·0–1·7)
2	36 (0·021)	1·1 (0·7–1·6)
≥3	29 (0·033)	1·8 (1·2–2·8)

N=number of cases of asthma; RR=relative risk (hazard ratio), adjusted for ethnic origin, and for stratified baseline hazards by sex and age group.
*Denominator=person-years of follow-up.

Table 2: **Effect of sports on incidence of asthma diagnoses**

was relevant, models containing indicator variables for each type of sport or a linear term for total number of sports played were compared with our final model with the Akaike Information Criterion (AIC) to see whether a model with information on specific sports was better than models without such information. We also assessed effects of community, history of allergy, family history of asthma, membership of a health insurance plan, BMI, current maternal smoking, and socioeconomic status. Analyses were done with the Statistical Analysis System (version 8.1) software package.

Role of the funding source
The California Air Resources Board helped establish the air pollution monitoring network and helped collect the air pollution data from this network for use in the study.

Results

5762 (79%) of eligible children completed baseline questionnaires. 479 children were excluded because they were not at school at the time of a questionnaire administered during the entry year by an interviewer, and an additional 883 were excluded for a history of asthma. We excluded 312 children because of missing or "not known" answers to questions on wheezing, and 26 for a history of cystic fibrosis, severe chest injury, or chest surgery. 527 additional children were excluded who did not have at least 1 year of follow-up. 3535 children were included who did not have a history of asthma and who were available for follow-up, 2752 (78%) of whom had no history of wheezing. At study entry, 65 children had missing information about the number of sports played. 1934 (67%) children played sports. Only 273 (8%) of 3470 children played three or more team sports. Several factors were associated with number of team sports played (table 1). Girls were much less likely to play team sports than boys, and children in the top quartile of BMI were slightly less likely to play sports than those in other quartiles. Hispanic and non-Hispanic white children were more likely to play three or more team sports. Although family history of asthma was not associated with team sports, a child's history of allergy was associated. Children from families with low socioeconomic status and with the related characteristics of a mother who smoked and lack of health insurance, were less likely to play sports. Spending more time outside was also associated with playing sports. Children with wheeze were not less likely to play sports.

We analysed the relation between newly-diagnosed asthma and number of sports played (table 2). 265 children developed asthma, 259 of whom had provided complete information on sports. Across all communities there was a 1·8-fold increased risk (95% CI 1·2–2·8) for asthma in children who had played three or more team sports in the previous year. There was a linear trend of increasing asthma for the total of eight possible team sports played (relative risk 1·1 per team sport played, 1·0–1·3).

Table 3 shows the profile of each pollutant in high and low pollution communities. Even communities with low $ozone_{10-18}$ had high mean 4-year concentrations, up to 51 parts per billion. The high and low pollution communities were the same for NO_2, PM_{10}, $PM_{2·5}$, and acid, which was not surprising as 4-year mean concentrations of these pollutants were highly correlated across communities: from $r=0·65$ for NO_2 with PM_{10}, to 0·96 for $PM_{2·5}$ with PM_{10}. $Ozone_{10-18}$, although highly correlated with mean daily 1-h maximum ozone concentration (0·98) and with 24-h mean ozone (0·72), was not strongly correlated with the other pollutants. The highest correlation of $ozone_{10-18}$ with other pollutants was with acid (0·48).

Risk of developing asthma was not greater overall in children living in the six high pollution communities than children living in the six low pollution communities, after adjustment for stratified baseline hazards for age and sex, and for ethnic origin, irrespective of which pollutant was used to classify communities as high or low. The relative risks were 0·8 (0·6–1·0, $p=0·08$) for $ozone_{10-18}$, 0·7 (0·6–0·9) for daily maximum ozone, and 1·1 (0·9–1·4) for 24-h ozone. For NO_2, PM_{10}, $PM_{2·5}$, and acid, all of which shared the same high and low communities, the relative risk was 0·8 (0·6–1·0 $p=0·08$). Communities with high NO_2 and associated pollutants, and communities with high $ozone_{10-18}$ or daily maximum ozone were associated with a decreased risk of asthma; these associations were significant ($p<0·05$) only for daily maximum ozone.

The effect of team sports was similar in communities with high and low particulate matter (and associated pollutants, all of which gave the same high or low groupings of communities as did particulate matter). In both groups of communities there was a small increase in asthma among children playing team sports, which was largest among those playing three or more sports (table 4).

In high $ozone_{10-18}$ communities, there was a 3·3-fold increased risk of asthma in children playing three or more sports; an increase that was not seen in low $ozone_{10-18}$ communities (table 5). In high ozone communities there was a trend of increasing asthma with number of team sports played (relative risk 1·3 per sport, 1·1–1·6). There was a significant interaction between total number of sports played and ozone ($p=0·004$). In assessing interaction, we also tested models that used indicator variables for each sport or dummy variables for none, one, two, and three sports. The model that used total number of sports was found to give the best fit. In high ozone communities, risk of

	Low pollution communities (n=46)		High pollution communities (n=46)	
	Concentration (mean [SD])	Median (range)	Concentration (mean [SD])	Median (range)
Maximum 1-h ozone (ppb)	50·1 (11·0)	47·6 (37·7–67·9)	75·4 (6·8)	73·5 (69·3–87·2)
$Ozone_{10-15}$ (ppb)	40·0 (7·9)	40·7 (30·6–50·9)	59·6 (5·3)	56·9 (55·8–69·0)
24-h ozone (ppb)	25·1 (3·1)	25·1 (20·6–28·7)	38·5 (11·0)	33·1 (30·7–59·8)
PM_{10} (mg/m³)	21·6 (3·8)	20·8 (16·2–27·3)	43·3 (12·0)	39·7 (33·5–66·9)
$PM_{2·5}$ (mg/m³)	7·6 (1·0)	7·7 (6·1–8·6)	21·4 (6·0)	21·8 (13·5–30·7)
NO_2 (ppb)	10·8 (4·6)	12·1 (4·4–17·0)	29·2 (8·5)	29·5 (17·9–39·4)
Acid (ppb)	1·8 (0·7)	1·7 (0·9–2·6)	3·9 (0·7)	3·7 (3·3–4·9)

*These are the same six high and six low communities for PM_{10}, $PM_{2·5}$, NO_2, and acid, but not for other pollutants. Ppb=parts per billion; Acid=inorganic acid vapour.

Table 3: **4-year pollution concentrations in high and low pollution communities***

	Low PM communities		High PM communities	
	N (incidence)*	RR (95% CI)	N (incidence)*	RR (95% CI)
Number of sports played				
0	49 (0·023)	1·0	55 (0·021)	1·0
1	54 (0·032)	1·5 (1·0–2·2	36 (0·021)	1·1 (0·7–1·7)
2	22 (0·024)	1·2 (0·7–1·9	14 (0·018)	0·9 (0·5–1·7)
≥3	13 (0·033)	1·7 (0·9–3·2	16 (0·033)	2·0 (1·1–3·6)

PM=particulate matter; N=number of cases of asthma; RR=relative risk, adjusted for ethnic origin, and for stratified baseline hazards by sex and age group. *Denominator=person-years of follow-up.

Table 4: **Effect of number of team sports played on the risk of new asthma diagnosis in high and low PM (and other pollutant) communities**

asthma was increased for children playing at least one high activity sport, compared with no sports (1·6, 1·1–2·5), but not for children playing only a low activity sport (1·2, 0·7–2·1). In low ozone communities, the relative risk for high activity sports was 1·0 (0·7–1·4) and for low activity sports the risk was 0·9 (0·5–1·7). In models with individual sports entered as dummy variables, only tennis was significantly associated with asthma and only in high ozone communities (5·2, 1·3–20·4), but power was limited for identifying the effect of specific sports.

The overall pattern of effects of sports on asthma risk was similar in models that also included socioeconomic status, history of allergy, family history of asthma, insurance, maternal smoking, and BMI at study entry. Time spent outside was also associated with asthma in high ozone communities (1·4, 1·0–2·1), but not in low ozone communities (1·1, 0·8–1·6) in models that also included team sports. There was no significant interaction of number of sports played with history of allergy, family history of asthma, or time spent outside. However, when we compared the characteristics of the 20 children who played three or more sports in high ozone$_{10-18}$ communities with the nine children who played this number of sports in low ozone communities, three of seven of those in low ozone communities (two had missing information) had a family history of asthma, compared with none of 17 in high ozone communities (p=0·02, Fisher's exact test). In these 29 children, no other demographic or personal characteristic differed significantly between low and high ozone communities.

The effect of sports was similar in boys and girls, although the effect of playing three or more sports in high ozone communities, compared with no sports in high ozone communities, was somewhat greater in girls (4·7, 2·1–10·5) than in boys (2·5, 1·1–5·4).

Among children with no lifetime history of wheeze at study entry, the relative risk of new diagnosis of asthma in children playing three or more sports in a high ozone community was 4·4 (2·1–9·3). In children with a history of wheeze, the relative risk was 2·7 (1·1–6·4).

	Low ozone communities		High ozone communities	
	N (incidence)*	RR (95% CI)	N (incidence)*	RR (95% CI)
Number of sports played				
0	58 (0·027)	1·0	46 (0·018)	1·0
1	50 (0·033)	1·3 (0·9–1·9)	40 (0·021)	1·3 (0·8–2·0)
2	20 (0·023)	0·8 (0·5–1·4)	16 (0·020)	1·3 (0·7–2·3)
≥3	9 (0·019)	0·8 (0·4–1·6)	20 (0·050)	3·3 (1·9–5·8)

N=number of cases of asthma; RR=relative risk, adjusted for ethnic origin, and for stratified baseline hazards by sex and age group. *Denominator=person-years of follow-up.

Table 5: **Effect of number of team sports played on the risk of new asthma diagnosis in high and low ozone communities**

When ozone$_{10-18}$ was used to divide communities into tertiles, playing three or more team sports was associated with asthma only in the upper tertile (3·1, 1·8–5·5). The range of exposure across the four communities in the upper tertile was 56·8–69·0 parts per billion. Playing three team sports was associated with a small, not significant decrease in relative risk of asthma in the lowest tertile (0·7, 0·3–1·8) and in the middle tertile (0·9, 0·2–3·1). However, these estimates for the effect of team sports were based on few cases, and the models converged only if Asian, Black, and other races were combined into one category.

When the effect of sports was analysed in communities divided into combinations of high and low mean ozone$_{10-18}$ with high and low mean concentrations of other pollutants, there was no interaction between sports, ozone, and other pollutants. In communities with high ozone$_{10-18}$ and low levels of other pollutants, there was a 4·2-fold (1·6–10·7) increased risk of asthma in children playing three or more sports, compared with children who played no sports. In communities with a combination of high levels of ozone and other pollutants, there was a 3·3-fold (1·6–6·9) increased risk of asthma in children playing three or more sports. There was little effect of playing team sports in low ozone communities, irrespective of whether other pollutants were present.

Discussion

Our results show that playing multiple team sports in a high ozone environment is associated with development of physician-diagnosed asthma. The results are consistent with a large increased risk both for new-onset asthma and for exacerbation of previously undiagnosed asthma, because playing multiple sports was associated with asthma in children with no lifetime history of wheezing at baseline and children with a previous history of wheezing. The larger effect of high activity sports than low activity sports, and an independent effect of time spent outdoors, also only in high ozone communities, strengthens the inference that exposure to ozone may modify the effect of sports on the development of asthma in some children. Exercise-induced asthma by itself is unlikely to have been an explanation for these results, because asthma onset was associated with exercise only in polluted communities.

The high prevalence of asthma in competitive figure skaters might be related to NO_2 generated by ice grooming equipment.[5] However, prevalence of asthma greater than 40% has been reported in competitive cross-country skiers,[3] a group inhaling cold air, but who might not be heavily exposed to air pollution. Competitive long distance and speed runners and swimmers (especially atopic individuals) have high prevalence of asthma, bronchial hyperresponsiveness, or both, and these rates were higher in atopic individuals.[4] However, the role of atopy in sports-induced asthma is unclear. Atopy did not modify the risk of asthma associated with nordic skiing.[3] We saw no interaction between history of allergy and sports, but our indicator for allergy based on reported history might have resulted in misclassification of atopy, compared with skin testing. Our results suggest that asthmatic children playing three or more team sports were less likely to have a family history of asthma in high ozone communities than in low ozone communities. In as much as family history is suggestive of atopy, this result is in contrast with those of other studies. Although previous studies of sports and asthma have focused on competitive athletes, one other prospective population-based study has been done in Danish children.[12] Information about physical activity and team sports were not provided, but physical fitness was associated with a lower risk of subsequent development of

asthma; a finding more consistent with the reduced risk of asthma (although not significant) in low ozone communities in our study.

Experimental studies have shown the acute effects of ozone in exercising individuals.[7] Combustion-related air pollution has not been associated with asthma in many, mainly cross-sectional, studies.[8] Our cross-sectional results showed that exposure to air pollution exacerbated chronic symptoms of asthma,[13] but there was no association between asthma prevalence and air pollutants.[9] This conclusion is in accord with results from a comparison of asthma prevalence between East and West Germany; a lower rate was measured in East Germany, where pollution from burning coal was much higher.[14] However, the German communities had profound differences other than pollution, which might have confounded these ecological comparisons. Large cross-sectional studies of children in Taiwan showed associations of asthma prevalence with ozone and other pollutants[15] and with traffic-related pollutants.[16] In 24 communities in the French PAARC study,[17] asthma prevalence in adults was associated with SO_2, which the investigators suggested might have been an indicator for other pollutants, such as ozone, which was not measured. In one of the few other large prospective studies of asthma and air pollution,[18] in non-smoking adult Seventh Day Adventists in California, USA, an increased risk for new-onset of asthma was noted in communities with high ozone concentrations.

The negative association between asthma and $ozone_{10-18}$ (and daily maximum ozone and particulate and related pollutants) is not inconsistent with the large effect of playing team sports, because few children (8%) played three or more team sports. Thus, the effect of sports would not be likely to affect greatly the overall rates of asthma in high ozone communities. Nevertheless, the low rates in high pollution communities are puzzling, since it is not plausible that ozone and other combustion-related pollutants protect against asthma. Possible explanations include selection bias, for example, if parents with children with a history of asthma exacerbated by air pollution (or some similar characteristic associated both with asthma and with air pollution) moved to less-polluted communities, thus increasing the prevalence of children at high risk for developing asthma in unpolluted communities. However, the association between air pollution and asthma persisted after we adjusted for family history.

Measurement error can affect studies of air pollution and health. Because we established a network of nearby community-level monitors specifically for the Children's Health Study,[9] we improved on the assignment of individual exposure levels compared with most other such studies. However, a more accurate assessment of individual dose is essential to identify the contribution of air pollution to asthma prevalence and incidence; rates of asthma vary widely between communities and between countries, for reasons that are largely unknown, but are thought to be environmental.[19]

Heavy exercise increases ventilation rates 17-fold;[20] children playing more than two team sports might have been the more highly motivated athletes. Increased ventilation rate and oral breathing displaces pulmonary uptake of ozone to more distal sites in the lung, further increasing the effect of ambient exposure on ozone deposition in the distal airways and centriacinar region, where the largest morphological effects of ozone have been seen in work in animals.[21] Additionally, outdoor activity (independent of exercise) should be an important modifier of exposure to ambient ozone, because outdoor ozone concentrations in the 12 communities can be as much as five-times higher than indoor concentrations.[22,23] Ozone concentration between 10·00 h and 18·00 h is generally higher than at other times of day, and 10·00–18·00 h is the time period when most team sports are played outdoors in southern California. The association between incident asthma and time spent outside further supports the inference that dose of ozone might affect the pathogenesis of asthma. Furthermore, the association between sports and asthma occurred only in the top tertile of communities ranked by ozone exposure, although our study did not have sufficient statistical power to identify a threshold level of exposure at which such an effect might occur.

Participation in endurance sports or in heavy physical training can result in the recognition of exercise-induced bronchospasm,[24] which might not otherwise have been diagnosed. Exercise-induced bronchospasm is associated with the bronchial hyper-reactivity characteristic of asthma, for which exercise challenge has been proposed as a screening test. The increase in asthma with sports in our study could result from chronic exacerbation of exercise-induced bronchospasm by sports to the point that medical attention was sought and a diagnosis was made that might not have been made in more sedentary children. Because the association between sports and asthma occurred only in high ozone communities, such detection bias would imply, at the least, that exercise-induced bronchospasm was being exacerbated by air pollution, a conclusion that is consistent with other studies in which ozone has been associated with asthma exacerbation.[7] However, if the results were caused only by unmasking of pre-existing asthma by sports and air pollution, a larger effect of sports might have been expected in children with a previous lifetime history of wheezing at study entry.

In healthy people, airway reactivity is increased after 5 h of exercise—equivalent to a day of moderate to heavy work or play during exposure to 0·08 parts per million ozone.[25] Ozone also increases responses to other allergens present in ambient air. Exposure to 0·16 parts per million of ozone during light exercise increased the bronchial hyper-reactivity of children with mild asthma to house dust-mite allergen.[26] In mice, ozone concentrations as low as 0·13 parts per million increased the sensitising effect of exposure to aerosol allergens.[27] The increased pulmonary dose of ambient ozone resulting from heavy exercise, combined with exposure to outdoor and indoor allergens, is one possible mechanism for inducing new onset asthma or for exacerbating existing asthma to the point that medical attention would have been sought.

Asthma could be caused by exposure to pollutants other than ozone such as ambient this gas; people with asthma have an increased response to bronchial challenge with dust-mite allergen after exposure to this gas.[28] Although no effect of sports on asthma was seen in communities with high concentrations of pollutants other than ozone, statistical power was too low to rule out an independent association of other pollutants with development of newly-diagnosed asthma, or to identify interaction between sports, ozone, and other pollutants. Additionally, other pollutants that we did not include, such as those originating from diesel exhaust, could have resulted in the association of sports with asthma.

Study limitations include the potential for misclassification of asthma, which could be affected by access to care and differences in diagnostic practice between physicians,[29] or by poor reporting by children or parents. However, participant report of physician-diagnosed asthma has been the main criterion of asthma used in epidemiological studies of children,[19] and the validity of this approach, assessed by repeatability of

response, is good.[30] Self-report, at least in adults, reflects what physicians actually said to patients, and physician assessment of asthma has been recommended as the epidemiological gold standard for this disease.[31] Our list of sports did not include some high-activity sports such as running, which has been shown to be associated with asthma in cross-sectional studies of athletes,[4] and bicycling, which has been shown in amateur cyclists exposed recreationally to low ambient levels of ozone to result in acute decreases in lung function and increases in symptoms.[32] These exceptions might have resulted in some misclassification of team sports. However, the effect of misclassification would not have been likely to have differed with stratum of ozone exposure, and so would probably have resulted in an underestimate of a true effect of sports. Finally, variation in loss to follow-up between subgroups of children might have biased estimates of associations. However, in children aged 9–10 and 12–13 years available for follow-up at study entry, 78% were examined in either year 4 or year 5 of the study, and follow-up did not differ significantly by participation in team sports, residence in a high compared with low $ozone_{10-18}$ community, or wheeze at study entry.

We conclude that the incidence of new asthma diagnoses is associated with heavy exercise in communities with high levels of ambient ozone, and that in these conditions, air pollution and outdoor exercise might contribute to development of asthma in children.

Contributors

All authors participated in study design, analysis, interpretation, and in drafting the report.

Conflict of interest statement

None declared.

Disclaimer

The statements and conclusions in this report are those of the contractor and not necessarily those of the California Air Resources Board. The mention of commercial products, their source, or their use in connection with material reported herein is not to be construed as either an actual or implied endorsement of such products.

Acknowledgments

We thank David Bates and the rest of our External Advisory Committee: Morton Lippmann, Jonathan Samet, Frank Speizer, John Spengler, James Whittenberger, Arthur Winer, and Scott Zeger. We also thank the study field team, the 12 communities, the school principals, teachers, students, parents, Clint Taylor (California Air Resources Board), and staff at the participating air quality districts. Edward B Rappaport and Isabelo Manila provided programming support. This study was supported by the California Air Resources Board (A033–186), the National Institute of Environmental Health Science (1PO1ES0939581–02 and 5P30ES07048–05), the Environmental Protection Agency (CR824034–01–3), the National Heart, Lung and Blood Institute (1RO1HL61768), and the Hastings Foundation.

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EXHIBIT 6

Georgia's NOx SIP Call

Ron Methier
Branch Chief
Georgia EPD, Air Protection Branch
June 17, 2002

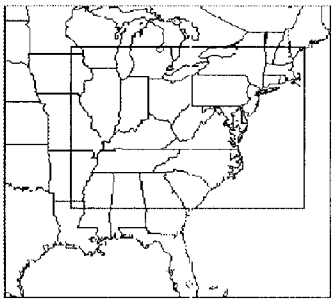
Agenda

- Why the NOx SIP?
- What is proposed?
- How will NOx SIP work?
- When?
  - Schedule
  - Public input

1990 Clean Air Act

- One-hour ozone nonattainment
- Attainment plans due 11/94
- Georgia and many other states could not show how to attain
  - Transport a problem
- Ozone Transport Assessment Group (OTAG)

OTAG Modeling Grid



OTAG Conclusions

- Little transport from SE to other regions
- Interstate transport, including across SE
- NOx and VOC controls in urban areas have impact in those areas
- NOx controls in rural areas can have a significant impact on urban areas longer distances away

Purpose of NOx SIP Call

- NOx SIP (State Implementation Plan) Call follows effort by the Ozone Transport Assessment Group to deal with regional ozone transport
- Reduce regional transport of ozone across boundaries of 21 eastern states (plus D.C.)
- Help downwind ozone nonattainment areas achieve the 1-hr ozone standard

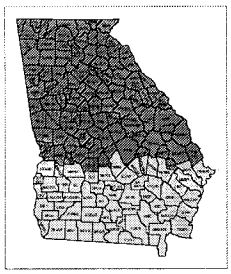
NOx SIP Requirements

- Overall reduction of NOx emissions across broad region
- One million tons of NOx reduced
- State "budgets" include all NOx sources
- EPA rule suggests how to meet these budgets, but states have flexibility in choices

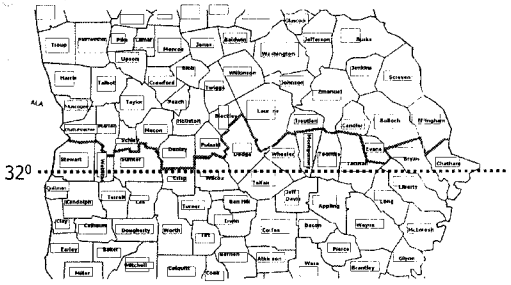
Timeline of NOx SIP Call

- Oct. 98 – NOx SIP Call issued by EPA; SIPs due Sept. 99
- May 99 – Court stayed SIP Call
- March 00 – Court upheld most of SIP Call but vacated rule for Georgia. Court agreed that south Georgia did not contribute to ozone nonattainment in other states
- February 02 – EPA proposed rule to bring northern part of Georgia into NOx SIP Call

Counties Affected by NOx SIP Call



Counties Affected by SIP Call



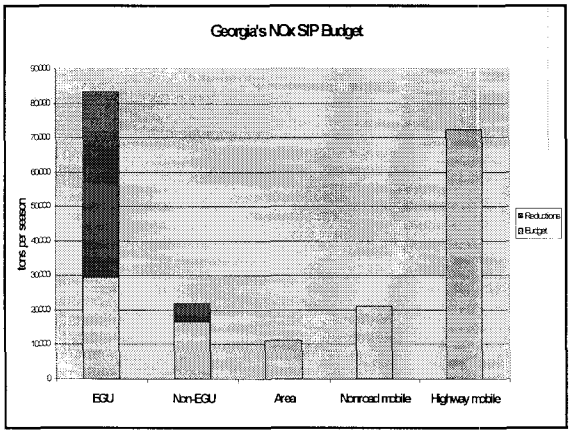
Determination of NOx Budget

- Established NOx budget for 2007 for north Georgia
- Budget based on highly cost effective NOx reductions from utilities, industrial boilers, cement kilns and stationary internal combustion (IC) engines
- Budget assumes no additional controls for mobile and area sources
- States allowed to get NOx reductions from any source categories

Georgia's NOx SIP Call Budget

(based on 82% Control of IC engines)

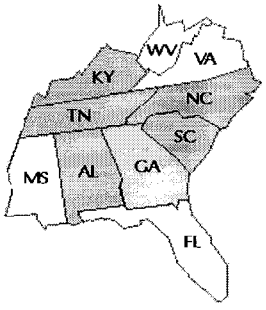
NOx SIP Call Budget Component	2007 Base (tons/season)	2007 Budget (tons/season)	Reductions Required (tons/season)
Electricity Generating Units (EGUs)	83,365	29,416	53,949
Non-EGUs	21,933	16,624	5,309
Area	11,212	11,212	0
Nonroad Mobile	21,069	21,069	0
Highway Mobile	72,335	72,335	0
Total	209,914	150,656	59,258



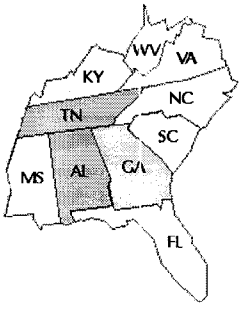
Relationship of NOx SIP Call to Atlanta Attainment SIP

- Compliance with NOx SIP Call by states impacting Georgia will help metro Atlanta achieve 1-hour ozone standard
- Neighboring states will reduce by 2004

The Five States that Impact Georgia



The Two States that Georgia Impacts



NOx SIP Call Plan vs. Atlanta Attainment SIP

- NOx SIP Controls
  - Mid-kiln tire firing at cement kilns
  - Low emission retrofits for large IC engines
  - Cap and trade for utility and industrial boilers/turbines
    - Utility boilers - 0.15 lb/ mmBtu
    - Industrial boilers – 60% NOx reduction
- Atlanta SIP Controls
  - Mid-kiln tire firing at cement kilns
  - Low emission retrofits for large IC engines
  - Coal-fired utility boilers at 7 plants limited to average of 0.20 lb/mm Btu
  - Required by May 1, 2003, two years in advance of NOx SIP compliance date

Relationship of NOx SIP Call to Atlanta Attainment SIP

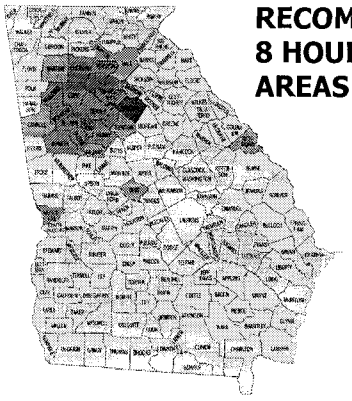
- Control measures affecting budget components not reduced in NOx SIP Call (e.g., mobile, area) can be used in development of Georgia's NOx SIP, provided reductions are real, quantifiable and enforceable

NOx Reductions Beyond Those Included in NOx SIP Call

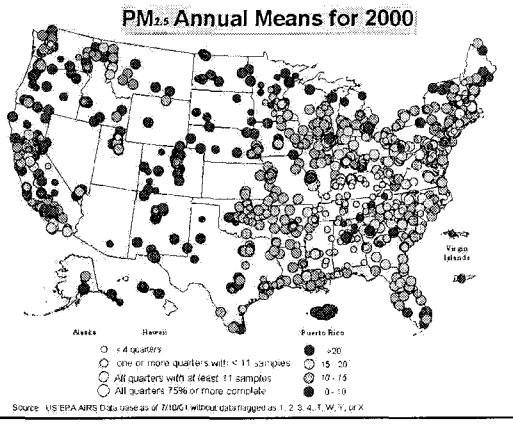
- Expansion of open burning ban
- Enhancements to vehicle inspection and maintenance program
- Shutdown of cement kiln
- Over-control of IC engines

NOx SIP Not Proposed For:

- New local vehicle controls
- No transportation conformity impacts
- 8 hour ozone standard
- Fine particulate matter standard



RECOMMENDED 8 HOUR OZONE AREAS



NOx SIP Call – How Will It Work?

- NOx reductions from Cement Kilns and Large IC Engines
  - Permits requiring these reductions have been/will be issued
- Use "excess" NOx reductions from cement kilns and IC engines and consider the use of NOx reductions from mobile and/or area sources
- NOx Budget Trading Rule

NOx Budget Trading Rule

- All remaining required NOx reductions (>90%) will come from NOx Budget Trading Rule for large boilers and combustion turbines at utilities (EGUs) and industries (non-EGUs)
  - Fossil-fuel fired and >250 mmBtu/hr heat input or serve generator >25 MW
- Rule and SIP set a fixed budget or cap (i.e., an allocation pool) and a method for allocating allowances
  - (an allowance is an authorization to emit one ton of NOx)
- Sources may install controls, increase efficiency, or purchase allowances

NOx Budget Trading Rule (con't)

- Size of allocation pool – minimum of 30,835 allowances [29,416 allowances - EGUs] [1,419 allowances - non-EGUs]
- Rule allows trading of allowances across states
- Georgia EPD will determine allowance allocations and issue permits
- EPA will administer multi-state trading program by tracking emissions and trading
- EPA developed model budget trading rule for states and promotes use of a trading rule as part of a state's NOx SIP

NOx Budget Trading Rule (con't)

- Why do interstate trading vs. traditional command and control?
  - Rule is for broad, regional reductions
  - Favored by vast majority of commenters (63 FR 57457)
  - Administrative burden on permitting authority is significantly less
  - Cost of compliance with trading is significantly less (63 FR 25920)
  - All other affected states are participating in the interstate trading rule

NOx Budget Trading Rule (con't)

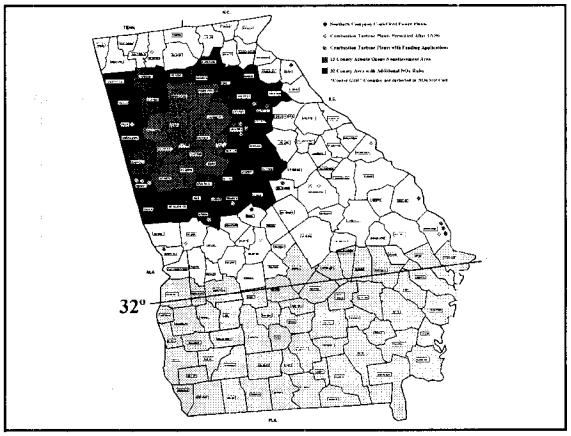
- Emission Trading Rules Are Not New
  - Acid Rain Program (48 States) for SO2 emissions has been in place since 1995
    - Reductions have been greater than anticipated
    - Costs have been lower than anticipated
  - Ozone Transport Region (Northeast US) for NOx emissions has been in place since 1999

NOx Budget Trading Rule (con't)

- Key issues to Rule development:
  - Set-aside of allowances
  - Initial allocation of allowances
  - Reallocation of allowances
  - Compliance Supplement Pool
- The following discussion relates primarily to EGUs. While the same key issues apply to non-EGUs as EGUs, the majority of the existing non-EGUs support a long initial allocation period with no new source set-asides

NOx Budget Trading Rule (con't) Set-aside of Allowances

- Allowances could be set aside for new sources.
  - Under EPA's model trading rule, sources commencing operation after May 1, 1995 are considered "new sources".
  - Since May 1, 1995, EPD has issued permits to 18 new utility plants totaling 11,785 megawatts of capacity
  - The larger the new source set-aside, the more new sources that will receive allowances from the allocation pool, versus adding controls and/or buying allowances
  - The larger the new source set-aside, the less allowances that existing sources will receive, thereby, increasing the stringency of the rule for those sources
  - Moving the May 1, 1995 cutoff date for "new sources" forward would decrease the need for and/or the size of the new source set-aside



NOx Budget Trading Rule (con't) Set-aside of Allowances

- Allowances could be set aside for renewable energy or energy efficiency projects
- Allowances for set-aside could be increased by using reductions from control measures not targeted for reduction by NOx SIP Call
- EPA's model trading rule includes a 5% new source set-aside for initial allocation period followed by a 2% set-aside for reallocation
- EPA's model trading rule does not include a set aside for renewable energy or energy efficiency projects

NOx Budget Trading Rule (con't) Initial Allocation of Allowances

- Initial Allocation could be of short duration (3 to 5 years)
  - Sources receiving allowances would only know their allowance allocations for a few years
  - Other sources would be able to get into the allocation pool in a relatively short period
- Initial Allocation could be of longer duration (10 years or more)
  - Sources receiving allowances would be assured of a set level of allowances for longer period, aiding in planning activities
  - Other sources would have to purchase allowances for longer period of time

NOx Budget Trading Rule (con't) Reallocation of Allowances

- More frequent reallocation of allowances
  - New sources would eventually get into the allocation pool and be awarded allowances
  - More frequent reallocation results in new sources getting in the pool sooner
  - Allowance allocations to existing sources decrease over time as new sources get into pool
- Infrequent or no reallocation of allowances (after initial allocation)
  - Sources not receiving allowances during the initial allocation period would have to purchase allowances in the market longer

NOx Budget Trading Rule (con't) Initial Allocation/Reallocation of Allowances

- EPA's model trading rule has a 3 year initial allocation period, followed by reallocation of allowances on an annual basis

NOx Budget Trading Rule (con't) Compliance Supplement Pool

- Pool of allowances in excess of the trading rule cap
- EPA's model rule provides for issuance of allowances based on early reductions
- Allowances from pool have to be used in first two years of trading rule
- Size of pool ~ 10,700 allowances

NOx Trading Rule – Other States

State	New Source Set-aside for Initial Allocation Period	Initial Allocation Period	Cutoff Date for Existing Sources	Reallocation Frequency	Compliance Supplement Pool
AL	0% [6.3 % assuming 5/1/95 cutoff date]	4 years (2004-07)	Complete Appl. by 10/2/00	Every 3 yrs. [allowances to new sources upon retirement of existing sources]	Available for early reduction credits
TN	4.3%	15 years (2004-18)	Begin operation by 5/1/95	Annually	Available for early reduction credits
SC	4%	3 years (2004-06)	Begin operation by 5/1/98	Every 3 yrs.	Available for early reduction credits
NC	varies, 3.6% - 2006, 5.3% - 2007	Permanent w/ review every 5 yr.	Permitted by 10/31/00	Every 5 yrs?	Available for early reduction credits

Timeline (con't)

- Summer 02(?) – EPA expected to issue final rule
- Summer 03(?) – Georgia required to submit SIP to EPA
- May 1, 2005 – Affected sources in GA required to achieve NOx reductions required by SIP

EPD's Next Steps

- Evaluate verbal and written comments from the public and regulated companies regarding the development of Georgia's NOx SIP
- Conduct further meetings with interested parties
- Finalize proposed NOx budget trading rule and SIP and initiate rulemaking process

EXHIBIT 7

Georgia Department of Natural Resources
Environmental Protection Division • Air Protection Branch
4244 International Parkway • Suite 120 • Atlanta • Georgia 30354
404/363-7000 • Fax: 404/363-7100
Lonice C. Barrett, Commissioner
Harold F. Reheis, Director

MAR 0 6 2002

Mr. D. Blake Wheatley
Assistant Vice President
Longleaf Energy Associates, LLC
400 Chesterfield Center, Suite 110
St. Louis, Missouri 63017

RE: Longleaf Energy Station, Early County
Application for Pulverized Coal-fired Power Plant
Application No. 13615
Dated February 14, 2002

Dear Mr. Wheatley:

We have conducted a cursory review of your application. During our review, we discovered several critical flaws and omissions that must be resolved before we can review the application.

In order to provide prompt notification of these major deficiencies, this letter will focus on the pulverized coal-fired boilers, the auxiliary boiler, and the overall permitting process. It does not address the PM-10 sources, the other auxiliary equipment, and the computer modeling (both PSD and air toxics). These items will be addressed, to the extent necessary, after you have resolved the issues described herein.

1. Applications Being Returned and Permit Review Timing

The permitting of a new coal-fired power plant under the PSD regulations is a very complex and lengthy process. An application such at this one, which is revised many times, often takes longer to review and issue a permit than if the company had waited until the application was complete to submit the entire application. And in those cases when the application is very fragmented, it is easier for us to miss an applicable requirement. Therefore, we are sending you back the remaining copies of the application that are in our possession (a couple were already sent to EPA Region 4 and the Federal Land Managers). We request that you resolve deficiencies that are noted below and resubmit the entire application to us at that time.

We realize that this may have an effect on the permit issuance timing. However, even if the application had been complete when it was first submitted, it is doubtful that we could have issued a permit by the date specified in the application. You should plan your construction schedule accordingly. Similarly, if the facility is unable or unwilling to meet all the permitting requirements, we will not be able to issue a permit at all.

2. EPA Region 4 Comment Letter on Similar Facility

On February 26, 2002, EPA Region 4 wrote a letter commenting on the preliminary determination and draft PSD permit for a very similar source, the Thoroughbred Generating Company, LLC project located in Muhlenberg County, Kentucky. We have noted that many of the comments EPA made regarding that facility apply to your application as well. A copy of this letter is attached for your reference (Attachment 1).

3. Plant Configuration

Your application is for a pulverized coal-fired steam-electric power plant. The application does not discuss any other methods for generating electricity from the combustion of coal, such as pressurized fluidized bed combustion or integrated gasification combined cycle. You should discuss these technologies and explain why you elected to propose a pulverized coal-fired steam-electric power plant instead.

4. Impact on Water Resources

It is our understanding that this facility would need a significant amount of water (greater than 10 million gallons per day) in order to operate at capacity. Please provide the source(s) of water and quantity of the water you expect to use as well as how you will handle your water discharges. Also, please indicate whether any EPD permits are needed for water usage/discharge and if those permits have been applied for.

5. "Top-Down" BACT Analysis for Pulverized Coal-fired Boilers and Auxiliary Boiler

One of the most important parts, if not the most important part, of the PSD application is the BACT review. U.S. EPA and Georgia EPD require that the BACT analysis be done in a "top-down" fashion. This procedure consists of five discrete steps. A summary of these steps from the NSR Workshop Manual is attached (Attachment 2) for your reference. By attempting to consolidate many or all of these steps into one step, you have omitted very important parts of the process. You should redo this analysis and ensure that you follow each of the detailed "top-down" steps that are described in the NSR Manual.

6. BACT Emission Limits

It appears that you have limited your consideration of potential BACT control technologies and corresponding BACT emission limits to those that you found in the RACT/BACT/LAER Clearinghouse (RBLC). This is not acceptable. The attached EPA Region 4 comment letter mentions numerous other sources of achievable BACT levels that you should be able to review. In addition, Babcock & Wilcox presented a paper titled "How Low Can We Go" at the 2001 Mega Symposium. This paper (Attachment 3) reports that there are emission control technologies for eastern bituminous coal that can achieve 0.016 lb/mmBtu NOx, 0.04 lb/mmBtu SO2, and 0.006 lb/mmBtu PM-10. For western PRB coal, achievable emission levels of 0.008 lb/mmBtu NOx, 0.04 lb/mmBtu SO2, and 0.006 lb/mmBtu PM-10 are reported. Keeping in mind that the Permitting Authority must consider all information submitted through the comment period on the draft permit in assessing BACT, at the present time EPD is considering these levels as BACT. These levels must be included in your analysis.

7. Ambient Impacts Analysis

On page 11, you correctly identify that a source impact analysis must be done for every pollutant emitted in significant amounts. However, no such analysis was done for ozone. We have confirmed with EPA Region 4 that no ozone computer modeling must be done for PSD purposes because there is no approved method for doing so. However, we believe that current ozone levels around Early County and the expected impact on ozone from the proposed source must be discussed in order to satisfy this requirement (see 40 CFR 52.21(k) and (m)).

8. Emissions During Startup and Shutdown - BACT

As you know, the emission rates of virtually all PSD pollutants and some HAPs can be significantly greater during startup and shutdown than they are during steady state operation. However, your application states that you intend for no BACT limitations to apply during these events. Please be advised that there are no automatic or blanket exemptions for excess emissions during startup and shutdowns under the PSD regulations. The EPA Region 4 comment letter addresses this issue. You should investigate the use of cleaner fuels, such as natural gas, LPG, or distillate fuel oil (the application mentions distillate fuel oil, but only a limited amount for and for only a few hours) while the boiler and the emission control equipment are starting up.

As EPD has done in other PSD permits, the BACT limits would technically apply during startup and shutdown. In accordance with Georgia Rule 391-3-1-.02(2)(a)7., excess emissions caused by startup or shutdown may be allowed provided that certain conditions met. In addition we would most likely limit the duration and/or frequency of the startup and shutdown events. You should submit a proposal for limiting these events.

9. Emissions During Startup and Shutdown – NAAQS and PSD Increments

It appears that you did model emissions from the proposed source during startup (discussion page 70). However, it appears that you compared the source impacts alone, with no background sources, to the NAAQS. We believe that you should compare the source impacts, with background sources, to the NAAQS. And you should compare the source impacts, with other PSD increment consuming sources, to the PSD Increments.

10. Class I Area Impacts

The application mentions the Class I Areas that are within 200 km of the proposed source. However, it does not include any Class I Area impact analysis. The application does mention that it will be submitted at a future date. You should do this as soon as possible. In order to ensure that your analysis is done correctly, you should contact the FLMs for the potentially affected Class I Areas. Please note that EPD should be copied on any written correspondence that is exchanged between the source and the FLM. EPD will follow up with the FLMs located within 200 km of the proposed site to ensure that their concerns have been addressed.

11. Acid Rain Regulations

The application does not address the requirements of the Acid Rain Regulations. No Acid Rain application has been submitted and no timetable for submitting the application was reported. 40 CFR 72.30(b)(2) requires you to submit a complete Acid Rain application at least 24 months before the facility commences operation. Please indicate when the Acid Rain application will be submitted (note: you do not need to submit the actual application in your response, just indicate when it will be submitted).

12. Case-by-Case MACT Determination

The proposed facility is subject to a case-by-case MACT determination in accordance with 40 CFR 63.43. No such analysis was submitted in the application.

13. Conclusion

Your attention to this matter is appreciated. If you have any questions, you may contact me at 404/363-7143.

Sincerely,

James A. Capp
Manager, NOx Permitting Unit
Stationary Source Permitting Program

Enclosures

CC: Mr. Jim Little, EPA Region 4
Ms. Sandra Silva, US FWS
Mr. David Wergowske, USDA Forest Service