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# City of Alexandria, Virginia

## MEMORANDUM

DATE: NOVEMBER 7, 2003

TO: THE HONORABLE MAYOR AND MEMBERS OF CITY COUNCIL

FROM: PHILIP SUNDERLAND, CITY MANAGER

SUBJECT: AIR QUALITY ISSUES RELATED TO THE OPERATIONS AT MIRANT POTOMAC RIVER GENERATING PLANT

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The purpose of this memorandum is to address air quality issues related to two types of emissions from the Mirant Potomac River Plant, particulate matter ("PM") emissions and oxides of nitrogen ("NOx") emissions. The memorandum provides a summary of the information included in the Mirant Power Plant Emissions and Health Effects Report ("Report") (Attachment 1), submitted to the City in late August by Elizabeth Chimento and Poul Hertel, identifies the air quality issues related to the Mirant Alexandria plant emissions that are raised by the Report, and describes the steps the City is pursuing to address these issues.

## I.

## Particulate Matter Emissions from the Mirant Plant

### Summary of Studies Presented in Report

The Report is an excellent compilation of some of the more recent studies relating to air pollution and the health effects of fine particulates, both in general and from coal fired power plants in the Washington Region, including the Mirant plant in Alexandria. The information below summarizes the studies included in the Report, which can be categorized into three types:

1. A number of recent general health studies indicate that substantial long-term exposure to fine particulate matter – i.e., particulates of a diameter less than 2.5 microns ("$PM_{2.5}$") – may pose a risk to human health. The central theme of these studies, including Levy et al. and Pope et al. (JAMA article) which are addressed in the Report, is that there is a correlation between substantial, long-term exposure to ambient concentrations of $PM_{2.5}$ and increases in cardiopulmonary mortality, lung cancer and other adverse health impacts, even when the concentrations are below the National Ambient Air Quality Standards ("NAAQS") for $PM_{2.5}$ that have been established by the federal government.[1]

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[1] The NAAQS for $PM_{2.5}$ is 15 micrograms per cubic meter ($\mu g/m^3$) annual mean, and 65 $\mu g/m^3$ measured on a 24 hour basis. Further discussion of these standards is on page 6 below.

2. The Levy study finds that if "Best Available Control Technology," or BACT (a term referring to the most effective, currently available pollution control technology) for PM, NOx and sulfur dioxide ($SO_2$) were installed on the five coal fired power plants in the Washington region (including the Mirant plant in Alexandria), regional $PM_{2.5}$ concentrations would be reduced by up to 0.9 $\mu g/m^3$ on an annual basis, which in turn would produce health benefits within the region, including reductions in cardiovascular admissions and pediatric hospital emergency room visits. The Levy study also finds that these health benefits would occur predominantly in sensitive populations, such as the elderly, diabetics, asthmatic children and those with lower incomes. The Levy study focused on a 250 mile radius area around Washington, D.C., with a population of 47 million people.

3. Separate reports from Penn State University and the Virginia Department of Environmental Quality ("VDEQ") addressed the makeup of particulate matter collected from homes in the immediate neighborhood of the Mirant plant. These reports concluded that coal dust was present in the neighborhood samples of particulate matter, as was flyash, although to a much lesser extent. The Penn State report also concluded that the majority of the particulates in the sample were course particulates, greater that 10 microns in size.

   The Penn State report (attached to the Report) concluded that this coarse coal dust came from operations (not from the stacks) at the Mirant plant (e.g., from the coal pile or coal handling processes). The VDEQ report concluded that, while coal dust was a significant component of the particulate matter found in the neighborhood samples, the level of particulates, including coal dust and flyash did not exceed what is typically found in an urban setting and did not show that the Mirant plant was producing particulates in violation of relevant regulations. (These VDEQ conclusions are set out in a June 10, 2003, memorandum attached to the Report, and in an August 12, 2003, memorandum which is Attachment 2 to this memorandum.)

   Neither of these two studies was quantitative; thus, neither determined the amount of coal-related and flyash particulates in the neighborhood around the Mirant plant that had come from the plant.

**Background Information on Fine Particulate Matter**

Particulate matter refers to all solid and liquid particles found in the air. Attachment 3 summarizes the size characteristics of particulates. A size of 1μm (1 micron) is equal to one-thousandth of a millimeter or one-millionth of a meter. $PM_{2.5}$ refers to fine particulates that are less than 2.5μm in diameter; particulates less than 10μm in diameter are referred as $PM_{10}$. Particulates smaller than 2.5μm are respirable, have a greater chance of getting deposited in lungs and, thus, are considered to have a far greater potential health impact compared to larger or coarse particulates.

The primary source of fine particulates typically found in urban areas consists of various combustion activities -- specifically, "point sources" such as power plants, "area sources" such as industrial boilers and commercial and home heating systems (including wood burning stoves), and "mobile sources" such as buses, trucks, automobiles, trains and aircraft. As earlier noted, the current National Ambient Air Quality Standard for $PM_{2.5}$ is an annual mean of 15 $\mu g/m^3$. The NAAQS for $PM_{10}$ is an annual mean of 50 $\mu g/m^3$. Alexandria and the metropolitan Washington area are currently meeting these standards. (For additional information on fine particulates, see pages 18 and 19 of the Report.)

**Particulate Emissions from the Mirant Plant**

Three types of particulate matter are "produced" at the Mirant plant.

The first is primary particulate matter some of which is released as a result of the combustion process through the plant's stacks as uncollected flyash (i.e., not captured by the control equipment described below), and some of which is released during the handling of collected flyash. Mirant operates air pollution control equipment to control flyash particulate emissions. The plant uses hot and cold electrostatic precipitators (ESPs) to control flyash emissions leaving the stacks, and uses baghouses to control flyash emissions from the silos where flyash collected by the ESPs is stored prior to being loaded on to trucks. The ESPs control greater than 99% of the primary particulate emissions leaving the stacks. Nonetheless, Mirant reported to VDEQ that the plant emitted 588.3 tons of $PM_{10}$ from its stacks during calender year 2002.[2] A portion of these particulate emissions are less than 2.5 microns, and are referred to as primary $PM_{2.5}$.

The second type of particulates generated by the Mirant plant consists of secondary $PM_{2.5}$. These are referred to as secondary particulates because they are formed away from the plant when gaseous emissions from the plant react in the atmosphere to produce $PM_{2.5}$, typically in the form aerosols, nitrates, sulfates and similar compounds. These $PM_{2.5}$ particulates are formed downwind and miles from the plant. Because of their nature and the location of their formation, secondary $PM_{2.5}$ emissions are not measured or reported by power plants.

The third type of particulates produced by the Mirant plant is in the form of coal dust that is released during the delivery, handling and storage of coal at the facility. These particulates tend to be coarse and large ($PM_{10}$ or larger), and in some cases are visible to the naked eye.

Mirant employs the following measures to control coal dust at the plant.

- Uses a car dumper water spray header to suppress dust during the unloading the individual coal cars;

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[2] A July 2001 study conducted by a consultant for Mirant identified the flyash silos as a source of particulate emissions, and calculated that up to 29 tons a year of $PM_{10}$ could be emitted from the silos, some of which would be $PM_{2.5}$.

- Uses a car dumper door curtain to enclose the area during the unloading process;
- Uses a primary process surfactant spray, coupled with an enclosed chute, during coal transport;
- Maintains the coal chute with a surfactant spray nozzle to reduce the coal dust plume when coal is dropping to the pile;
- Routinely compacts the coal pile with a bulldozer and maintains the surface in a manner not conducive to releasing fugitive dust; and
- Maintains the coal handling area in the main plant building under a slightly negative pressure so that any leakage in this area flows into the plant.

Even with these measures, the City has received complaints in reference to particulates observed by the naked eye in the neighborhood of the plant. In addition, it has been reported by citizens that the coal pile is periodically higher than in previous years. The Penn State and VDEQ studies confirm that particulates from the Mirant plant -- the first and third types of PM identified above -- are leaving the site and being released into the community. These particulates tend to be relatively large in size and weight and, as a result, settle to the ground quickly and near the plant. For example, Penn State found the mean size of the coal dust particles in the neighborhood sample it analyzed to be 45μm, and the mean size of the flyash particles to be 30μm. These larger, coarse particulates tend to be a nuisance, and present considerably less of a health risk (if any) because they are filtered by our natural immune systems, such as the throat and nose, and do not make it deep into the lungs, the way $PM_{2.5}$ particulates do.

**Particulate Monitoring**

There are currently two types of particulate monitoring in the area around the Mirant plant. The closest monitor is a $PM_{10}$ station operated by the City. This was installed at the Health Department at 517 North St. Asaph Street in October 2001 in response to community concerns about particulate emissions from the plant. Previously, the City had been monitoring $PM_{10}$ on the west end, at Cameron Station; this was suspended when the property was sold for redevelopment. Based upon the data produced by the monitoring station at the Health Department, the levels of $PM_{10}$ found in the north end of Old Town are similar to the $PM_{10}$ levels found in the west end of the City in the early 1990s, and the levels currently found in urban areas throughout Northern Virginia.

The City continues to monitor for $PM_{10}$ at the Health Department. Attachment 4 summarizes the ambient $PM_{10}$ data from monitoring stations in and around Alexandria. Attachment 5 shows the location of other monitoring stations, which are operated by VDEQ in Northern Virginia, and their distance from the Mirant plant.

There are three $PM_{2.5}$ ambient air monitoring stations operated by VDEQ in Northern Virginia. None of these stations is within the City. Two stations are within 4.5 miles of the Mirant plant, one to the north and one to the south. Attachment 6 summarizes the ambient $PM_{2.5}$ data from these monitoring stations (Attachment 5 shows their location and distance from the plant). The VDEQ monitoring section has consistently maintained that the $PM_{2.5}$ monitoring it currently undertakes adequately covers the Northern Virginia region, including Alexandria. Based on discussions with VDEQ, available monitoring data, and dispersion characteristics of $PM_{2.5}$, the levels of $PM_{2.5}$ in the City and the immediate neighborhood are expected to be comparable to those throughout the Washington region.

**Activity at the Federal level on PM Standards**

The National Ambient Air Quality Standards for a pollutant are set at the national level by the Environmental Protection Agency ("EPA") after an evaluation of the sources, atmospheric levels, exposures and health effects of the pollutant. EPA promulgated the standards for $PM_{2.5}$ in 1997, but said that it would further review the standards before any areas could be designated as non-attainment for $PM_{2.5}$ and any new $PM_{2.5}$ controls would be required. EPA is in process of conducting this review, and has drafted a "Criteria Document" which evaluates the sources, atmospheric levels, exposures and the health effects of $PM_{2.5}$.

On September 2, 2003, EPA staff recommended an annual $PM_{2.5}$ standard between 12 $\mu g/m^3$ and 15 $\mu g/m^3$ (the current standard) and a 24-hour $PM_{2.5}$ standard between 30 $\mu g/m^3$ and 50 $\mu g/m^3$ (the current 24 hour standard is 65 $\mu g/m^3$). Staff also recommended that a new standard for the larger fraction of PM be set, which would cover particulates ranging from $PM_{2.5}$ to $PM_{10}$. Clearly, more debate will occur at the federal level before enforceable air quality standards for $PM_{2.5}$ will be in effect.

## II.

## NOx Emissions from the Mirant Plant

The process of burning coal generates oxides of nitrogen or NOx. There is a National Ambient Air Quality Standard for NOx which the Washington region does not meet. NOx emissions are important because they contribute to acid rain and are a precursor pollutant for the formation of ozone. Ozone is a summertime gaseous pollutant that is formed by a chemical reaction when NOx and volatile organic compounds react with sunlight. Ozone is the only pollutant for which the Washington region is not meeting the applicable NAAQS. The Washington region is classified as a "severe" non-attainment area for ozone. As a result, Virginia is required to develop and submit to EPA, for approval, a State Implementation Plan ("SIP") demonstrating how Virginia will meet the ozone standard by 2005.

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[3] The Clear Skies Initiative, proposed by President Bush in February 2002, is currently being debated at the national level. This program proposes a multiple pollutant approach targeting NOx, mercury and sulfur dioxide emissions. If enacted, the program will likely have significant impacts on pollution controls and regulations governing older power plants, like the Mirant Potomac River plant.

The revised SIP for ozone requires that, beginning in 2003 NOx emissions from the Mirant plant in Alexandria be limited to an average rate of 0.15 pounds per million BTU of heat input, resulting in a cap of 1,019 tons during the ozone season from May through September. This emissions cap is enforced through a state operating permit that was issued by VDEQ in 2000. This past September, VDEQ issued a Notice of Violation ("NOV") to Mirant, charging it with violating its permit by exceeding the allowed level of NOx emissions during the 2003 ozone season (Attachment 7). Mirant has responded to the NOV with a letter dated September 19, 2003, disputing the NOV (Attachment 8).

VDEQ and Mirant are currently discussing options for achieving compliance with, or otherwise dealing with, the existing Mirant permit and its NOx cap. Among the proposals currently being discussed is the installation of "separate overfire air operational improvements" on the plant's boiler units 3, 4, and 5, which is projected to result in a 30% reduction of emissions from these boilers over three years. This level of emission reductions, however, would not produce the amount of reductions necessary for the plant to comply with the currently applicable NOx cap.

Another option for Mirant to achieve the required NOx reductions, is to install NOx-control technology similar to that now in use at the waste-to-energy facility ("selective non-catalytic reduction" or "SNCR" technology). This involves the injection of ammonia into the flue gas stream to control NOx emissions. This technology raises some important safety concerns, in that it would involve the delivery of large quantities of liquid ammonia (or a similar compound) by train or tanker truck to the Mirant plant and the potential for ammonia slippage, where excess ammonia is released from the stack .

Another means for Mirant to achieve NOx compliance is to produce or obtain NOx reductions at one or more other NOx-producing facilities in the region, and to "trade" these reductions for the reductions otherwise required at the Potomac River plant. Under this alternative, no additional controls would need to be installed at the Mirant plant in Alexandria.

A further way for Mirant to meet the NOx emissions requirements at its Alexandria plant is to change the type of fuel that is burned at the plant, from coal to natural gas. This would require bringing a high pressure gas line to the plant, at a very significant cost.

All of these options have significant positive and negative impacts that require further analysis.

## III.

## Ongoing and Future City Efforts to Address These Issues

In order to assist the City and neighbors of the Mirant plant understand the nature of the particulate emissions coming from the Mirant plant, the health impacts, if any, of the plant's $PM_{2.5}$ emissions in the immediate neighborhood, and the pros and cons of the various options relating to the control of NOx at the plant, we plan to retain the services of a member of the

faculty of the Harvard School of Public Health, Dr. Jonathan Levy, who was an author of one of the studies attached to the Report. Working with the authors of the Report, we are developing a series of questions and a scope of work for Dr. Levy. We hope to have a final scope worked out in the next week.

The following issues are of particular significance and need to be understood before any decisions can be reached regarding emissions from and operations at the Mirant plant. The issues address (i) $PM_{2.5}$ emissions from the Mirant plant, (ii) the larger, coarse particulates that come from the plant, and (iii) NOx emissions from the plant.

- The amount of $PM_{2.5}$ that is emitted from the Mirant plant.
- The extent to which the Mirant plant's $PM_{2.5}$ emissions contribute to the ambient levels of $PM_{2.5}$ that are found near the plant and elsewhere in Alexandria.
- The most effective means of reducing $PM_{2.5}$ emissions from the Mirant plant, if reductions are needed to reduce unacceptable levels of this pollutant near the plant or elsewhere in Alexandria or the region.
- The extent to which coarse particulates (i.e., significantly larger than $PM_{2.5}$) that are emitted from the Mirant's stacks or arise from its coal and flyash handling operations contribute to the levels of such particulates that are found near the plant or elsewhere in Alexandria.
- The most effective means of reducing the amount of coarse particulates that leave the Mirant plant site and settle in the nearby neighborhood or elsewhere in Alexandria.
- The pros and cons to Alexandria of adding new control equipment to the Mirant plant to further reduce its NOx emissions vs. adding new equipment or taking other steps to further reduce NOx emissions at one or more other NOx-producing facilities in the region. (This is intended to address the "trading" issue -- i.e., considering air quality, the risks associated with NOx control technology and other factors, whether Alexandria would be better served by NOx reductions at the Mirant plant or at one or more plants in the region.)

In addition to moving forward with this analytical work, we have prepared a letter from the Mayor to VDEQ (Attachment 10) expressing the City's interest in the particulate emissions coming from the Mirant plant and related nuisance and health impacts. The letter also notifies VDEQ of the City's desire to participate in discussions related to Mirant's NOx compliance issues and to the issuance of a new Title V air permit for the Mirant plant, and asks that no decisions be reached on any of these matters without City participation.

Also, City staff will continue to monitor VDEQ's enforcement action against Mirant for its exceedance of the NOx emissions cap during 2003 ozone season. Staff will continue its ongoing

discussions with VDEQ concerning air quality monitoring near the Mirant plant. We also will continue to monitor regional, state and federal issues and legislation that may directly or indirectly impact the Mirant Plant and its operations.

City staff currently inspect the Mirant plant at least twice every year. More frequent visits are made in response to noise or air pollution complaints. Staff will now increase the frequency of inspections to quarterly inspections, in order to better ensure that particulate control measures at the plant are in place and functional. Staff will also evaluate how existing measures can be improved and initiate discussions with Mirant on the installation of additional controls.

Finally, we will keep you informed on the work performed by Dr. Levy, who will be working with staff and the authors of the Report.

Attachment 1: Mirant Power Plant Emissions and Health Effects Report prepared by Elizabeth Chimento and Poul Hertel
Attachment 2: VDEQ Memorandum dated August 12, 2003
Attachment 3: Particle Size Chart
Attachment 4: $PM_{10}$ data for Northern Virginia
Attachment 5: Air Monitoring Stations and Power Plant Locations
Attachment 6: $PM_{2.5}$ data for Northern Virginia
Attachment 7: NOV issued by VDEQ dated September 10, 2003
Attachment 8: Letter from Mirant to VDEQ dated September 19, 2003 disputing the NOV.
Attachment 9: Glossary of Terms
Attachment 10: Letter from Mayor to VDEQ

cc: Richard Baier, P.E., Director, Transportation & Environmental Services
Charles Konigsberg, M.D., Director, Alexandria Health Department
Michele Evans, Assistant City Manager
Bernard Caton, Legislative Director
William Skrabak, Chief, Div. Environmental Quality, T&ES

ATTACHMENT 2

# COMMONWEALTH OF VIRGINIA
# DEPARTMENT OF ENVIRONMENTAL QUALITY

## MEMORANDUM

TO: Jeff Steers, Regional Director, NVRO

THROUGH: John Bowden, Deputy Regional Director, NVRO

FROM: Kelly Lease, Air Compliance Inspector, NVRO
Charles D. Forbes, Air Compliance Manager, NVRO

SUBJECT: Summary of Investigation into Dust Complaints on Pitt Street, Alexandria, in the Vicinity of Mirant's Potomac River Generating Station.

DATE: August 12, 2003

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The purpose of this memo is to summarize NVRO's investigations into the subject matter. The case is closed.

Background

On April 22, 2003, the Northern Virginia Regional Office Air Compliance Unit (NVRO) collected dust samples in response to citizen complaints/concerns regarding potential particulate emissions from Mirant's Potomac River Generating Station (Mirant). This facility is a coal-fired power plant near Pitt Street. Dust samples were collected at residences at 1200 and 1202 Pitt Street (approximately one-quarter mile from Mirant). A microscopist from DEQ's Office of Air Quality Assessment (OAQ), Ms. Carolyn Stevens, analyzed the samples using a polarizing light microscope at 150 times magnification and estimated that uncombusted coal dust constituted up to 50 percent of each sample. Significant quantities of coal fly ash were also observed in the samples.

A letter summarizing these preliminary findings was sent to Mirant, and a meeting requested to discuss the details. In response, Mirant requested a portion of the NVRO samples. These were delivered to Mirant on June 25, 2003. According to Mirant, the samples were transported to the Mirant Service Center in Forestville, Maryland, where they were examined using a Scanning Electron Microscope (SEM). Mirant also reports that, on June 27, 2003, it examined coal samples taken from coal stocks and fly ash samples collected from the pollution control equipment at the plant. No NVRO representatives were present during this examination. A meeting was held at the SEM location on June 30, 2003. Attendees at the meeting included Kelly Lease (NVRO) and Ken Whitlock, City of Alexandria Division of Environmental Quality. Mirant attendees consisted of the following:

Debra Knight, Environmental Coordinator – Potomac River Generating Station
Narayanan Iyer, Supervisor Chemistry - Potomac River Generating Station
Michael Stumpf, General Superintendent – Plant Operations - Potomac River Generating Station
Mr. Neal Titer, Metallurgy Laboratory Engineer

Summary of June 27-30 Analyses and Discussions

Mirant presented a significant quantity of photographs and SEM spectra, allegedly produced during the June 27 analysis. It is impossible to determine if the source being analyzed was the NVRO samples. Moreover, it is generally difficult to distinguish unknown and reference sample origins based on apparent particle size or other gross physical characteristics. NVRO is still unclear as to how the reference coal samples were taken or how they were handled prior to analysis.

To summarize Mirant's assertions regarding its microscopy. particles originating from Mirant do not resemble those found in the NVRO samples. Similarly. particles from Mirant do not exhibit the analytical spectra provided for NVRO samples. Mirant vigorously asserts that none of the particles in NVRO samples are coal or fly ash from its plant. However. Mirant was unable to replicate the June 27th SEM analysis during the June 30th meeting. Moreover. as the analysis progressed. Mr. Titer began experiencing technical difficulties with the SEM and Mirant was unable to demonstrate the repeatability of Mirant's reference coal spectra. Citing that the electron filament of the SEM was likely at the end of its useful life. further examination of particles was ceased..

Mr. Stumpf stated that he considered Mirant's analyses across two days to be conclusive proof that the Pitt Street samples do not contain materials from the Mirant facility. Furthermore. Mr. Stumpf stated that Mirant will not change its coal handling procedures without conclusive and defensible evidence that coal dust in the surrounding environment originates from its facility. As further evidence that NVRO's samples cannot originate from the Mirant facility. Ms. Knight presented wind data collected at the facility from 1994 through early 1998. These data show that prevailing winds at the plant originate from the northwest and south-southwest (attached). The Pitt Street residences are generally south-southwest of the plant (i.e.. upstream of prevailing winds).

Follow Up Investigations by DEQ

OAQ has re-examined the NVRO samples and stands by its original conclusion that the Pitt Street samples include a significant amount of coal dust. As the analyst of record is not trained in SEM techniques. she cannot account for the apparent difference between the results.

On August 8. 2003. NVRO's Deputy Regional Director and the Air Compliance Manager visited the general area adjacent to the Mirant facility and walked a 3-4 block area immediately surrounding the plant (starting from Pitt Street). The weather was warm and dry. with light winds from the northeast.

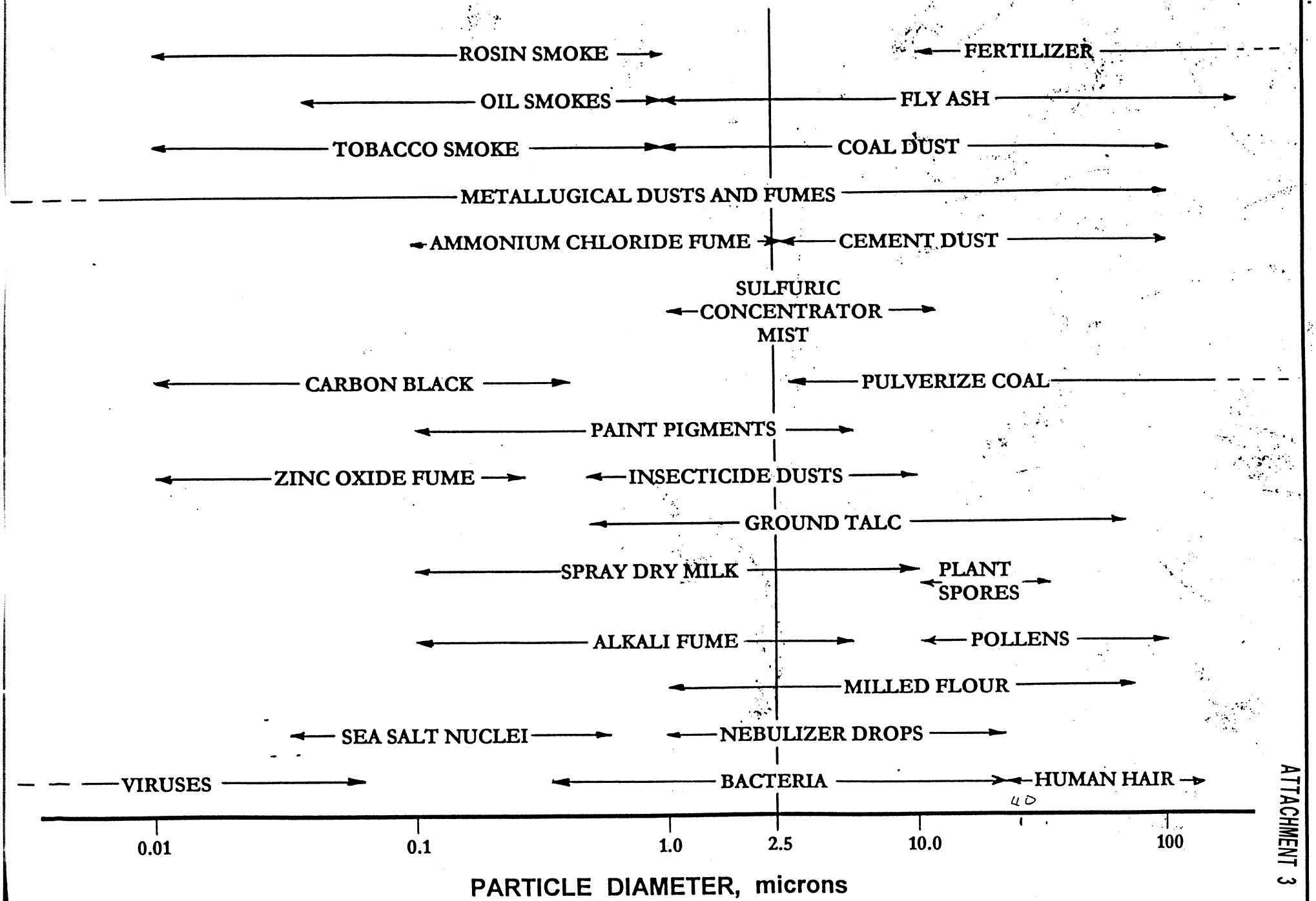
Horizontal surfaces on plants. houses. lamp posts. utility boxes. and other structures were examined to look for signs of excessive dust. While some dust was found. the level did not appear to exceed that typically encountered in this type of high-density urban setting. Coincidentally. a coal train was being unloaded at facility during the visit. No visible emissions were being generated by this operation. nor was any other part of the facility (including coal piles). Close inspection of horizontal surfaces at the residences closest to this operation indicate a level of dust levels similar to that observed on Pitt Street. i.e.. insignificant amounts

Conclusion

The practical choices for resolving this matter seem to center on either accepting the validity of Mirant's SEM analysis (and concluding that it is not the source of coal and fly ash). or undertaking a more rigorous course of sampling at the Pitt Street location. Mirant has indicated that it will vigorously contest any allegations of it violating the pertinent dust control regulations in the absence of the most rigorous analytical regime. Given the field observations made by DEQ personnel on August 8th. combined with Mirant's presentation. there seems little evidence that the plant is emitting dust in violation of the relevant regulations. NVRO therefore concludes that the complaint does not warrant further action at this time.

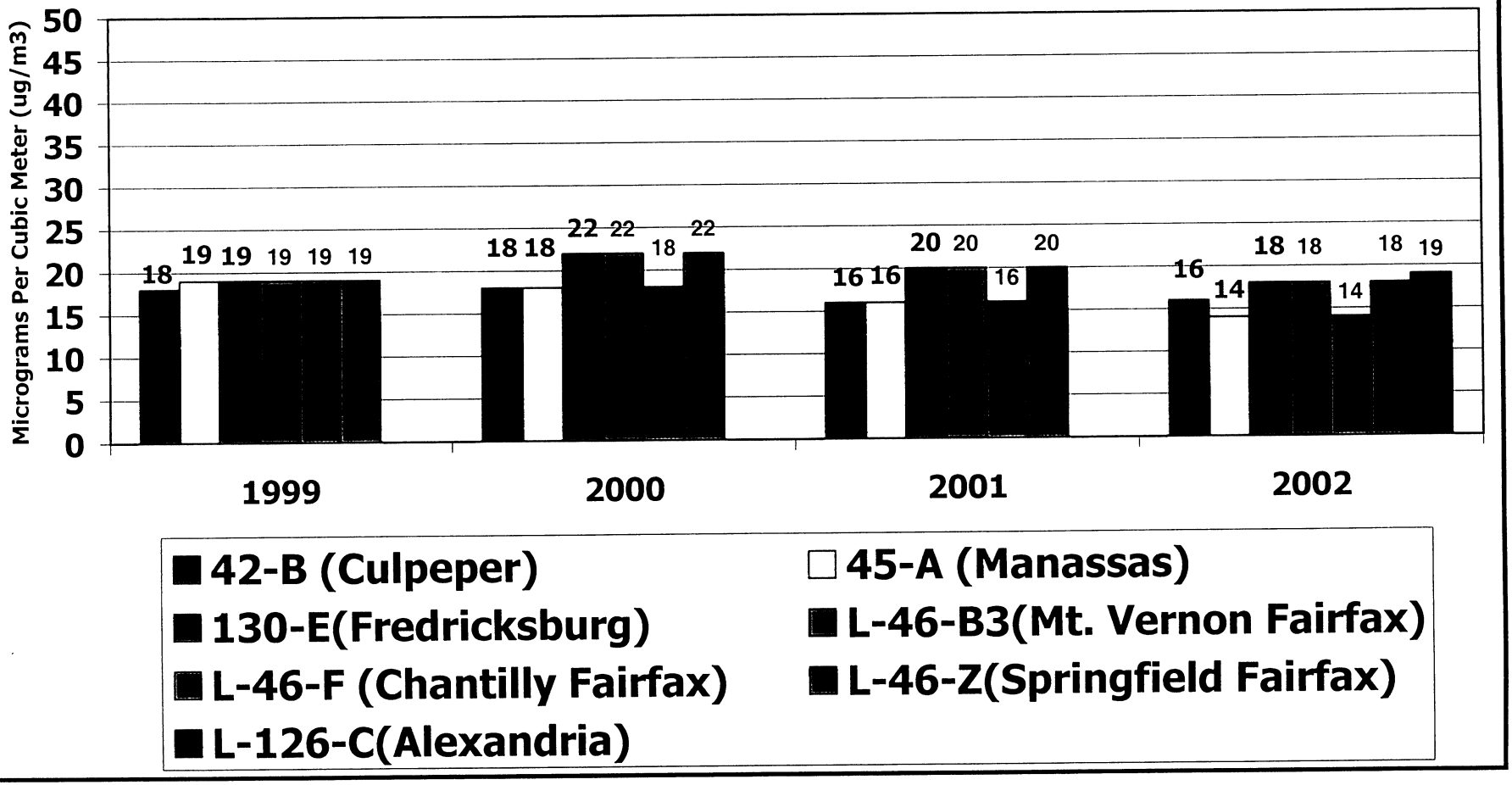
cc. Alice G. Nelson. CO

# PARTICULATE SIZE COMPARASIONS



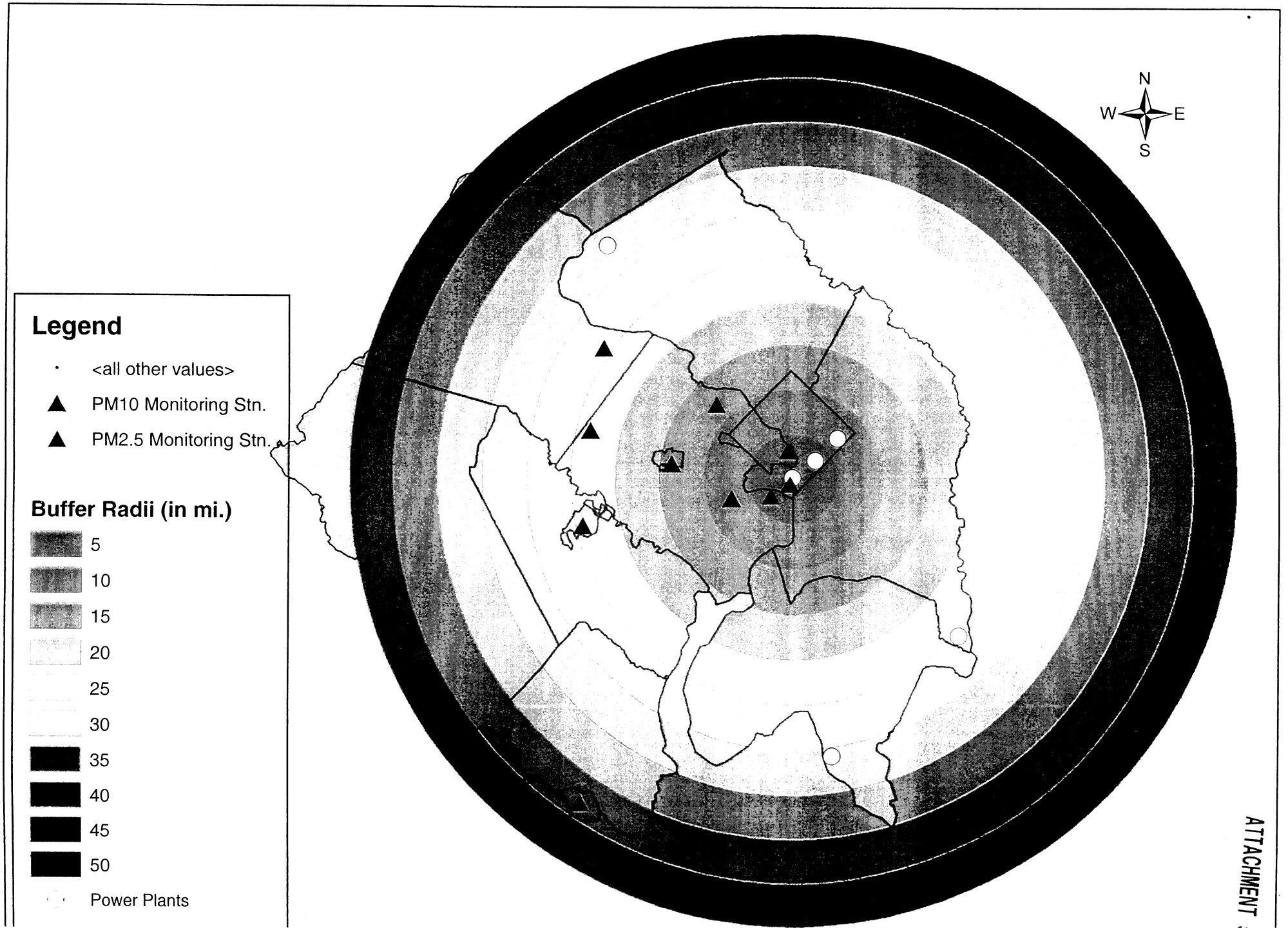

ATTACHMENT 3

# Northern Region
# PM10 Annual Means
## (Primary Standard 50 ug/m3)

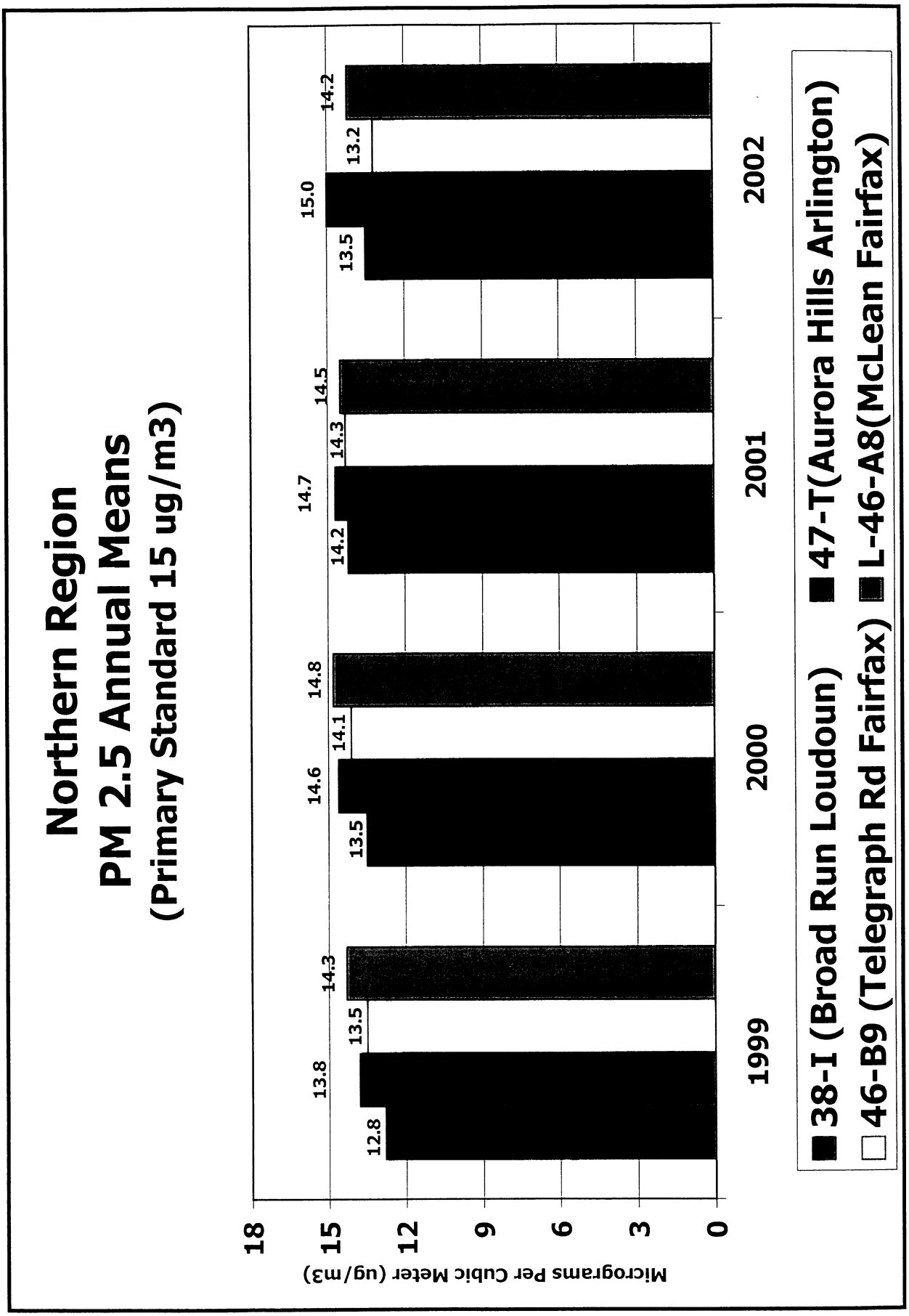



ATTACHMENT 4

# AIR MONITORING STATIONS AND DISTANCE FROM MIRANT (in Mi.)




ATTACHMENT


Northern Region
PM 2.5 Annual Means
(Primary Standard 15 ug/m3)
Micrograms Per Cubic Meter (ug/m3)
18
15
12
9
6
3
0
12.8
13.8
13.5
14.3
13.5
14.6
14.1
14.8
14.2
14.7
14.3
14.5
13.5
15.0
13.2
14.2
1999
2000
2001
2002
38-I (Broad Run Loudoun)
46-B9 (Telegraph Rd Fairfax)
47-T(Aurora Hills Arlington)
L-46-A8(McLean Fairfax)

September 10, 2003

Ms. Debra Knight
Environmental Coordinator
Mirant Potomac River, LLC
1400 North Royal Street
Alexandria, VA 22314

## NOTICE OF VIOLATION

RE: NOV
Mirant Potomac River, LLC
DEQ Reg.#: 70228

Dear Ms. Knight:

This letter notifies you of information upon which the Department of Environmental Quality (the Department) may rely to institute an administrative or judicial enforcement action. It is neither a case decision under the Virginia Administrative Process Act, Code § 9-6.14:1 *et seq.*, nor an adjudication. The Department requires that you respond to this letter within 10 days.

## FACTS AND LEGAL REQUIREMENTS

1. Section 110 (a)(1) of the Federal Clean Air Act (CAA) requires that the Commonwealth of Virginia maintain a State Implementation Plan (SIP), approved by the U.S. Environmental Protection Agency (EPA), to provide for the attainment and enforcement of National Ambient Air Quality Standards (NAAQS).

2. The Potomac River Generating Station is located in Alexandria, VA, which has been designated by EPA as a severe ozone nonattainment area under the CAA. Alexandria's air quality, as well as that for the entire Northern Virginia, Washington, D.C., and suburban Maryland region, does not meet the NAAQS for ozone.

3. Consistent with the Virginia SIP, in accordance with 9 VAC 5-80-800 C.2.b, and as mandated by Section 110 (a)(2)(D)(1) of the CAA, the

Department issued a Virginia Stationary Source Permit to Operate to the Potomac River Generating Station on September 18, 2000 (the Permit) that limited facility-wide emissions of oxides of nitrogen (NOx) in order to help ensure that emissions from the station do not contribute significantly to exceedances of the NAAQS for ozone in the Northern Virginia, Washington, D.C., suburban Maryland severe ozone nonattainment area. The Permit was approved by EPA and has been incorporated into the Virginia SIP. 65 Fed. Reg. 78,1000 (December 14, 2000).

4. Condition 3 of the Permit limits the Potomac River Generating Station to emitting no more than 1,019 tons of NOx (measured as $NO_2$) each year during the ozone season period of May 1 through September 30.

5. Condition 4 of the Permit requires the Potomac River Generating Station to comply with the NOx emissions limit contained in Condition 3 commencing in calendar year 2003.

6. On June 4, 2003, the Department requested from Mirant NOx emissions data for the Potomac River Generating Station for the purpose of assessing the station's compliance with the terms and conditions of the Permit, in particular Condition 3.

7. On August 26, 2003, Mirant's counsel informed DEQ by electronic mail that NOx emissions from the Potomac River Generating Station for the period of May 1, 2003, through July 31, 2003 were 1,174 tons. This total exceeds the emissions limit contained in Condition 3 of the Permit.

8. Upon information and belief, the Potomac River Generating Station has continued to emit additional, significant quantities of NOx during August and September 2003, thereby further exceeding the emissions limit contained in Condition 3 of the Permit.

## ENFORCEMENT AUTHORITY

Code § 10.1-1316 of the Air Pollution Control Law provides for an injunction for any violation of the Law, the Air Board regulations, an order, or permit condition. The same statute provides for a civil penalty up to $25,000 per day of violation of the Law, regulation, order, or permit condition. Code § 10.1-1307 authorizes the Board to issue orders, and Code § 10.1-1309 authorizes the Board to issue special orders to address such violations. In addition, Code § 10.1-1186

authorizes the Director of DEQ to issue special orders to any person to comply with the Air Law and regulations, and to impose a civil penalty of not more than $10,000.

The Court has the inherent authority to enforce its injunction, and is authorized to award the Commonwealth its attorneys' fees and costs.

## FUTURE ACTIONS

The staff must make a recommendation about how to proceed with this matter and whether to initiate an enforcement action based upon these facts. Before taking any further action, however, we would like to discuss this matter with you.

Your point of contact is Kelly Lease at (703) 583-3937. Please contact me within ten days of the date of this letter if you dispute any of the facts I have stated or if there is other information you believe the DEQ should consider. At the same time, please inform me of any corrective action you have instituted or plan to institute and the schedule for doing so.

A meeting to discuss resolution of this matter will be arranged when you talk with me. During this meeting, all aspects of the situation will be discussed. You may be asked to enter into a Consent Order with the Department to formalize your plan and schedule of corrective action and to settle any outstanding issues regarding this matter, including the payment of civil charges.

Sincerely,

Kelly E. Lease
Environmental Inspector Senior

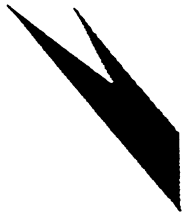
Cc: Compliance File
Charles Forbes, ACM-NVRO
John Bowden, DRD-NVRO
Michael G. Dowd, DOE-CO

ATTACHMENT 8

Mirant Mid-Atlantic, LLC
901 F Street NW, Suite 800, Washington, D.C., 20004
T 202-585-3800 F 202-585-3704

September 19, 2003

Ms. Kelly E. Lease
Commonwealth of Virginia
Department of Environmental Quality
Northern Virginia Regional Office
13901 Crown Court
Woodbridge, VA 22193-1453



MIRANT

Dear Ms. Lease:

I am writing on behalf of Mirant Potomac River, LLC ("Mirant") in response to the Notice of Violation issued by the Department of Environmental Quality ("the Department" or "DEQ") on September 10, 2003 alleging violations of Conditions 3 and 4 of Mirant's Virginia Stationary Source Permit to Operate the Potomac River Generating station ("Operating Permit"). In broad overview, Mirant contends: 1) that the Conditions of the Operating Permit it is alleged to have violated are void and unenforceable for the 2003 ozone season; and 2) that in issuing the NOV, the Department has reversed the position taken in on-going negotiations with Mirant, upon which Mirant relied throughout the Summer to its detriment. As a result, DEQ jeopardizes significant environmental improvements Mirant has offered to make and other benefits to the community.

Background

When the Department proposed issuing the Potomac River Operating Permit on April 24, 2000, it published a "Statement of Basis" that articulated the reasons for permit issuance. As the Statement demonstrates, the Department was utilizing this permit to accomplish compliance with Virginia's Clean Air Act ("CAA") obligations to respond to EPA's call for a State Implementation Plan ("SIP call"), to address the Washington area's nonattainment with the ozone standard and to comply with obligations imposed as a result of Northern Virginia being subject to EPA's companion rulemaking promulgated pursuant to CAA § 126. (Statement of Basis at 1-2, attached as Ex. A).

Consistent with the requirements of the SIP Call and CAA § 126 rulemaking, the Operating Permit provided for "cap" and "trade" compliance. Recognizing the success that the Northeast States (including Maryland) have had with their regional "cap and trade" program[1], EPA adopted the cap and trade approach in both its SIP Call and § 126 programs. The cap and

[1] Virginia elected not to sign the Memorandum Of Understanding for the Northeast Ozone Transport Region and thus did not promulgate regulations pursuant to that agreement. *Ozone Transport Commission Memorandum of Understanding 94-1.* However, Virginia is expressly included in both the expanded region subject to the SIP Call rule and CAA § 126 rule issued by EPA.

trade approach allows a company to *elect* either to reduce emissions to a cap level utilizing add-on pollution control technology or to acquire "allowances" from other facilities through trading to cover emissions that exceed the cap. This cap and trade option allows companies the flexibility to install the most advanced and expensive controls on selected plants to achieve the greatest reduction of NOx emissions and to obtain allowances through trading for those plants that will exceed the emission cap. Under a cap and trade program, while some plants will emit above the 0.15 lb/MM Btu emission rate target (acquiring allowances for the excess), others will emit below that level and generate allowances to trade. In this way, the affected sources *average* the targeted emission rate on a regional basis, which is the objective of the program.

When Virginia issued the Potomac River Operating Permit, it established the "cap" in Conditions 3 and 4 of the permit by applying the SIP Call/CAA § 126 regional NOx emissions rate goal of 0.15 lb/MM Btu to the Potomac River units, which calculated as 1019 tons of NOx per ozone season. The permit established the "trade" feature of the cap and trade program in Condition 7, which reads:

> As an alternative to compliance with Condition 3, the permittee may comply with 40 CFR Part 97 or a regulation of the Board approved by EPA as meeting the requirements of 40 CFR Part 96. This condition may be implemented for the units covered by either of the cited regulations once they become effective. The DEQ reserves the right to amend this permit as may be necessary should it determine that the use of this alternative compliance measure will prevent the attainment or maintenance of the air quality standards in the Washington, DC Ozone Nonattainment area.

There can be no doubt that Condition 7, quoted above, was meant to provide the plant with flexibility on how to comply. As DEQ wrote in the Statement of Basis with respect to this feature of the Operating Permit:

> PEPCO [succeeded by Mirant] may control its NOx emissions by any means it chooses, so long as emissions of other pollutants are not significantly increased. The reductions necessary to stay within the emissions cap for the Potomac River G.S. may even be taken at other facilities and credited to the Potomac River G.S. if they are reductions that would benefit the air quality within the Washington, DC nonattainment area and are not otherwise required. As of this writing, an SAPCB rule is under development that will authorize trading of allowed NOx emissions ("allowances") to meet federally-imposed emissions budgets such as in the Section 126 Petition Rule. That rule may be utilized by PEPCO to comply with this permit, but only to the extent that it benefits the Washington, DC area. This flexibility allows PEPCO to contribute to improvement of the DC area air quality by the most cost-effective means with the least disruption of services.

(Statement of Basis at 3).

At the time this permit was issued on September 18, 2000 the regulations at 40 CFR Part 97 provided for a cap and trade program that established allowance trading beginning in 2003. It was known at the time that the Potomac River plant would be subject to the federally-mandated "cap and trade" program by way of regulation anyway and so there was no reason to object to inclusion of these provisions in the Operating Permit. Similarly, when Mirant acquired the Potomac River plant from PEPCO in December, 2000, allowance trading in 2003 was provided under 40 CFR Part 97 and thus available to Mirant for Potomac River by virtue of Condition 7 of the permit quoted above.

Mirant's generating asset acquisition from PEPCO included all of the Washington area plants, the largest of which are located in Maryland. Mirant developed a broad strategy for compliance with the requirements of the impending regional SIP Call under which Potomac River would obtain allowances for emissions above the cap and the largest initial emission reductions would occur at Mirant's Maryland plants—all of which are in the Washington ozone non-attainment area. This strategy complied fully with the cap and trade program that was anticipated to begin in 2003.

On April 30, 2002—a little over one year ago—as a result of litigation delays associated with the SIP Call and CAA § 126 rulemakings, EPA changed the cap and trade program in 40 CFR Part 97 to provide for a commencement date of May 31, 2004. 67 FR 21522, 21524 (April 30, 2002). The implementing regulations promulgated by Virginia similarly provided for a commencement date of 2004 for the Virginia cap and trade program. 9 VAC 5-140-60C(3). While the 2004 commencement date benefited utilities in the states subject to the SIP Call (including VA), this change left Mirant with a "cap" obligation under Conditions 3 and 4 of its Operating Permit that commenced in 2003, but no "trade" opportunity because Condition 7 of the Operating Permit cross-referenced federal and state regulations that delayed the cap and trade program until 2004. Neither the federal mandate nor Mirant's Operating Permit (as evidenced by the Statement of Basis) ever contemplated imposition of a "cap" without a trading opportunity.

Conditions 3 and 4 of the Permit Are Void and Unenforceable for the 2003 Ozone Season

The practical effect of the elimination of the trading opportunity under the permit was to place Mirant Potomac River in an untenable position where it was not realistically *possible* to comply with the 2003 permit "cap." April 30, 2002—when the trading opportunity was eliminated by operation of law—was too late for Mirant to comply in the Summer of 2003 by any means. Absent a trading opportunity, Mirant Potomac River would have had to reduce electricity generation during the Summer, 2003 by 60% to comply with this cap, without a trading opportunity. Obviously a permit provision that amounted to reducing power production by more than half during the peak demand season of the year would have been appealed by the permittee. In this case, however, the permit was modified by operation of law which left Mirant with no opportunity to appeal. The Virginia law guarantees a permittee the right to appeal unacceptable permit conditions. 9 VAC § 5-170-160 (2003). The draconian modification of this permit without opportunity to appeal the change is a violation both of Virginia law and due process. 9 VAC § 5-80-1000, *Tennessee Valley Auth. v. Whitman*, 336 F.3d 1236 (11thCir.

2003) The bringing of an enforcement proceeding based on this draconian permit modification would just add insult to injury.

To reach the extremely low NOx emission levels prescribed by this permit cap without reducing electricity generation by 60% would have required the use of Selective Catalytic Reduction ("SCR") technology on many if not all of the five Potomac River units. Only SCR technology has pollutant removal efficiencies that would accomplish this level of reduction at the Potomac River units. Even with adequate time, Mirant could not comply in this way. SCR is not a feasible alternative at this site because of the physical space limitations. Moreover, SCR would cost approximately $100 million and would not yield NOx emission reductions as favorable as retrofitting on other Washington area plants with SCR technology. Thus, SCR at Potomac River is neither feasible nor prudent. Even if it were feasible, SCR retrofitting at a fifty year old coal plant such as Mirant Potomac River is hugely complex, requiring a minimum of two years for the engineering, development of specifications, custom parts ordering and installation during a Spring or Fall outage period. As EPA wrote in relation to the SIP Call, "lead time for installation of controls on complicated SCR retrofits at facilities with up to six boilers ranges from 21 to 34 months." 63 FR 57448 (October 27, 1998). Consequently, the modification in 2002 of Mirant Potomac River's Operating Permit to eliminate the "trading" option made it practically impossible for Mirant to comply with these conditions of the Operating Permit in 2003 by any means and deprived Mirant of any opportunity to appeal.

In an analogous situation in Maryland, the state court invalidated Maryland's first cap and trade regulation because the Maryland Department of the Environment had required BGE to comply within one year of promulgation or face enforcement. The trial judge invalidated those regulations from the bench, stating:

> But I do want them to take serious consideration of the fact that it's physically impossible for these utilities to comply by May 1st, 1999 and that to impose penalties automatically for failure to comply, to me seems grossly unfair and wrong to comply by that date.

(Order and Excerpt of bench ruling, attached as Ex. B.)

DEQ cannot modify Mirant's Operating Permit (even by operation of law) in the draconian manner that it has without giving Mirant an opportunity to appeal. Likewise, it cannot sustain a civil enforcement proceeding for violation of a permit requirement with which Mirant could not realistically comply as a result of the elimination of the trading opportunity. An effort to do so would be a violation of Virginia law and administrative due process. *United States v. Hoechst Celanese Corp.*, 128 F.3d 216, 224 (4th Cir. 1997), *Cf. Tennessee Valley Auth., supra.*

DEQ Has Precipitously Reversed Its Position and Jeopardized Major Environmental Benefits Offered by Mirant

When Mirant became aware of the delay in its trading opportunity, it entered into discussions with DEQ regarding an alternative approach to compliance. Mirant was reassured by

DEQ that the disparity between the 2003 "cap" requirement and 2004 "trading" opportunity could be addressed either in the context of Mirant's superseding Title V operating permit or as a separate agreement with DEQ. In the course of those discussions, DEQ offered Mirant a higher "cap" to cover its emissions at the Potomac River plant in the Summer, 2003. As of July 1, 2003, DEQ offered an cap of 1807 tons and, most recently, in exchange for further reductions to which Mirant was willing to commit at its Maryland plants, DEQ agreed to a cap of 2336 tons at Potomac River for the Summer, 2003 ozone season. The 2336 cap represented a 10% reduction in emissions from the prior year. Mirant has honored these discussions and been careful to maintain its operations so as to comply with the 2336 cap that had been agreed to in principle by DEQ. In fact, at Mirant's current pace, it projects that the ozone season NOx emissions at this plant will be in the 2100 ton range, representing almost a 20% reduction from the prior year.

To justify the 2336 ton cap at Potomac River, Mirant had offered further reductions of the NOx emissions from the Maryland plants during the 2003 ozone season. Mirant offered reductions of approximately 20%, but in fact will have achieved NOx emission reductions in the 35% range during the 2003 ozone season at those plants.

Perhaps most significantly, as a result of the issuance of this NOV near the end of the 2003 ozone season in which DEQ has totally reversed its position, other significant local environmental improvements offered by Mirant are in jeopardy. Specifically, Mirant had offered to install Separated Over-fired Air (SOFA) pollution controls, the first of which would commence operation in 2004. In the context of a larger settlement, Mirant had offered to follow-up with installation of SOFA at two additional Potomac River units. In addition, Mirant had offered to fund a significant environmental initiative in the DC Ozone Nonattainment area. These considerable environmental benefits to the community are not legally required and are at risk as a result of DEQ's action.

For all of the above reasons, Mirant Potomac River asserts that it has not violated its permit. Although it would prefer not to have to do so, Mirant is prepared to defend this position in an enforcement proceeding if necessary. Nevertheless, Mirant requests the opportunity to meet with DEQ to discuss these issues and is prepared to honor the offers it made and continue its on-going discussions with DEQ with a view toward resolving outstanding issues.

Sincerely,

Wesley L. McNealy
Director of Environmental, Safety and Health

CC: Michael G. Dowd - VADEQ

# EXHIBIT A

**COMMONWEALTH OF VIRGINIA**
**DEPARTMENT OF ENVIRONMENTAL QUALITY**

STATEMENT OF BASIS

Of the
State Operating Permit for the
Potomac Electric Power Company (PEPCO)
Potomac River Generating Station

To Implement the $NO_x$ Emission Reductions of the Proposed
State Implementation Plan (SIP) Revision,
Phase II Attainment Plan for the
Washington DC-MD-VA Ozone Nonattainment Area

By John R. McKie, PE

April 24, 2000

## Background

Section 110 of the Clean Air Act requires each state to submit a state implementation plan (SIP) for bringing all areas into "attainment" of the National Ambient Air Quality Standards (NAAQS) and for maintaining attainment of the NAAQS once attainment has been achieved. Due to the common failure of many areas, including the Washington, DC metropolitan area and the Northern Virginia portion thereof, to attain the one-hour NAAQS for ozone, the Clean Air Act Amendments (CAAA) of 1990 imposed specific measures and schedules to attain the standard on the ozone "nonattainment" areas. These measures and schedules vary, depending on the severity of the nonattainment status. On the basis of monitored ambient concentrations, the CAAA defined the Washington, DC area as being a "serious" nonattainment area. Section 181 of the Clean Air Act set the attainment date for a serious nonattainment area as November 15, 1999.

Monitoring data has not demonstrated that the Washington, DC area has achieved attainment. However, air quality modeling performed on behalf of the jurisdictions composing that area has indicated that with the measures proposed for inclusion into the relevant SIP's the area would be in attainment if it were not for the transport of ozone and its precursor pollutants from outside the Washington, DC area. The U.S. Environmental Protection Agency (EPA) is taking action to control long-range transport of ozone and its precursors by 2003. It takes three consecutive years of monitoring data to demonstrate compliance. Therefore, with the proposed measures in the SIP's and the EPA action against long-range transport, the Washington, DC area should be able to demonstrate compliance by 2005. One of these measures, which EPA (Region-III office) has already indicated that it would accept as adequate, is the

limiting of nitrogen oxides ($NO_x$) emissions from the area electric utility plants to 0.15 pounds per million Btu's of heat (fuel) input to the boilers. This permit has been created as a vehicle for implementing this measure at the Potomac Electric Power Company's (PEPCO) Potomac River Generating Station in Alexandria, Virginia.

The Potomac River G.S. consists of five coal-fired boilers; two rated at a heat input of 970.1 million Btu's per hour, the other three at 980.7 million Btu's per hour. $NO_x$ emissions from these units are currently limited by an "Acid Rain" (Clean Air Act Title IV) permit to an annual average of 0.45 pounds per million Btu's of heat input. The $NO_x$ emissions as a whole for the PEPCO system are limited by a consent agreement implementing Reasonably Available Control Technology (RACT) for the Potomac River G.S. Most of the units in the system, all of which are in the Washington, DC ozone nonattainment area, have assigned $NO_x$ RACT emission rate limits on which the total system limit is based. Taking into consideration the daily heat inputs of each unit, the total daily emissions from the system cannot exceed those that would result if each unit were meeting its assigned RACT emission rate limit, yet individually, units are not required to meet their assigned RACT limits. The assigned limit for each of the Potomac River G.S. units is 0.38 pounds per million Btu's. This limit is the same as the general RACT limit found in State Air Pollution Control Board (SAPCB) regulation *9 VAC 5-40-311* for dry bottom, coal-fired (tangential or face-fired) boilers.

**Implementation**

The instrument that the Virginia Department of Environmental Quality (DEQ) has chosen to enforce this proposed measure is a state operating permit. SAPCB regulation 9 VAC 5-80-800 C.2.b. allows the use of such a permit to "establish a source-specific emission standard or other requirements necessary to implement the federal Clean Air Act or the Virginia Air Pollution Control Law." A permit issued for this reason requires no application. This state operating permit contains no provisions other than those relevant to the ozone attainment plan. Eventually, the conditions of the state operating permit will be rolled into the federal operating ("Title V") permit for the facility.

Issuance of a state operating permit can occur without public participation; however, a revision of the SIP cannot. This permit is being issued as an implementation tool of a SIP revision. In this case, the Washington, DC area ozone attainment plan, therefore, this permit (in draft form) is being subjected to public comment. Following consideration by DEQ of the comments received, the draft permit will be revised if warranted and issued. The permit and supporting documentation will be forwarded to EPA for final approval as satisfactory to implement the attainment plan SIP revision. PEPCO or any future owner of the facility will have to begin meeting the requirements of the permit by May 2003.

Section 126 of the Clean Air Act allows states to petition the EPA to take action against sources of $NO_x$ outside their borders if those sources prevent the petitioning states from achieving or maintaining compliance with the ozone standard. In response to four such petitions, EPA in December, 1999, issued a rule which set limits, also

known as "caps," on the amount of $NO_X$ that many Southern and Midwestern power plants, including PEPCO's Potomac River G.S. may emit during the high-ozone season (May 1 through September 30). These caps were set for each individual boiler unit and based on operation occurring at emission rates of 0.15 pounds per million Btu of heat input. Therefore, by setting a caps on $NO_X$ emissions that satisfy the Section 126 Petition Rule, a permit can satisfy the requirements of the attainment plan as well as the Section 126 Petition Rule. The requirements of the "126" rule are actually a little more stringent, because the attainment plan emissions limits are based on total facilities and not the individual units the facilities comprise. PEPCO has stated its desire to have a permit limit that demonstrates compliance with both sets of requirements. As of this writing, the Section 126 Petition Rule is in litigation; therefore, at this time it is not appropriate to issue a permit that includes enforceable requirements of that rule. However, the $NO_X$ limit in this permit is the same as the total of the individual unit limits in the Section 126 Petition Rule, so if and when the "126" rule is enforced as written, there will be no conflict.

PEPCO may control its $NO_X$ emissions by any means it chooses, so long as emissions of other pollutants are not significantly increased. The reductions necessary to stay within the emissions cap for the Potomac River G.S. may even be taken at other facilities and credited to the Potomac River G.S. if they are reductions that would benefit the air quality within the Washington, DC nonattainment area and are not otherwise required. As of this writing, an SAPCB rule is under development that will authorize trading of allowed $NO_X$ emissions ("allowances") to meet federally-imposed emissions budgets such as in the Section 126 Petition Rule. That rule may be utilized by PEPCO to comply with this permit, but only to the extent that it benefits the Washington, DC area. This flexibility allows PEPCO to contribute to improvement of the DC area air quality by the most cost-effective means with the least disruption of services.

## Permit Contents

Condition Number

1. States the purpose and authority for issuing the permit. The Clean Air Act citation given is the basis for EPA requiring that Virginia submit an ozone attainment plan. The state regulation cited authorizes the SAPCB to issue a state operating permit to establish a source-specific emission standard or other requirements necessary to implement the federal Clean Air Act.

2. Specifies the emitting units to which the permit conditions apply. In this case, the units are all of the boilers supplying steam for electric power generation.

3. Sets the $NO_X$ emission limit for the facility and the period during which it applies. The limit is a cap on the total emissions, determined by summing the Section 126 Petition Rule individual unit limits, which were based on an emission rate of 0.15

pounds per million Btu of heat input. The limit, therefore, satisfies the Washington, DC area ozone attainment plan requirement for the facility to meet 0.15 pounds per million Btu of heat input. The limit is only in effect during the ozone season.

4. States compliance will be determined by continuous emissions monitoring and the due date by which the demonstration must begin. The date, year 2003, is the beginning of the three-year compliance demonstration period for year 2005 as specified in the Washington, DC area ozone attainment plan. PEPCO already has continuous emissions monitors (CEM's) for purposes of determining compliance with acid rain and reasonably available control technology (RACT) provisions of the Clean Air Act.

5. Covers monitoring, record keeping, and reporting requirements. Details of these are to be set by DEQ within 60 days following issuance of the permit, which is expected to be well before compliance with the emissions limit will begin. It is desirable not to set details of the compliance verification in this permit, so that DEQ may minimize the administrative burden and time requirements of subsequently improving the compliance methodology, if desired. PEPCO will have the opportunity to provide input to DEQ regarding setting the requirements.

6. Specifies duration of records retention. The state operating permit rule allows DEQ to require retention for three or more years. Because the Title V rule, to which this facility is subject, requires at least five years of retention, this permit also requires five years.

7. Allows emissions trading as a means of demonstrating compliance. This is discussed in the last paragraph of the preceding section. It should be noted that while the condition grants PEPCO the right to use emissions trading, such trading must be deemed by DEQ as consistent with the ozone attainment plan purpose, namely improvement of air quality in the Washington, DC nonattainment area.

8. Requires that PEPCO notify any new owner of the facility about this permit and send a copy of the notice to DEQ. DEQ would then make the necessary administrative amendments to the permit to show that it is transferred to the new owner.

9. States that a copy of the permit must remain on the premises. Besides being a regulatory requirement, it serves as a reminder to the facility staff of its obligations under the attainment plan as well as assuring the availability of inspection of the permit by DEQ personnel and others.

# EXHIBIT B

| | | |
|---|---|---|
| **BALTIMORE GAS AND ELECTRIC COMPANY, et al.** | * | **IN THE** |
| | * | **CIRCUIT COURT** |
| **Plaintiffs,** | * | **FOR** |
| **v.** | * | **BALTIMORE CITY** |
| **MARYLAND DEPARTMENT OF THE ENVIRONMENT, et al.,** | * | **Consolidated Case Nos. 98170117-CC5318 and** |
| **Defendants.** | * | **98225118-CC7049** |

* * * * * * * * * * * * *

## ORDER

Upon consideration of the Motions for Summary Judgment filed by plaintiffs, Baltimore Gas and Electric Company and Potomac Electric Power Company, the responses thereto, the Motion for Judgment filed by defendants, Jane T. Nishida and the Maryland Department of the Environment, and following oral argument, it is this 24th day of February, 1999

ORDERED, pursuant to § 10-125(d) of the State Government Article of the Maryland Code that Code of Maryland Regulation 26.11.27.04A, imposing a compliance deadline of May 1, 1999, is declared invalid and Regulations 26.11.27 and .28 are hereby remanded to the Maryland Department of the Environment.

HONORABLE JOSEPH H.H. KAPLAN
**********
THE JUDGE'S SIGNATURE APPEARS
ON ORIGINAL DOCUMENT

cc: Counsel of Record

bgepepco.ord

TRUE COPY
TEST
FRANK [illegible], CLERK

CIRCUIT COURT FOR BALTIMORE CITY

| | | |
|---|---|---|
| BALTIMORE GAS AND ELECTRIC COMPANY AND POTOMAC ELECTRIC POWER COMPANY, Plaintiffs, V. JANE T. NISHIDA, SECRETARY OF THE ENVIRONMENT, AND THE MARYLAND DEPARTMENT OF THE ENVIRONMENT, Defendants. | ) | Case Nos. 98225118/CC7049<br>98170117/CC5328 |

REPORTER'S OFFICIAL TRANSCRIPT OF PROCEEDINGS
(Motions Hearing)

Tuesday, February 9, 1999

BEFORE:

THE HONORABLE JOSEPH H. H. KAPLAN, CHIEF JUDGE

APPEARANCES:

For the plaintiff BGE:
DEBORAH JENNINGS, ESQ.

For the plaintiff PEPCO:
WILLIAM BROWNELL, ESQ.

For the defendants:
KATHY KINSEY, ESQ.
VICKIE LYNN GAUL, ESQ.

Recorded on videotape

TRANSCRIBED BY:
Charles F. Madden
Official Court Reporter
507 Courthouse West
100 North Calvert Street
Baltimore, Maryland 21202

that is what the Judge intends to do, the Court intends to do, that it simply remand the rule back to the agency.

MR. BROWNELL: Your Honor, if the rule is remanded to the agency, we believe it's important that Your Honor also instruct the agency to take this compliance deadline issue seriously.

We have provided ways to work with the compliance deadline issue, and I believe we would be willing to work with the agency to try and work this issue out on the remand.

MS. JENNINGS: And BGE would, as well, Your Honor. We would prefer a remand and to try to work it out.

THE COURT: Now, since both sides would prefer a remand, rather than my tampering with the rule, the regulation as it presently exists, I will do just that. I will remand it to the Commission.

But I do want them to take serious consideration of the fact that it's physically impossible for these utilities to comply by May 1st, 1999 and that to impose penalties automatically for failure to comply, to me, seems grossly unfair and wrong to comply by that date.

So that they ought to get another date that

ATTACHMENT 9

# GLOSSARY OF AIR POLLUTION TERMS

(Definitions adapted from Georgia Department of Environmental Protection Web Site)

**Attainment area** -- A geographic area in which levels of a criteria air pollutant meet the health-based primary standard (national ambient air quality standard, or NAAQS) for the pollutant. An area may have on acceptable level for one criteria air pollutant, but may have unacceptable levels for others. Thus, an area could be both attainment and non-attainment at the same time. Attainment areas are defined using federal pollutant limits set by EPA.

**BACT** -- Best available control technology. It is an emission limitation that considers the cost of energy, environment, and economics in developing a degree of emission reduction that is achievable through application of good production processes, control systems, and techniques. In no event can BACT allow emissions of a pollutant in excess of a NSPS or a NESHAPS. BACT is determined on a case-by-case basis, is applied to each pollutant regulated under the Clean Air Act (federal).

**Carbon monoxide (CO)** -- A colorless, odorless, poisonous gas, produced by incomplete burning of carbon-based fuels. including gasoline, oil, and wood. Carbon monoxide is also produced from incomplete combustion of many natural and synthetic products. For instance, cigarette smoke contains carbon monoxide. When carbon monoxide gets into the body, the carbon monoxide combines with chemicals in the blood and prevents the blood from bringing oxygen to cells, tissues, and organs. The body's parts need oxygen for energy, so high-level exposures to carbon monoxide can cause serious health effects. Massive exposures to CO can cause death. Symptoms of exposure to carbon monoxide can include vision problems, reduced alertness, and general reduction in mental and physical functions. Carbon monoxide exposures are especially harmful to people with heart, lung, and circulatory system diseases.

**Clean Air Act (CAA)** -- The original Clean Air Act was passed in 1963, but our national air pollution control program is actually based on the 1970 version of the law. The 1990 Clean Air Act Amendments are the most far-reaching revisions of the 1970 law. In this glossary, we refer to the 1990 amendments as the 1990 Clean Air Act.

**Clean fuels** -- Low-pollution fuels that can replace ordinary gasoline. These are alternative fuels, including gasohol (gasoline-alcohol mixtures), natural gas, and LPG (liquefied petroleum gas).

**Combustion** -- Burning. Many important pollutants, such as sulfur dioxide, nitrogen oxides, and particulates (PM-10 or PM 2.5) are combustion products, often products of the burning of fuels such as coal, oil, gas, and wood.

**Continuous emission monitoring systems (CEMS)** -- Machines, which measure, on a continuous basis, pollutants released by a source. The 1990 Clean Air Act requires continuous emission monitoring systems for certain large sources.

**Control technology; control measures** -- Equipment, processes, or actions used to reduce air pollution. The extent of pollution reduction varies among technologies and measures. In general, control technologies and measures that do the best job of reducing pollution will be required in

the areas with the worst pollution. For example, the best available control technology/best available control measures (BACT, BACM) will be required in serious non-attainment areas for particulates, a criteria air pollutant. A similar high level of pollution reduction will be achieved with maximum achievable control technology (MACT) which will be required for sources releasing hazardous air pollutants.

**Criteria air pollutants** -- A group of very common air pollutants regulated by EPA on the basis of criteria (information on health and/or environmental effects of pollution). Criteria air pollutants are widely distributed all over the country. A National Ambient Air Quality Standard exists for each criteria pollutant (particulate matter, sulfur dioxide, nitrogen dioxide, ozone, carbon dioxide, and lead).

**Emission** -- Release of pollutants into the air from a source. We say sources emit pollutants. Continuous emission monitoring systems (CEMS) are machines, which some large sources are required to install, to make continuous measurements of pollutant release.

**Enforcement** -- The legal methods used to make polluters obey the Clean Air Act. Enforcement methods include citations of polluters for violations of the law (citations are much like traffic tickets), fines, and even jail terms. EPA and the state and local governments are responsible for enforcement of the Clean Air Act, but if they don't enforce the law, members of the public can sue EPA or the states to get action. Citizens can also sue violating sources, apart from any action EPA or state or local governments have taken. Before the 1990 Clean Air Act, all enforcement actions had to be handled through the courts. The 1990 Clean Air Act gave EPA authority so that, in some cases, EPA can fine violators without going to court first. The purpose of this new authority is to speed up violating sources' compliance with the law and reduce court time and cost.

**Hazardous air pollutants (HAPs)** -- Toxic chemicals that cause serious health and environmental effects. Health effects include cancer, birth defects, nervous system problems, and death due to massive accidental releases such as occurred at the pesticide plant in Bhopal, India. Hazardous air pollutants are released by sources such as chemical plants, dry cleaners, printing plants, and motor vehicles (cars, trucks, buses, etc.)

**Major source** -- Under the PSD regulations it is a facility, belonging to one or more of 28 source categories, having the potential to emit 100 tons per year of a pollutant regulated under the federal Clean Air Act (CAA). For categories other than the 28 sources, the potential emission level can not exceed 250 tons per year. A major source in a nonattainment area (ozone Washington Metro) has the potential to emit 50 tons per year of the pollutant for which the area is in nonattainment (for ozone area we use the pollutants volatile organic compounds and nitrogen dioxide). Also, a Title V source. A major source for the purpose of Title V in the CAA is a stationary source that has the potential to emit 100 tons per year a pollutant regulated under the CAA and/or a source that has the potential to emit 10 tons per year for a single hazardous air pollutant or 25 tons per year of a combination of all hazardous air pollutants.

**Material safety data sheets (MSDS)** -- Product safety information sheets prepared by manufacturers and marketers of products containing toxic chemicals. These sheets can be obtained by requesting them from the manufacturer or marketer. Some stores, such as hardware

stores, may have material safety data sheets on hand for products they sell.

**Mobile sources** -- Motor vehicles and other moving objects that release pollution; mobile sources include cars, trucks, buses, planes, trains, motorcycles and gasoline-powered lawn mowers. Mobile sources are divided into two groups: road vehicles, which includes cars, trucks, and buses, and non-road vehicles, which includes trains, planes, and lawn mowers.

**Monitoring (monitor)** -- Measurement of air pollution is referred to as monitoring. EPA, state, and local agencies measure the types and amounts of pollutants in community air. The 1990 Clean Air Act requires states to monitor community air in polluted areas to determine if the areas are being cleaned up according to schedules set by law.

**NAAQS** -- National ambient air quality standards. Ambient standards developed by EPA that must be attained and maintained to protect public health. "Secondary" NAAQS are necessary to protect the public welfare. NAAQS exist for particular matter, sulfur dioxide, nitrogen dioxide, ozone, carbon dioxide, and lead.

**Nitrogen oxides (NOx)** -- A criteria air pollutant. Nitrogen oxides are produced from burning fuels, including gasoline and coal. Nitrogen oxides are smog formers, which react with volatile organic compounds to form smog. Nitrogen oxides are also major components of acid rain.

**Non-attainment area** -- A geographic area in which a criteria air pollutant level is higher than allowed by the federal standards. A single geographic area may have an acceptable level for one criteria air pollutant, but have unacceptable levels of one or more other criteria air pollutants. Thus, an area can be both an attainment and non-attainment area at the same time. Sixty percent of Americans are estimated to live in non-attainment areas.

**NSR** -- New source review. NSR means any new source locating in ozone nonattainment area that will emit volatile organic compounds (VOC) and /or oxide of nitrogen (NO) in certain amounts. These sources must: under go a new source review that provides for offsetting emissions for any increases in the emissions of these two pollutants; use the lowest achievable emissions technology to control emissions; apply for a construction permit; and meet other state requirements before the new emission from the source can be permitted. Existing sources, located in the ozone nonattainment area, that emit these two pollutants and plan a change their operational methods that will cause an increase in the emissions of these two pollutants must apply for a modification permit and under go a review similar to a new source.

**NSPS** -- New source performance standards. These are federal EPA emission standards for certain air pollutants that are emitted from new, modified, or reconstructed stationary emission sources which reflect the use of best available control technology

**NESHAPS** -- National Emission Standards for Hazardous Air Pollutants.

**Offset** -- A method used in the 1990 Clean Air Act to give companies, which own or operate large (major) sources in non-attainment areas, flexibility in meeting overall pollution reduction requirements when changing production processes. If the owner or operator of the source wishes to increase releases of a criteria air pollutant, an offset (reduction of a somewhat greater amount of the same pollutant) must be obtained either at the same plant or by purchasing offsets from

another company.

**Oxygenated Fuel (Oxyfuel)** -- Special type of gasoline, which burns more completely than regular gasoline in cold start conditions; more complete burning results in reduced production of carbon monoxide, a criteria air pollutant. In some parts of the country, carbon monoxide release from cars starting in cold weather makes a major contribution to pollution. In these areas gasoline refiners must market oxygenated fuels, which contain a higher oxygen content than regular gasoline. Some gasoline companies sold oxyfuels in cities with carbon monoxide problems before the 1990 Clean Air Act was passed.

**Ozone** -- A gas which is a variety of oxygen. The oxygen gas found in the air consists of two oxygen atoms stuck together; this is molecular oxygen. Ozone consists of three oxygen atoms stuck together into an ozone molecule. Ozone occurs in nature; it produces the sharp smell you notice near a lightning strike. High concentrations of ozone gas are found in a layer of the atmosphere -- the stratosphere -high above the Earth. Stratospheric ozone shields the Earth against harmful rays from the sun, particularly ultraviolet B. Smog's main component is ozone; this ground-level ozone is a product of reactions among chemicals produced by burning coal, gasoline and other fuels, and chemicals found in products such as solvents, paints, and hair sprays.

**Ozone hole** -- Thin place in the ozone layer located in the stratosphere high above the Earth. CFCs and related chemicals have linked stratospheric ozone thinning to destruction of stratospheric ozone. The 1990 Clean Air Act has provisions to reduce and eliminate the production and use of ozone destroying chemicals. Ozone holes have been found above Antarctica and above Canada and northern parts of the United States, as well as above northern Europe.

**Particulates: particulate matter** Particulate matter (PM) refers to all solid and liquid particles found in the air. Attachment 3 summarizes the size characteristics of particles and particle dispersoids. A size of 1μ is equal to a thousandth of a millimeter or millionth of a meter. $PM_{2.5}$ refers to particulates with size less than 2.5μ and particulates with size less than 10μ are referred as $PM_{10}$. The particles smaller than 2.5μ (microns) are respirable and have a greater chance of getting deposited in the lungs and thus considered to have greater impacts compared to larger or coarse particles. The main sources of fine particulate matter in urban areas are activities involving the combustion process, specifically, point sources such as power plants, area sources such as industrial boilers, commercial and home heating (including wood burning stoves), and mobile sources such as buses, trucks, automobiles trains, and aircraft. The current National Ambient Air Quality Standard (NAAQS) for $PM_{2.5}$ is an annual mean of 15 $\mu g/m^3$ (micrograms per cubic meter of air). The NAAQS for $PM_{10}$ is an annual mean of 50 $\mu g/m^3$. Alexandria and the metropolitan Washington area are currently meeting these standards.

**Permit** -- A document that resembles a license that is required by the Clean Air Act for big (major) sources of air pollution, such as power plants, chemical factories and, in some cases, smaller polluters. Usually permits are issued by states, but if EPA has disapproved part or all of a state permit program, EPA will issue the permits in that state. The 1990 Clean Air Act includes

requirements for permit applications, including provisions for members of the public to participate in state and EPA reviews of permit applications. Permits contain information on all the regulated pollutants at a source. Permits include information on which pollutants are presently released, how much pollution the source is allowed to release, and the control measures necessary to meet pollutant release requirements. Permits are required both for the operation of plants (operating permits) and for the construction of new plants. The 1990 Clean Air Act introduced a nationwide permit system for air pollution control.

**Pollutants (Pollution)** -- Unwanted chemicals or other materials found in the air. Pollutants can harm health, the environment and property. Many air pollutants occur as gases or vapors, but some are very tiny solid particles: dust, smoke, or soot.

**Primary standard** -- A pollution limit based on health effects. Primary standards are set for criteria air pollutants.

**PSD** -- Prevention of significant deterioration. This term refers to regulations that requires a major new source or an existing source making majoring modifications to be permitted by the state before construction is started if they are located in an attainment area.

**RACT** - Reasonably available control technology. It is usually an emission limit set by a state air program and is the basis for emission rates used in their SIP. It usually applies to sources in attainment areas and in most cases is less stringent than the NSPS level of control.

**Reformulated gasoline** -- Specially refined gasoline with low levels of smog-forming volatile organic compounds (VOCs) and low levels of hazardous air pollutants. The 1990 Clean Air Act requires sale of reformulated gasoline in the nine smoggiest areas. Some reformulated gasolines were sold in several smoggy areas before passage of the 1990 Clean Air Act.

**Secondary standard** -- A pollution limit based on environmental effects such as damage to property, plants, or visibility. Secondary standards are set for criteria air pollutants.

**State Implementation Plan (SIP)** -- An accumulation of actions and programs a states carries out to control emissions. This includes such things as rules and regulations, plans to control ozone, and ambient air standards.

**Smog** -- A mixture of pollutants, principally ground-level ozone, produced by chemical reactions in the air involving smog-forming chemicals. A major portion of smog-formers comes from burning petroleum-based fuels such as gasoline. Other smog-formers, volatile organic compounds, are found in products such as paints and solvents. Smog can harm health, damage the environment and cause poor visibility. Major smog occurrences are often linked to heavy motor vehicle traffic, sunshine, high temperatures and calm winds, or temperature inversion (weather condition in which warm air is trapped close to the ground instead of rising). Smog is often worse away from the source of the smog-forming chemicals, since the chemical reactions that result in smog occur in the sky while the wind blows away the reacting chemicals from their sources.

**Source** -- Any place or object from which pollutants are released. A source can be a power plant,

factory, dry cleaning business, gas station, or a farm. Cars, trucks, and other motor vehicles are sources. Consumer products and machines used in industry can also be sources. Sources that stay in one place are stationary sources; sources that move around, such as cars or planes, are called mobile sources.

**State implementation plan (SIP)** -- A detailed description of the programs a state will use to carry out its responsibilities under the Clean Air Act. State implementation plans are collections of the regulations used by a state to reduce air pollution. The Clean Air Act requires that EPA approve each state implementation plan. Members of the public are given opportunities to participate in review and approval of state implementation plans.

**Stationary source** -- A place or object from which pollutants are released which stays in place. Stationary sources include power plants, gas stations, incinerators, and houses.

**Stratosphere** -- Part of the atmosphere, the gases that encircle the Earth. The stratosphere is a layer of the atmosphere 9-31 miles above the Earth. Ozone in the stratosphere filters out a harmful type of sunlight called ultraviolet B, which has been linked to health and environmental damage.

**Sulfur dioxide** -- A criteria air pollutant. Sulfur dioxide is a gas produced by burning coal, most notably in power plants. Some industrial processes, such as production of paper and smelting of metals, produce sulfur dioxide. Sulfur dioxide is closely related to sulfuric acid, a strong acid. Sulfur dioxide plays an important role in the production of acid rain.

**TITLE V PERMIT**- - Is a federal operating permit program adopted and implemented by the VDEQ. The basic program elements specify that major sources in Virginia will submit an operating application to the EPD according to a schedule. EPA and the affected states will review the permit issuance. The pubic also has an opportunity to comment on the permit, which is renewable every five years. Minor changes to the permit can be made without opening the permit for public participation.

**Ultraviolet B (UVB)** -- A type of sunlight. The ozone in the stratosphere filters out ultraviolet B rays and keeps them from reaching the Earth. Ultraviolet B exposure has been associated with skin cancer, eye cataracts, and damage to the environment. Thinning of the ozone layer in the stratosphere results in increased amounts of ultraviolet B reaching the Earth.

**Vapor recovery nozzles** -- Special gas pump nozzles that reduce the release of gasoline vapor into the air gas is pumped into car tanks. There are several types of vapor recovery nozzles. Therefore, nozzles may not look the same at all gas stations. The 1990 Clean Air Act requires the installation of vapor recovery nozzles at gas stations in smoggy areas.

**Volatile organic compounds (VOCs)** -- Organic chemicals all contain the element carbon (C). Organic chemicals are the basic chemicals found in living things and in products derived from living things, such as coal, petroleum, and refined petroleum products. Many of the organic chemicals we use do not occur in nature, but were synthesized by chemists in laboratories. Volatile chemicals readily produce vapors at room temperature and normal atmospheric pressure. Vapors escape easily from volatile liquid chemicals. Volatile organic chemicals include gasoline,

industrial chemicals such as benzene, solvents such as toluene and xylene, and tetrachloroethylene (perchloroethylene, the principal dry cleaning solvent). Many volatile organic chemicals, such as benzene, are also hazardous air pollutants.

ATTACHMENT 10

City of Alexandria, Virginia
301 King Street, Suite 2300
Alexandria, Virginia 22314

William D. Euille
Mayor

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Home (703) 836-2680
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alexvamayor@aol.com

November 7, 2003

The Honorable W. Tayloe Murphy, Jr.
Secretary of Natural Resources
733 Ninth Street Office Building
Richmond, Virginia 23219

Re: Mirant Potomac River Power Generating Station, 1400 North Royal Street, Alexandria, Virginia

Dear Secretary Murphy:

I write to share with you the City of Alexandria's concerns regarding operations at the Mirant Potomac River Power Generating Station which is located in Alexandria. Our concerns stem from the effects that the plant's operations and emissions are, or may be, having on Alexandrians, particularly those residing in the immediate vicinity of the plant.

One concern relates to fine particulate emissions that may be coming from the plant and their health impacts on the community. Studies suggest that particulate matter less than 2.5 microns in size may have adverse health impacts. The City requests that any permit issued by the Virginia Department of Environmental Quality for the Mirant plant, including any Title V permit, adequately address the issues relating to these emissions. We also request that no permit action be taken without the City being able to discuss our concern with your staff.

The City also has a concern relating to fugitive dust emissions that result from operations at the Mirant plant. The City has often received complaints concerning such emissions, which leave the plant property from the plant's coal pile or as a result of coal unloading and flyash loading operations. The DEQ's own analysis of samples taken from the neighborhood and the plant shows that particulates, including coal dust, are leaving the plant site, and comprise up to 50 percent of the dirt and dust found in the adjacent neighborhood (see enclosed copy of the DEQ memorandum, dated June 10, 2003). Before any new or revised permit is issued for the Mirant plant, the City asks that VDEQ review the measures now being used at the plant to control coal and flyash emissions. Improved measures would include the installation of negative air pressure air handling systems, along with appropriate air filters.

A further City concern relates to the NOx emissions from the Mirant plant. The existing State operating permit for the plant, which was issued in 2000, limits NOx emissions during the ozone season. On September 10, 2003, VDEQ issued a Notice of Violation to Mirant for failing to meet these limits. The City is not aware of any current efforts by Mirant to install additional air pollution control equipment or to upgrade or modify its current control equipment to meet these NOx limits.

The City understands that VDEQ is now in negotiations with Mirant regarding its existing operating permit and a future Title V permit. We also understand that these negotiations are addressing the possibility of the Mirant plant being allowed to achieve NOx compliance by trading NOx emissions reductions at other facilities. It is not clear what impact such "trading" may have upon the City, although our initial impression is that the impact may not be favorable. In any event, if "trading" were permitted by VDEQ, we believe that the significant savings Mirant would experience should be used to fund local NOx mitigation measures here in Alexandria.

For these reasons, I ask that, before any action is taken by VDEQ in conjunction with Mirant's current permit, Mirant's NOV, or the issuance of a Title V permit for the Mirant plant, the City be able to address our concerns with your staff.

I appreciate your consideration of this request, and I look forward to your reply. In the meantime, if you have any questions, please contact Richard Baier, the Director of our Department of Transportation and Environmental Services, at 703.838.4966.

Sincerely,

William D. Euille
Mayor

Enclosure

cc: Robert G. Burnley, Director, NVRO, DEQ
Philip Sunderland, City Manager

# COMMONWEALTH OF VIRGINIA
# DEPARTMENT OF ENVIRONMENTAL QUALITY

## MEMORANDUM

TO: Charles D. Forbes (DEQ/NVRO)

FROM: Kelly Lease, Air Compliance Inspector, NVRO

SUBJECT: Analysis of Dust Samples Taken at 1200 and 1202 Pitt Street, Alexandria, Virginia

DATE: June 10, 2003

---

The purpose of this memo is to present the analytical results for dust samples collected in response to citizen complaints regarding particulate emissions from Mirant's Potomac River Generating Station, a coal-fired power plant located within the City of Alexandria. Dust samples were collected from areas in the front and rear of residences at 1200 and 1202 Pitt Street in Alexandria at the request of Ms. Elizabeth Chimento, a resident of 1200 Pitt Street. Her residence is approximately one-quarter mile from the plant.

The samples were collected on April 22, 2003, by Ms. Kelly Lease and Mr. David Hartshorn with the Department's Northern Virginia Regional Office (NVRO). In addition to Ms. Chimento, Mr. Poul Hertel, also an Alexandria resident, was present during the sampling event. The samples were delivered to the Department's Office of Air Quality Assessment, with an unbroken chain of custody.

The samples were visually examined using a polarizing light microscope at 150 times magnification. The results of this examination are attached. To summarize these results and follow up discussions with the analyst of record, uncombusted coal dust was estimated to constitute up to 50 percent of each sample. Coal combustion products were also present, including partially combusted coal and partially fused ash, at approximately 10 percent of each sample. These combustion products appeared to have not been exposed to high temperature. It should be noted that this analysis has the following limitations: a) a particle count was not performed; and b) as black particles, coal dust stands out against lighter particles of other materials. This can result in over-estimating their relative proportion within the sample. The remainder of the constituents in the sample were either biological (e.g., pollen, fungus, algae, stellate hairs) or materials common to urban environments (e.g., asphalt, mineral particulate, rubber, fibers, paint, wood dust).

In reviewing these results, it is important to note that these results cannot be considered quantitative. The sampling methodology used (i.e., simply brushing dust from surfaces into vials) does not support defensible conclusions regarding constituent concentrations in ambient air, nor when materials found in the sample were generated. Furthermore, no chemical analysis was performed on the samples. Simply put, NVRO can make no formal determination regarding the compliance status of the Potomac River Generating Station as a result of this sampling, but the results warrant further discussions with Mirant and closer monitoring of the facility. To this end, and with your approval, NVRO's compliance inspection strategy for the Potomac River Facility will be modified to focus attention on coal-handling procedures and particulate emission control features at the facility. A meeting with Mirant will be scheduled to discuss this report and review the operating records and current conditions at the plant. At this time, the pertinent dust control regulations (9 VAC 5-40-90 - Standard for Fugitive Dust/Emissions) will be reviewed.

cc: Carolyn Stevens (DEQ Office of Air Quality Assessment)
Lalit Sherma (Alexandria Health Department)
Debra Knight (Mirant Mid-Atlantic, LLC)

**Virginia Dept. of Environmental Quality**
**Office of Air Quality Assessment**
**Microscopic Analysis Form**

**Submittal No. M0498**

**Submitted by:** Kelly Lease

**Date sample received:** 5/14/03

**Sample description:** Twelve vials containing sample material collected from 1200 and 1202 Pitt Street in Alexandria, VA, in response to a citizen complaint

**Analytical results:** (Include date of completion of analysis and signature of responsible party)

**Identified:**

1. Vial labeled "Glass Table Deck": Sample moderate to large amounts of asphalt, coal dust, mineral particulate (quartz, biotite, calcite, clay, etc), pollen, fungus, assorted fibers, rubber, coal combustion products
2. Vial labeled "1200 Front Window": Sample contained moderate to large amounts of asphalt, pollen, coal dust, mineral particulate; smaller amounts of algae, paint, partially fused ash, rubber
3. Vial labeled "1202 Window Ledge": Sample contained moderate to large amounts asphalt, mineral particulate, pollen, stellate hairs, coal dust; smaller amounts of coal combustion products, fungus
4. Vial labeled "1202 Entry": Sample contained moderate to large amounts of asphalt, pollen, mineral particulate, coal dust, wood dust, stellate hairs; smaller amounts of assorted fibers, paint, coal combustion products, rubber, partially fused ash
5. Vial labeled "1202" Entryway": Same as #4, including some fungus
6. Vial labeled "1200 Entryway": Same as #4
7. Vial labeled "1200 E Entry": Same as #4
8. Vial labeled "1202 Front Window": Sample contained mostly pollen; also contained asphalt, mineral particulate, coal dust, stellate hairs, paint, coal combustion products, fungus
9. Vial labeled "1200 Side Window": Sample contained mostly pollen; also contained asphalt, coal dust, mineral particulate, rubber, coal combustion products
10. Vial labeled "Rear Windowsill": Sample contained mostly pollen; also contained small amounts of coal dust, coal combustion products, stellate hairs

Two samples, "1200 Front Window" and "Rear Windowsill", were collected on tape, and could not be analyzed. Most of the coal contained in the samples did not appear to have been exposed to any combustion process.

C. M. Stevens
5/30/03

ATTACHMENT 1

# Mirant Power Plant Emissions and Health Effects Report

Elizabeth Chimento
Poul Hertel

*August 20, 2003*

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H. Virginia State Air Pollution Control Board Regulations (9 VAC 5-40-90)
I. Aust, Ann. Effects of Metals Bound to Particulate Matter on Human Lung Epithelial Cells. *Health Effects Institute* Synopsis
J. Reanalysis of the Harvard Six Cities Study and the American Cancer Society Study of Particulate Air Pollution and Mortality. *Health Effects Institute* Synopsis

IX. Web Sources


- Air Quality Issues of Electricity Production: Ozone (Smog) and Fine Particulates. Power Scorecard
  http://www.powerscorecard.org/issue_detail.cfm?issue_id=3

- Air quality guidelines. World Health Organization, Geneva
  ttp://www.who.int/peh/air/Airqualitygd.htm

- Aust, Ann. Effects of Metals Bound to Particulate Matter on Human Lung Epithelial Cells. *Health Effects Institute*
  http://www.healtheffects.org/Pubs/st110.htm

- Fly Ash Resource Center
  http://www.geocities.com/CapeCanaveral/Launchpad/2095/flyash.html

- Particulates, Fine Particulates: What They Are and How They Affect Us. Ministry of Water, Land and Air Protection, British Columbia.
  http://wlapwww.gov.bc.ca/air/particulates/fpwtaaht.html

- Reanalysis of the Harvard Six Cities Study and the American Cancer Society Study of Particulate Air Pollution and Mortality. A Special Report of the Institute's Particle Epidemiology Reanalysis Project. Final version, July 2000. *Health Effects Institute*
  http://www.healtheffects.org/pubs-special.htm

- Report of a WHO working group on health aspects of air pollution with particulate matter, ozone and nitrogen dioxide
  http://www.euro.who.int/eprise/main/WHO/Progs/AIQ/Home

- Revised Analyses of Time-Series Studies of Air Pollution and Health *Health Effects Institute*
  http://www.healtheffects.org/Pubs/st-timeseries.htm

# INTRODUCTION[1]

After observing considerable residue around residences near the Mirant power plant, we launched an investigation in the Spring of 2001 to scientifically determine both the cause and constituency of the deposits. At this point, the basis of our knowledge was anecdotal, necessitating empirical analysis to ascertain the source and content of the material. To this end, we approached and consulted Jonathan Levy of the Harvard School of Public Health, Gareth Mitchell of Pennsylvania State University Coal and Organic Petrology Laboratories, and the Virginia Department of Environmental Quality. In the interim, the Journal of the American Medical Association published (*March 6, 2002*) a study detailing adverse health effects from particulate matter. The article, which reviewed a consistent accumulation of foundational evidence linking mortality to fine particulate matter, raised our awareness of health effects associated with coal-fired power plant operations and emissions. It also heightened our local concerns and intention to identify the neighborhood residue.

As indicated in the scientific studies summarized below, the results demonstrate a large part of the residue originates at the Mirant plant. Further, the occurrence of fly ash and partially fused ash in the test samples legitimize our initial health concerns regarding particulates emitted by the power plant. Subsequent research, using a long-range wind dispersion model, concludes that proximity to the plant results in higher levels of particulate exposure, thereby creating greater health risks.

[1] Elizabeth Chimento and Poul Hertel wish to acknowledge a contribution of $150.00 for each, from their respective civic associations, to attend the International Coal Conference sponsored by the US Geological Survey, September 24-26, 2001. However, the writers pursued this project independently and are solely responsible for this report.

# LEVY ET AL. STUDY

Jonathan Levy[2] et al.'s Executive Summary of "The Influence of Population Heterogeneity on Air Pollution Risk Assessment: A Case Study of Power Plants Near Washington, DC" (Levy, Greco, Spengler, Harvard School of Public Health, Boston, May 2002, copy attached[3]) provides a seminal analysis of the D.C. metropolitan area's power plant emissions and their health effects[4]. Focusing on old coal-fired plants exempted from the Clean Air Act's regulation and located within a 50 mile radius of the D.C. metro area, Levy et al. target five facilities for study: Benning, Chalk Point, Dickerson, Possum Point and Potomac River, here in Alexandria, Virginia (Levy et al., Executive Summary 1). The study concludes that, by reducing emissions, more than 200 deaths per year, as well as numerous hospital admissions, emergency room visits and asthma attacks could be prevented in the entire five plant region (Exec. Sum. 6).

The Levy group initiated its analysis to determine health impacts of fine particulate matter ($PM_{2.5}$)[5] released by these facilities. To obtain this data, they quantified the direct emissions of $PM_{2.5}$, called primary particles, as well as sulfur dioxide ($SO_2$) and nitrogen oxides (NOx), known as secondary particles, which form over time in the air (Exec. Sum. 2). The majority of regulations controlling power plant emissions, however, focus on $SO_2$ and NOx but do not consider primary particles ($PM_{2.5}$).

Levy et al. then "addressed the question of health benefits which would have been obtained in 1999, had emission rates commensurate with Best Available Control Technology (BACT) been required at that time" (Exec. Sum. 1). To estimate health benefits, three end points were established: premature mortality, cardiovascular hospital admissions for the elderly and asthma emergency room visits for children, all of which had previously been linked with air pollution (Exec. Sum. 3). Levy incorporated the baseline estimate of premature mortality risk from the Health Effects Institute's re-analysis of the American Cancer Society cohort study, ACS II, which established the association between long-term $PM_{2.5}$ exposure and mortality rates (Exec. Sum. 3).

Employing the method of a long-range atmospheric dispersion model to "estimate the effects of emissions on ambient concentrations and using epidemiological findings to quantify the health effects associated with concentration changes," Levy and colleagues completed their study (Exec. Sum. 1).

The analysis demonstrated that reducing emissions could provide public health benefits by lowering premature deaths, hospital visits and asthma attacks (Exec. Sum. 6).

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[2] Jonathan Levy, Sc.D, Harvard School of Public Health's leading scientist in power plant/health issues, has conducted case studies on pollution and coal-fired power plants in Massachusetts, Illinois and, most recently, Washington, DC. In May 2002, he briefed the U.S. Senate Environment and Public Works Committee on "Health Impacts of Power Plants: Case Studies in Massachusetts, Illinois, and Washington D.C." (Sources, p. 6 , "Health Impacts of Air Pollution from Washington DC Area Power Plants," Summary prepared by Jonathan Levy specifically for Clean Air Task Force, May 2002).

[3] This study also published in Environmental Health Perspectives. The Importance of Population Susceptibility for Air Pollution Risk Assessment: A Case Study of Power Plants Near Washington, DC. Vol 110, No.12 (Dec. 2002): 1253-60.

[4] The analysis also examined the effects of $PM_{2.5}$ on different subpopulations in the region by taking into account individual risk factors.

[5] $PM_{2.5}$ refers to particulate matter of 2.5 microns or less.

The Levy et al. study of D.C. area power plants and accompanying health effects demonstrates that if Best Available Control Technology (BACT) were installed, premature deaths, hospital visits and asthma attacks caused by ambient 2.5 pollution would be considerably curtailed. Also, Levy's focus on 2.5 particulates (primary particles) and their effect on health reveals that these particles amass closer to the plant and decrease more quickly with distance, thereby jeopardizing the health of residents and workers in proximity to the plants. This is especially the case for Alexandria's Potomac River plant, which is located in a densely populated urban area. **Finally, the health benefits achievable, if Best Available Control Technology were installed, become dramatically clear in Levy's quantitative analysis. For the Potomac River plant alone, approximately 40 deaths, 43 hospital admissions, 560 emergency room visits and 3,000 asthma attacks per year would be prevented.**

# POPE ET AL./ JAMA ARTICLE

Connecting with Levy's focus on health issues associated with power plants in the D.C. area, the Journal of the American Medical Association published, in March 2002, the landmark analysis, "Lung Cancer, Cardiopulmonary Mortality, and Long-term Exposure to Fine Particulate Air Pollution" (Pope, Burnett, Thun, Calle, Krewski, Ito, Thurston. JAMA 287, 9, 2002, 1132-41, attached) which also examines health concerns relating to fine particulate (2.5 microns) air pollution. The scientists discovered that long-term exposure to particulate and sulphur-oxide air pollution, common to metropolitan areas, correlates with all-cause, lung cancer and cardiopulmonary mortality (1132).

This study, tracing the historic progression in research of fine particle pollution's effects, notes that in the **1970's it was established that mortality increased with highly concentrated particles and sulphur oxide pollutants** (1132). In the **1990's, research discovered that low concentrations of particulate air pollution affected health and mortality,** as well (1132). The gravity of these findings instituted a paradigm shift in environmental research, necessitating a re-evaluation of health guidelines and air quality standards. As a result, the EPA in 1997 imposed limits to fine particles measuring less than 2.5um (in diameter) in its air quality standards (1132).

Whereas **most studies to this point had focused on short-term exposure to small particulate pollution and its effects on health, newer research suggested that long-term exposure might be more detrimental to public health** (1132). Therefore, based on one of two major studies linking mortality to long-term pollution ($PM_{2.5}$) exposure, Pope et al. determined to assess the correlation between long-term exposure to fine particulates and all-cause, lung cancer and cardiopulmonary mortality.

Incorporating individual risk factors, such as smoking, education, marital status, body mass index (BMI), alcohol consumption, occupational exposures, diet and vital status data, the researchers connected these variables with national ambient air pollution data (1135). The statistical basis for the study was taken from the American Cancer Society's "Cancer Prevention Study II (CPS-II)," in which approximately 1.2 million adults were participating in an ongoing mortality study (1133). The Pope group enrolled only those participants, however, who lived in US metropolitan areas having available pollution data, thereby limiting their enrollment pool to 500,000. Participants completed questionnaires, including selecting applicable individual risk factors. Using the methodology of the standard "Cox proportional hazards survival model" and subjecting it to statistical analysis allowing for risk factor variables, as well as accumulating and charting death statistics, Pope et al. conducted their study (1134).

**The statistics showed that long-term exposure to particulate and sulphur-oxide air pollution, common to metropolitan areas, increases all-cause, lung cancer and cardiopulmonary mortality** (1132). Also, **"each 10-ug/m3 elevation in long-term particulate air pollution (average $PM_{2.5}$ ambient concentrations) was associated with approximately a 4%, 6%, and 8% increased risk of all-cause, cardiopulmonary and lung cancer mortality, respectively"** (1137). Finally, the link between fine particle air pollution and lung cancer and cardiopulmonary mortality remained constant, after allowing for individual risk factors and, after "controlling for regional and other spatial differences" (1141).

The American Medical Association's publication of the Pope et al. study in JAMA achieves a benchmark in fine particulate air pollution research, dissemination and public health protection by mainstreaming this study to the more than 340,750 medical community subscribers[6] it serves.

6 Jennna Ludwig, Circulation Dept., JAMA, Chicago.

# MITCHELL/ PENN STATE STUDY

## Pitt Street Test samples

Whereas the Levy et al. report and the Pope et al./JAMA article focus on 2.5 micron particles, which cause and/or aggravate serious health conditions, the Penn State analysis concentrates on determining the constituent elements of the North Old Town neighborhood dust samples[7]. The study concludes that non-combusted coal particles and partially combusted coal dust comprise "a significant amount" of the residential samples and that fly ash, although in lesser amounts, is also present ("A Petrographic Evaluation of Three Dust Samples and a Coal," p. 7, copy attached).

The analysis, conducted by Gareth D. Mitchell of the Coal and Organic Petrology Laboratories, Pennsylvania State University, using reflected light optical microscopy at 625X magnification (Mitchell 4), determines that the coal dust probably originates from a raw materials handling source, such as "railroad car tops, dumping operations, stockpiling and reclaiming, transportation, crushing operations" or all of the preceding possibilities (2). Further, the Penn State study reveals that coal spilled along the railroad track at the Mirant site matches the coal dust particles, thereby indicating that the coal dust on neighboring residential windowsills originates at the Mirant plant.

It is noteworthy that laboratory slides evidencing fly ash of the 1-2 micron size are identified in several of the plates (6). These smaller particulates are the most dangerous to health (see Levy study and Pope/JAMA article) and are easily inhaled through respiration. Although fly-ash particles are less dominant in the samples, they do represent an overall health concern and necessitate, at the very least, stringent testing and monitoring to ascertain counts of 2.5 micron particulates near the plant and adjacent neighborhood areas. The Levy study emphasizes that 2.5 and lesser micron particulates are sufficiently present in the air surrounding the Mirant Potomac River Generating Station to constitute a considerable hazard to public health (Levy Summary, page 2, this document).

In summary, the Penn State study demonstrates not only that coal dust accounts for a "significant amount" of the windowsill samples, but also that the Mirant power plant is the originating source.

[7] Samples of windowsill residue and coal sample from railroad track at Mirant power plant submitted to Penn State for analysis on 6/28/02 to establish residue constituents and point of origin.

## VIRGINIA DEQ TEST RESULTS

Like the Penn State analysis, the Virginia Department of Environmental Quality test results indicate that coal particles (non-combusted) and partially combusted coal dust account for a large part of the sample constituents[8]. The DEQ study, however, determines that "up to 50% of the residential samples is composed of "uncombusted coal dust" and another 10% consists of "coal combustion products, . . . including partially combusted coal and partially fused ash" (Memo, "Analysis of Dust Samples Taken at 1200 and 1202 N. Pitt Street, Alexandria, Virginia," attached).

The DEQ analysis was conducted by Carolyn Stevens, DEQ Office of Air Quality Assessment, Richmond, Virginia, using a polarizing light microscope at 150 times magnification to examine the samples (Memo).

Unlike the Penn State study results, the DEQ maintains that the 10% of "coal combustion products . . . including partially combusted coal and partially fused ash" found in the samples are not classified as fly ash, as the Penn State study indicates, but only as partially combusted constituent elements (Memo). This variance between the Penn State and DEQ studies is of interest as well as the discrepancy between the DEQ lab report's broad generalizations (DEQ Microscopic Analysis Form, attached) and the commentary memo's greater specificity, which inverts normative scientific procedure. For example, the lab report does not indicate the breakdown nor percentages of constituent elements in the samples nor does it indicate that "up to 50%" of the samples is comprised of coal dust, as the commentary memo states. Rather, the lab report simply generalizes that coal dust is one of "moderate to large amounts of asphalt, coal dust, mineral particulate, pollen, fungus, assorted fibers, rubber, coal combustion products" (DEQ Microscopic Analysis Form).

Nevertheless, these test findings have generated within the DEQ, a recommendation to address the Mirant plant's "coal handling procedures and particulate emission control features" (Memo) and to review with the plant Virginia DEQ dust control regulations, included in "#9 VAC 5-40-90 – Standard for Fugitive Dust/Emissions" (Commonwealth of Virginia State Air Pollution Control Board Regulations for the Control and Abatement of Air Pollution, p. 4-1:2, attached). At this time, the DEQ official response to their test findings on the Pitt Street samples remains forthcoming.

In conclusion, the Virginia DEQ's test results as well as the Penn State studies, indicate that a large amount of the neighborhood windowsill residue originates at the Mirant power plant.

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[8] Commonwealth of Virginia, Department of Environmental Quality, Memorandum from Kelly Lease, Air Compliance Inspector, Northern Virginia Regional Office to Charles D. Forbes, Air Compliance Manager, DEQ/NVRO, Re: "Analysis of Dust Samples Taken at 1200 and 1202 N. Pitt Street, Alexandria, Virginia," dated 6/10/03.

# HEALTH EFFECTS

Research in the United States suggests that fine particulates are responsible for tens of thousands of deaths caused by increases in lung and heart disease. Fine particulate air pollution triggers many kinds of respiratory illnesses, including asthma, bronchitis, pneumonia and emphysema. Senior citizens, infants and people who already have lung, asthmatic or heart problems are most at risk, but healthy younger adults and children can also be affected. The connection between asthma and fine particulates is noteworthy since asthma is the most common cause of medical emergencies in children[1].

> **"Each 10μg/m$^3$ elevation in long-term average $PM_{2.5}$ ambient concentration was associated with approximately a 4%, 6%, and 8% increased risk of all-cause, cardiopulmonary, and lung-cancer mortality, respectively"**
>
> *Pope et Al Journal of the American Medical Association March 2002*

Varieties of pollutants affect our air quality. During the 1990s research provided evidence that fine particles can damage human health even at concentrations previously thought to be unimportant. Particles with a diameter of 10 microns (millionths of a metre) or less, termed $PM_{10}$, are the most hazardous[2].

$PM_{10}$ are composed of a wide range of materials from a variety of sources:

- primary particles - arising from combustion sources
- secondary particles - mainly sulfate and nitrate formed by chemically combining Sulfur dioxide and nitrogen oxide with ammonia in the atmosphere.
- coarse particles - suspended soils and dusts, sea salt, biological particles and particles from construction work

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[1] Fine Particulates: What are they and how they affect us. February 2002. *Government of British Columbia; Ministry of Water, Land and Air Protection: Water, Air and Climate Change Branch*

See also the following two reports;
An Association between Air Pollution and Mortality in Six U.S. Cities
Douglas W. Dockery, C. Arden Pope, Xiping Xu, John D. Spengler, James H. Ware, Martha E. Fay, Benjamin G. Ferris, and Frank E. Speizer *New England Journal of Medicine* Volume 329:1753-1759 December 9, 1993 Number 24

Reanalysis of the Harvard Six Cities Study and the American Cancer Society Study of Particulate Air Pollution and Mortality. *Health Effects Institute.* A Special Report of the Institute's Particle Epidemiology Reanalysis Project. Final version, July 2000.

[2] An Association between Air Pollution and Mortality in Six U.S. Cities Douglas W. Dockery et al 1993
Reanalysis of the Harvard Six Cities Study and the American Cancer Society Study of Particulate Air Pollution and Mortality. *Health Effects Institute.* Final version, July 2000.

Further, the research indicated, as seen in the graph below, that **primary 2.5 particle concentrations "peaked closer to the plant and decreased more rapidly with distance than for sulfates or nitrates"** (Exec. Sum. 4). Therefore, proximity to the plant directly corresponded with higher levels of exposure to primary 2.5 particle pollution (Fig. 3 below, Levy et al. Environmental Health Perspectives. 1257). These results were consistent with Levy's earlier Massachusetts and Illinois power plant studies' findings, which used the same methodology (Exec. Sum. 6). Also, calculated on an annual average basis, concentration peaks for all pollutants occurred within 20 kilometers of the source (Exec. Sum. 4). For the Potomac River plant, that range covers all of Alexandria.

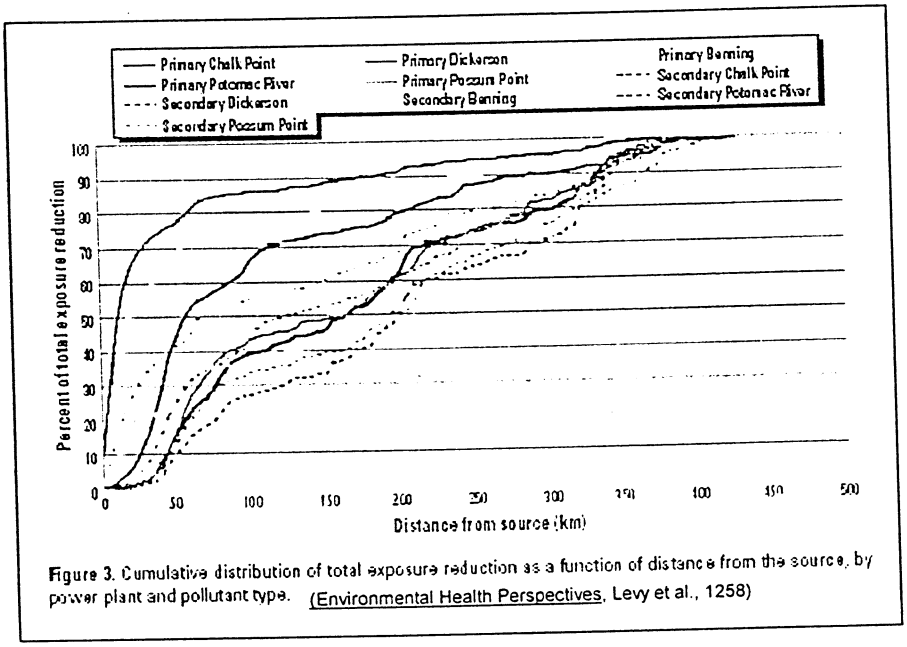


Figure 3. Cumulative distribution of total exposure reduction as a function of distance from the source, by power plant and pollutant type. (Environmental Health Perspectives, Levy et al., 1258)

The study's quantitative results evidence that more than 200 fewer deaths per year would occur in the entire five plant region if best available control technology (BACT) were in place (Levy, Environmental Health Perspectives. 1257). In Table 1 below (Levy, CATF, 3) Levy estimates the benefits accruing to each of the five plants individually, if BACT were implemented. For Alexandria's Potomac River plant, almost 40 lives per year would be saved. In addition, 43 hospital admissions, 560 emergency room visits and 3,000 asthma attacks would be prevented.

# TABLE 1

Attributed Plant Impacts and Benefits

| | Premature Deaths | Hospital Admissions | | Emergency Room Visits | Asthma Attacks | |
|---|---|---|---|---|---|---|
| | | Current Impacts | Hospital Admissions Prevented by Reduced Emissions | | Current Impacts | Asthma Attacks Prevented by Reduced Emissions |
| Benning | | 3 | 1 | | 180 | 99 |
| Chalk Point | | 110 | 82 | | 7,400 | 5,700 |
| Dickerson | | 53 | 43 | | 3,700 | 3,000 |
| Possum Point | | 57 | 44 | | 3,900 | 3,000 |
| Potomac River | | 66 | 43 | | 4,600 | 3,000 |

*Table 1, taken from The Clean Air Task Force's "Health Impacts of Air Pollution from Washington DC Area Power Plants," summarizes current health impacts and benefits for each of the five plants if the plants emitted less pollution, based on estimates calculated by Levy et al., excluding differential effects on disadvantaged populations.*

Not all $PM_{10}$ are created equal. It can be composed of very small particulates of about 0.1 to 0.2 microns in diameter. To simplify things, the literature often refers to a fine and coarse fraction of $PM_{10}$, since they generally differ in chemical composition source and behavior in the air:

- The fine fraction ($PM_{2.5}$)) contains particulates 2.5 microns or smaller. This fraction is most often generated by combustion processes and by chemical reactions taking place in the air.

From our lungs' point of view, bigger particulates are less harmful. Because of their weight, particulates larger than 10 micrometers settle to the ground quickly. If we do inhale them, they tend to collect in our throat and nose, the upper respiratory system, and are eliminated from our body by sneezing, coughing, nose blowing or through the digestive system. In other words, they do not travel very far into our lungs. They contain materials common to the crust of the earth and the ocean, reflecting the fact that natural sources such as windblown dust and sea salt spray are big contributors to the coarse fraction.

In contrast, particulates in the fine fraction ($PM_{2.5}$) can remain in the air for days to weeks. They can penetrate especially deep into our lungs, collecting in the tiny air sacs (called "alveoli") where oxygen enters the bloodstream. Consequently, they can cause breathing difficulties and sometimes, permanent lung damage.

**Studies**

"Epidemiological work conducted over several decades has shown that long-term residence in cities with elevated ambient levels of air pollution from combustion sources is associated with increased mortality"[3]. Subsequent studies found a strong relationship between not only sulfates and mortality but also fine particulate matter (all particles less than 2.5 microns in median aerodynamic diameter [$PM_{2.5}$]) and mortality rates.

Conclusive evidence of the adverse effects of air pollution and mortality has been around for years[4]. However, recent research has focused on a more narrow scope of parameters. These studies demonstrated conclusively that the pollution levels needed for harmful effects were much smaller than expected and that fine particulate matter contributes to excess mortality[5]. These findings included statistical techniques in which individual risk factors, like smoking habits, were factored into the study.

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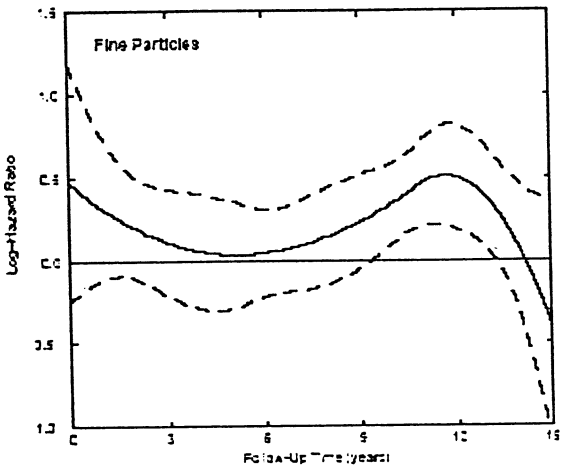
[3] Particle Epidemiology Reanalysis Project © 2000 Health Effects Institute, Cambridge MA

[4] See Firket J, The Cause of the symptoms found in the Meuse Balley during the fog of December, *1930 Bulletin Acad R Med Belgium.* 1931; 11:683-741
Ciocco A, Thomson DJ. A follow up of Donora ten years after: methology and findings. *American Journal of Public Health.* 1961; 51:155-164

[5] Abbey DE, Nishino N, Mcdonnell WF, Burchette RJ, Knutsen SF, Baeson LW, Yang JX. 1999 Long Term inhalable particles and other air pollutants related to mortality in nonsmokers. *American Journal Crit Care Med* 159(2):373-38

The seminal study by Dockery[6] and colleagues, the Harvard Six Cities study, found that fine particulate matter contributes to excess mortality (see figure below for time profile). In a similar study, Pope[7] and colleagues (American Cancer Society Study) reported that increased mortality in the form of cardiopulmonary disease and lung cancer was caused by fine particulate matter, sulfates, and does so at pollution levels commonly found in US cities. These studies hastened a new set of guidelines for $PM_{2.5}$ levels of acceptability by the Environmental Protection Agency.




"Both estimates suggest that the respective hazard ratio is a nonmonotone function of the follow up time. Specifically, the impact of fine particles on the mortality hazard decreases to near zero after five years of follow up, but later increases to reach a peak at about 10 to 12 years of follow up."
*Harvard Six Cities Study, page 152*

The business community challenged the validity of the studies and the legal wherewithal of the EPA to implement new guidelines, which prompted the EPA to seek validation of the original findings. Consequently, the EPA urged Harvard University and the American Cancer Society to allow other scientists to review their data. Consequently, Harvard University asked the Health Effects Institute (HEI) to review the studies in order to ascertain the validity of the conclusions. A full copy of the study[8] is available from the HEI web Site and it is voluminous and technical. Nevertheless, it does validate the original findings and mortality rates associated with the fine particulates.

In the March 2002 issue of the Journal of the American Medical Association Pope[9] and Colleagues assessed the effects of long term exposure to the fine particulate air pollution.

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[6] Dockery DW, Pope CA, XU X, SpenglerJD, Ware JH, Fay ME, Ferris BG, SpeizerFE, 1993. An association between air pollution and mortality in six US cities. *New England Journal of Medicine* 329: 1753-1759

[7] Pope CA, Thun MJ, Namboodiri MM, Dockery DW, SpenglerJD, Evans JS, SpeizerFE, Heath CW. 1995. Particulate air pollution as a predictor of mortality in a prospective study of US Adults. *American J Respir Crit Care Med* 151: 669-674

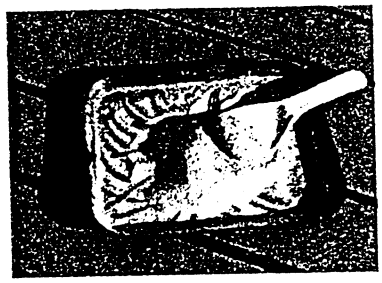
[8] Re-Analysis of The Harvard Six Cities Study and The American Cancer Society Study of Particulate Air Pollution and Mortality A special Report of the Institutes Particle Epidemiology Reanalysis Project; July 2000 *The Health Effects Institute.*

[9] Pope CA, Burnett RT, Thun MJ, Calle EE, Krewski D, Ito K, ThurstonGD. 2002. Lung, Cancer, Cardiopulmonary Mortality and Long Term Exposure to Fine Particulate Air Pollution. *Journal of the American Medical Association*: March 6 2002: 1132-1141

The authors found that there was an effect and that the effect persisted over time. "For every increase in each $10\mu g/m^3$ elevation in long-term average $PM_{2.5}$ ambient concentration was associated with approximately a 4%, 6%, and 8% increased risk in all-cause, cardiopulmonary, and lung-cancer mortality, respectively."[10]

The function is linear, meaning that the greater the exposure the greater the risk. Furthermore, correcting mortality rates for other adverse effects, such as tobacco smoking, drinking, obesity and location effects, does not alter the evidence of fine particulate matter on mortality.

**Fly Ash**[11]



The connection between Fly ash and health issues is important and yet very difficult to clarify. Because approximately 20% - 40% of fly ash particles are below 7 microns in diameter, they are in the respirable[12] range and absorbed by the deeper lung tissue. The study below demonstrates not only the adverse effects of fly ash on health, but also the connection between the size of the particulate matter and the effect on health. The smaller the particulates, the greater the effect.

During the past decade, research has consistently demonstrated the connection between inhaled particulate matter with both acute and chronic health effects. Although much research has been directed toward identifying plausible mechanisms linking particulate matter and pathophysiologic effects, many critical aspects are not understood. Dr. Ann E Aust [13] focused on the effects of fly ash, the particulate residue from coal-fired power plants. Coal contains metals that vaporize during combustion and then solubilized from fly ash within lung cells may cause toxic reactions.

The study confirmed that soluble extracts of coal fly ash generated reactive oxygen species in vitro and that transition metals were likely responsible. "Further, the smallest particles, which were rich in iron, were the most active."[14] This means that more iron was released from the smaller particles than from larger ones. The investigators then examined the effects of coal fly ash on human lung epithelial (tissue-layered) cells in culture. First, they demonstrated that coal fly ash particles entered the cells and

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[10] See Pope et al 2002

[11] The inorganic residue, that remains after pulverized coal is burned, is known as 'coal combustion byproducts' (CCB). Fly Ash is the finely-divided CCB collected by electrostatic precipitators from the flue gases. Boiler slag and bottom ash are the heavier and coarser coal combustion byproducts. The picture is from the Fly Ash resource Center Web site.

[12] Fly Ash Center, Fly Ash Safety Sheet

[13] Dr Ann E Aust of Utah State University, Logan UT. The complete report, Particle Characteristics Responsible for Effects on Human Lung Epithelial Cells, can be requested from Health Effects Institute. AUST 110

[14] Aust et al Statement Synopsis of Research Report 110 *Health Effects Institute*

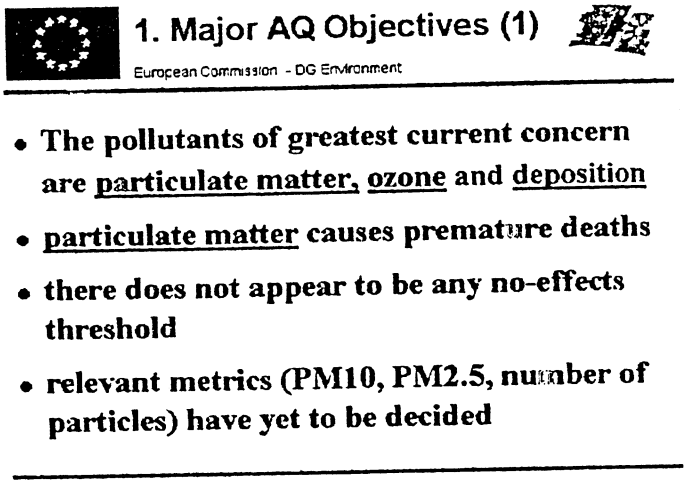
stimulated synthesis of the protein ferritin. Ferritin binds iron and is produced in response to increasing iron levels; thus, its presence indicates that iron was released intracellularly and that iron was available to provoke an inflammatory response by forming reactive oxygen species.

**Latest Developments**

Over the past decade, time-series studies conducted in many cities have contributed information about the association between daily changes in concentrations of airborne particulate matter (PM) and daily morbidity and mortality. In 2002, however, investigators at Johns Hopkins University and at Health Canada identified issues in the statistical model used in the majority of time-series studies. The authors suggested that there was a problem with the statistical software package used to analyze the data, because the convergence criteria might have been too loose. Consequently, the Environmental Protection Agency asked the Health Effects Institute to review all the studies using appropriate corrective measures. The Special Report details[15] the attempts to address several questions raised by these discoveries.

The impact of using more appropriate statistical convergence criteria on the estimates of PM effect in the revised analyses varied greatly across the studies. In some studies, stricter convergence criteria had little impact, and in a few the impact was substantial. "In no study were conclusions based on the original analyses changed in a meaningful way by the use of stricter criteria."[16]

In the European Community, the debate accepts that particulate matter is harmful, and is instead focused on how to regulate particles in the size of 2.5 to 10 microns in urban areas (see the European Commission Objectives to the right[17]). Furthermore, they recognize that the scientific studies have not been able to find a lower limit of exposure under which they can observe no health effects.




[15] Special Report, Revised Analyses of Time-Series Studies of Air Pollution and Health; May 2003 *Health Effects Institute.*

[16] Synopsis of a Special Report Revised Analyses of Time-Series Studies of Air Pollution *Health Effects Institute*

[17] Workshop in support of the Clean Air for Europe (CAFE) programme of DG ENV in Berlin, Germany, November 4-6, 2002; Why the Coarse fraction of PM10 is important for air quality management.
Jacobi, Stefan *European Commission*, DG Environment, Brüssel, Belgien

## PLANT OPERATION CONCERNS

The Mirant plant has the capacity to produce over 480 Megawatts of power a year. Because of its proximity to Reagan National Airport, the smokestacks (chimneys) are very short, unlike those of most coal-powered plants.

The inorganic residue, that remains after pulverized coal is burned, is known as 'coal combustion byproducts' (CCB). Fly Ash is the finely divided CCB collected by electrostatic precipitators after the combustion process. Subsequently, hammers hit the electrostatic precipitators to release the particulates. As the particulates fall, they are sifted into the ash house silo, which are essentially huge vacuum cleaners that use fabric filters (bags) to trap the particles. **According to the Mirant plant's consultant study, 29 tons per year of particulates are not captured by the bags and escape directly into the atmosphere. With the addition of a second ash house silo, this number could be reduced by 50%[18].**

The operational sources of emission and residue from the plant can be summarized as follows:

- Stacks/Chimneys
  Primary and secondary particulates from burning up to 4800 tons coal/day and 3800 tons/day on average.

- Ash House Silos
  The operation is not 100% effective since the Ash House silo bags capture only particulates above a certain size. The fly ash captured in the ash house silo requires 30-35 truck trips per day for removal from the property.

- Coal Pile
  Coal crushing and piling operations susceptible to wind currents.

[18] This was disclosed at a meeting, August 15, 2001 at the Mirant Plant, convened to discuss the consultant's study results. City staff was also present.

# CONCLUSION

As stated in the Introduction, we initiated this study in Spring 2001 to scientifically determine the source of our neighborhood residue. Pursuing that answer, both the Mitchell/Penn State and the Virginia Department of Environmental Quality analyses have confirmed that a large part of the residue originates at the Mirant Potomac River power plant. Further, the Levy et al., as well as studies in the Health Effects section, which are encapsulated by the Pope et al./JAMA article, have established the hazardous health effects associated with $PM_{2.5}$ emissions from power plants. In particular, the Levy study provides quantitative information on the health impacts as well as benefits for Alexandria's Potomac River plant, if best available control technology (BACT) were installed.

In conclusion, this scientific data validates and intensifies our original concerns regarding the residue emanating from the power plant. The empirical research, collected over a two and half year period, consistently demonstrates the health dangers and risks associated with coal burning power plants. Furthermore, these health effects impact not only North Old Town, but also the entire city. Therefore, remediation is needed to protect the health of all Alexandria citizens.

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Revised Analyses of Time-Series Studies of Air Pollution and Health *The Health Effects Institute*, Boston, MA.

## Fine Particulates[19]

### *What are fine particulates?*

Particulates are airborne tiny solid or liquid droplets of many shapes and sizes that come from a variety of sources. Some of these coarse particles - such as soot or smoke -- are large or dark enough to be seen by the naked eye. They are referred to as PM-10 since they are "particulate matter" 10 microns or smaller in size. These larger particulates are emitted from roads, materials handling, crushing and grinding operations and include wind borne dust.

Other particulates are so small they can only be seen with special microscopes. These "fine" particles measure less than 2.5 microns in diameter -- PM-2.5 - and are about the size of bacteria. These minuscule particulates are of particular concern since they can become lodged deep into the lungs and typically contain greater amounts of toxic substances than larger particulates.

A number of harmful substances have been found in $PM_{2.5}$:

- Sulphates produced from sulphur dioxide emissions are acidic in nature, and may react directly with our lungs.

- Elemental carbon produced during wood and engine combustion can pick up cancer-causing chemicals like benzo(a)pyrene and give them a free ride into our lungs.

- Hundreds of organic carbon compounds, besides benzo(a)pyrene, have been identified in exhaust from vehicles, combustion processes and even meat-cooking operations.

- Several studies have shown that toxic trace metals such as lead, cadmium and nickel are more concentrated in $PM_{2.5}$ than in bigger particulates.

Combusting fossil fuels such as coal, oil, diesel fuel or gasoline is the primary source of fine particulate pollution. In particular old coal-fired power plants, industrial boilers, diesel and gas-powered vehicles, as well as wood stoves, are the principal sources of fine particulates.

From our lungs' point of view, bigger particulates are less harmful. Because of their weight, particulates larger than 10 micrometers settle to the ground quickly. If we do inhale them, they tend to collect in our throat and nose, and are eliminated from our body by sneezing, coughing, nose blowing or through the digestive system.

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19 Fine Particulates: What They Are and How They Affect Us Ministry of Water, Land and Air Protection, Government of British Columbia. http://wlapwww.gov.bc.ca/air/particulates/fpwtaaht.html

Particulates in the coarse fraction of $PM_{10}$ are removed in the upper respiratory system. In other words, they don't travel very far into our lungs. They contain materials common to the earth's crust and the ocean, reflecting the fact that natural sources such as windblown dust and sea salt spray are big contributors to the coarse fraction.

Vegetation is another large natural source. Human activities that involve grinding or pulverizing, such as mining, quarrying and cement manufacturing, are also important. These particulates don't stay in the air too long, settling to the ground within a matter of a few hours to a few days.

In contrast, particulates in the fine fraction ($PM_{2.5}$) can remain in the air for days to weeks. They can penetrate especially deep into our lungs, collecting in the tiny air sacs (called "alveoli") where oxygen enters the bloodstream. As a result, they can cause breathing difficulties and sometimes permanent lung damage.

# MERCURY[20]

Although not part of the study, mercury emissions are becoming a greater concern associated with coal plants. Hence, this section is included for general informative purposes.

Mercury is present in trace amounts in coal and is released as a gas when coal is combusted. Growing concern over potential environmental effects of mercury is reflected by the move towards establishing emissions limits for sources such as coal combustion. For example, according to the International Energy Agency (IEA), mercury controls will be a legal requirement for many coal-fired plants in the USA by 2007.

Recent data from the Information Collection Request carried out in the USA have resulted in an increase in the understanding of mercury behavior in coal-fired systems. The retention of mercury within a coal-fired power plant depends largely upon its oxidation state. Soluble oxidized mercury is controlled with existing pollution control technologies such as bag houses, electrostatic precipitators (ESP) and flue gas desulferization (FGD) systems. Insoluble elemental mercury passes through the plant largely uncaptured. Chlorine and other flue gases can play a major role in the mercury oxidation state. There appears to be a strong relationship between coal type and mercury oxidation. In general, US bituminous coals produce more mercury in the oxidized state than sub-bituminous coals and lignite.

**Average mercury removal across various pollution control devices for different coal ranks**

| Control device | Temperature, °C | Bituminous coal | | Subbituminous coal | | Lignite | | All coals | |
|---|---|---|---|---|---|---|---|---|---|
| | | Hg removal | data* | Hg removal | data* | Hg removal | data* | | |
| Cold side ESP | 130-170 | 56% | 9 | 12% | 4 | 47% | 1 | 42% | 14 |
| Hot side ESP | 250-400 | 27% | 3 | 9% | 2 | | | 20% | 3 |
| Baghouse | 130-170 | 85% | 7 | 75% | 2 | 58% | 1 | 82% | 10 |
| Wet scrubbers | 130-170 | | | | | | | 26% | 2 |
| Wet FGD + cold ESP | 130-170 | 51% | 7 | 27% | 3 | 49% | 4 | 45% | 14 |
| Wet FGD + hot ESP | 130-170 | | | 35% | 3 | | | 35% | 3 |
| Wet FGD + baghouse | 130-170 | | | | | 73% | 2 | 73% | 2 |
| Wet FGD + wet scrubber | 130-170 | 12% | 1 | 18% | 2 | | | 16% | 3 |
| Spray dry FGD + ESP | 130-170 | | | 53% | 2 | | | 53% | 2 |
| Spray dry FGD + baghouse | 130-170 | 83% | 5 | 22% | 2 | 25% | 3 | 53% | 10 |

* number of plants studied — IEA Coal Research

"Existing pollution control systems can remove up to 90% of the incoming coal's mercury content in some cases but very little in others". Furthermore, the coal type is more important than the type of particulate control system or the type of FGD system with respect to mercury control.

[20] Mercury – emissions and control *International Energy Agency*
IEA Coal Research is a collaborative project of member countries of the International Energy Agency (IEA) to provide information about and analysis of coal technology, supply and use. The service is governed by representatives of ten countries (Austria, Canada, Denmark, Italy, Japan, the Netherlands, Poland, Sweden, the United Kingdom and the USA) and the European Commission.

# Executive Summary

## The Influence of Population Heterogeneity on Air Pollution Risk Assessment: A Case Study of Power Plants Near Washington, DC

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## BACKGROUND

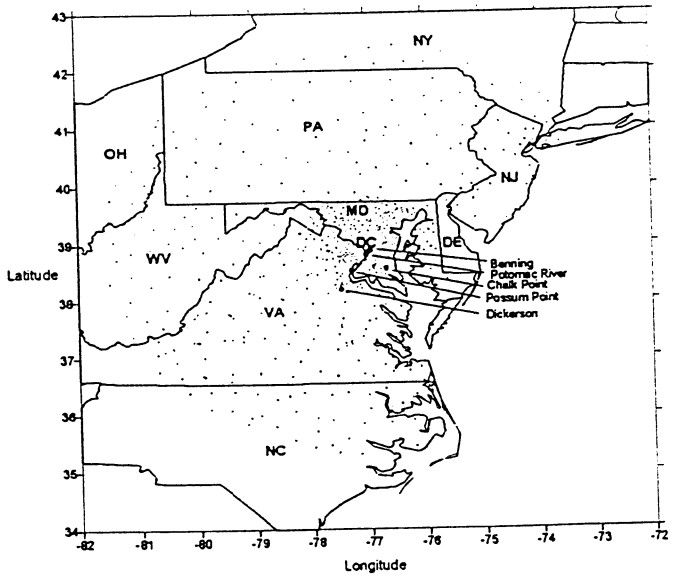
In previous investigations, we evaluated the health benefits of potential emission reductions from fossil-fueled power plants in Massachusetts and Illinois that had been "grandfathered" under the Clean Air Act. The basic analytical framework involved using a long-range atmospheric dispersion model to estimate the effects of emissions on ambient concentrations and using epidemiological findings to quantify the health effects associated with concentration changes. In this study, we have applied our analytical framework to five power plants near Washington, DC, with a focus on the potential importance of differences in population characteristics. Past analyses assumed that all individuals within age groups were equally likely to experience health effects, although that is unlikely to be the case given differences in susceptibility related to health status, income, race, or other factors.

We selected all grandfathered power plants located within a 50 mile (80 km) radius of Washington, DC, resulting in the choice of five facilities - Benning, Chalk Point, Dickerson, Possum Point, and Potomac River. For our case study, we addressed the question of the health benefits that would have been obtained in 1999 had emission rates commensurate with Best Available Control Technology been required at that time. Because of ongoing modifications at Possum Point and general electricity consumption

trends, the estimates we provide are not identical to the future benefits that would be obtained through regulation.

For our model, we focus exclusively on the health benefits associated with reductions in fine particulate matter ($PM_{2.5}$) concentrations. Thus, we quantify direct emissions of $PM_{2.5}$ (known as primary particles) as well as sulfur dioxide ($SO_2$) and nitrogen oxides (NOx), which form fine particles over time in the atmosphere. Most proposed and pending regulations for older power plants consider $SO_2$ and NOx but do not directly address primary particles. Any benefits associated with reductions of ozone, mercury, greenhouse gases, acid precipitation, or ecological or visibility endpoints were not included in our calculations.

Assuming that these plants moved from actual 1999 emissions to lower target levels (0.3 lb/MMBTU of $SO_2$, 0.15 lb/MMBTU of NOx, and 0.01 lb/MMBTU of $PM_{10}$), we estimated the corresponding concentration reductions at points within 400 km of Washington, DC. Our past work has shown that this captures a significant portion of the total effects without overextending the capabilities of our dispersion model. Our model region included 47 million people and is shown below:



As previously, we used the CALPUFF atmospheric dispersion model to estimate the reduced pollution exposure within the defined region. This model considers detailed meteorological patterns and chemical transformations of the pollutants, along with characteristics of the power plants. Our past work has shown that CALPUFF provides exposure estimates that are similar to values from earlier investigations and are relatively insensitive to changes in model assumptions.

For our health benefit estimates, we considered three endpoints – premature mortality, cardiovascular hospital admissions for the elderly, and asthma emergency room visits for children – which have been linked with air pollution and for which population heterogeneity might be important. This is not meant to provide a comprehensive list of health benefits, but rather to yield some illustrative estimates.

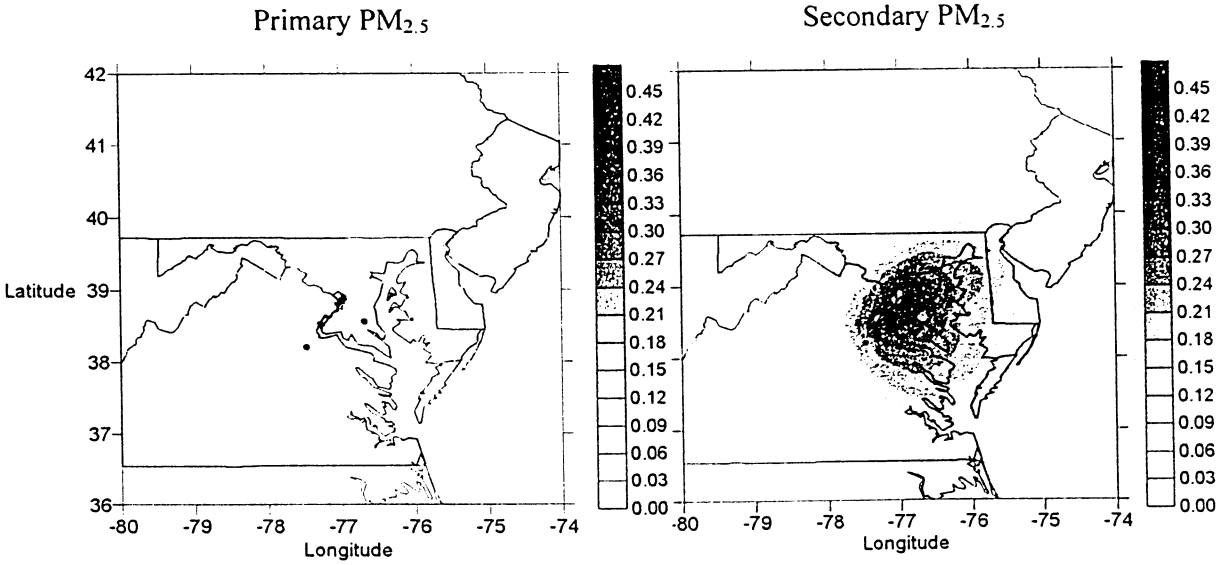
For each health endpoint, we surveyed the epidemiological literature to estimate the relationship between concentration changes and health effects and to evaluate whether this relationship depends on health status or demographic factors. We also looked at differences in background rates of disease and health care utilization, which would influence the total number of health outcomes in different groups.

We took our baseline estimate of premature mortality risk from the Health Effects Institute reanalysis of the American Cancer Society prospective cohort study, which found a significant association between long-term $PM_{2.5}$ exposure and mortality rates. This study also found a stronger association for people with lower educational attainment. This fact was coupled with evidence that people of lower educational attainment are at greater risk for premature mortality from all causes. For cardiovascular hospital admissions, the relationship was derived from a number of published studies, and

we investigated the influence of diabetic status on both the percentage increase in admissions per unit concentration and the baseline risk of an admission. Similarly, our concentration-response function for asthma emergency room visits was taken from a combination of studies, and we explored racial differences in asthma emergency room visit patterns.

## MAJOR FINDINGS

When we considered all five power plants together, the emission reductions led to annual average $PM_{2.5}$ (primary plus secondary) concentration reductions ranging from 0.009-0.9 $\mu g/m^3$ in our receptor region (compared with ambient levels of approximately 14-18 $\mu g/m^3$). As indicated in the figure below, concentrations peaked closer to the plant and decreased more rapidly with distance for primary particles than for sulfates or nitrates. On an annual average basis, concentration peaks were within 20 km of the source for all pollutants and power plants.



Given the estimated air pollution benefits, our central estimate is that emission controls would result in 230 fewer deaths per year in the modeled region (decreasing from 310 deaths per year given actual emissions to 80 given lower target emissions). Assuming equal particle toxicity, 63% of these benefits are due to secondary sulfates, with 19% due to secondary nitrates. The distribution of the benefits by power plant, pollutant, and affected population is given in the following table (estimates presented to two significant figures):

| | Baseline model (no stratification) | Full susceptibility model (education-stratified relative risk and baseline mortality) |
|---|---|---|
| Total mortality benefits | 230 fewer deaths/yr | 240 fewer deaths/yr |
| Primary PM | 42 | 43 |
| Secondary PM | 190 | 200 |
| Less than high school education | 58 | 130 |
| Within 50 km | 34 | 30 |
| Plant-specific benefits | | |
| Benning | 2 | 2 |
| Chalk Point | 91 | 98 |
| Dickerson | 47 | 50 |
| Possum Point | 44 | 46 |
| Potomac River | 44 | 45 |

This table indicates that accounting for educational attainment would not have a large influence on the total benefits, but would inform our understanding of who is most affected by air pollution and would therefore benefit most from controls. Under the susceptibility model, more than half of the benefits accrue among the 25% of individuals with less than a high school education. Although the broad geographic patterns are

affected minimally, benefits at the census tract level change by as much as a factor of three when we incorporate educational attainment into the model.

We also estimate annual benefits of approximately 60 fewer cardiovascular hospital admissions in the elderly (decreasing from 80 to 20) and 140 fewer asthma emergency room visits in children using our baseline model (decreasing from 180 to 40). As above, accounting for population characteristics does not change these values significantly but has a dramatic influence on the affected individuals. In our susceptibility model, diabetic individuals represent 13% of the elderly population but receive 54% of the cardiovascular hospital admission benefits. African-American children comprise 21% of the at-risk population for asthma-related emergency room visits but receive 64% of the benefits of emission controls when differences in health care utilization patterns are acknowledged.

## DISCUSSION AND IMPLICATIONS

Our study illustrates that emission reductions at a small set of power plants can provide tangible public health benefits, by providing small but quantifiable concentration reductions over a large geographic area. According to our best estimates, if emissions from five power plants in the Washington, DC area were reduced to levels achievable through current technology, over 200 fewer premature deaths per year would occur, along with 60 fewer cardiovascular hospital admissions in the elderly, 140 fewer asthma emergency room visits for children, and numerous other morbidity outcomes not quantified within our study. The magnitude of the benefits is in agreement with our past modeling efforts and estimates provided by other investigators.

In this study, we focused on the question of whether our benefit estimates would differ if we accounted for differences in susceptibility. We concluded that the magnitude and broad geographic distribution of benefits would not change significantly but that small-scale patterns and demographic differences would be substantially affected. For each health outcome, our model identified subpopulations that comprised a relatively small percentage of the population but provided more than half of the health benefits – individuals with less than high school education for mortality, diabetics for cardiovascular hospital admissions, and African-Americans for asthma emergency room visits. In all cases, individuals with lower socioeconomic status were disproportionately affected by air pollution and received a disproportionate share of the benefits of controls.

There are clearly multiple uncertainties in these conclusions, related to underlying uncertainties in our analytical model. We quantified many of these uncertainties in previous reports, but do not include them in the current study for brevity's sake. Previously, we illustrated that key CALPUFF assumptions (such as the incorporation of wet and dry deposition, chemical conversion mechanism, and size of the receptor region) had relatively small influences on the overall conclusions of the analysis. Broad questions about the applicability of CALPUFF can be raised, but the general concordance between our findings and those from other models supports a lack of substantial modeling bias. For the epidemiological evidence, there are numerous uncertainties related to the magnitude of the relationship between concentrations and health effects, including issues of relative toxicity and the believability of the cohort mortality evidence. The estimates reported in this study represent plausible central estimates, but alternative assumptions could lead to substantially higher or lower benefit estimates. Finally, our susceptibility

models are based on the assumption that national trends hold for our selected geographic area and represent a static causal relationship. While this assumption undoubtedly contributes some uncertainty, the general correspondence between low socioeconomic status and health is indisputable, and the susceptibility calculations are meant to be illustrative.

In summary, we have applied models validated in past investigations to develop a tool that can be used by policymakers to evaluate the benefits of control options. The findings from this investigation are meant as an input to the decision process, providing both quantitative measures of health benefits and qualitative descriptions of the characteristics of the individuals likely affected. Clearly, to draw conclusions about policy decisions, one would need to combine our health benefit estimates with control costs and other information. At a minimum, a comprehensive evaluation would need to consider economic implications of controls on producers and consumers, changes in production efficiency and plant utilization, and impacts of additional air and water pollutants (including upstream emissions). However, our study provides quantitative estimates of a subset of health benefits of emission controls, which can be combined with other pieces of evidence to inform air pollution control strategies.

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May 2002

# Health Impacts of Air Pollution from Washington DC Area Power Plants

## SUMMARY

For more than three decades, the nation's oldest and dirtiest power plants have avoided meeting tighter air pollution standards that new plants must meet. Power plants release a number of air pollutants, including soot-like particles known as fine particulate matter. Researchers at the Harvard School of Public Health examined the health impacts of fine particulate matter released by power plants near Washington DC. The study by Jonathan Levy, Susan Greco, and John Spengler examined five power plants: Benning, Chalk Point, Dickerson, Possum Point, and Potomac River. The key findings include:

- Over 250 deaths per year are linked to fine particulate matter from the five plants. Approximately 20 of these deaths are estimated to occur in Washington DC, 40 in Virginia, 60 in Maryland with the remainder occurring in nearby states. The impacts vary based upon the plant's size and proximity to population. Chalk Point was estimated to have the largest impact (about 100 deaths per year).

- If the five plants used readily available pollution control equipment, approximately 75% of the current deaths, asthma attacks, emergency room visits, and hospitalizations could be avoided.

- Disadvantaged groups are especially vulnerable to air pollution. Disadvantaged groups are more impacted from the five plants' emissions and receive more benefits from reducing their pollution than the population as a whole.
  - Although only 25% of the population studied has less than a high school education, this group suffers about half of the mortality impacts attributed to the plants.
  - While 21% of the population of children studied are African-American, they account for more than half of the incidents of pediatric asthma related emergency room visits attributed to the plants' emissions.
  - Diabetics comprised only 13% of the elderly population studied, yet they account for more than half of the incidents in elderly cardiovascular hospital admission reductions attributed to the plants.

Policy Implications

Requiring power plants to reduce air pollution would yield tremendous improvements in air quality and public health. Local impacts of power plants are significant, and vulnerable groups bear a disporpotiante risk. All power plants must meet modern emission standards.

## INTRODUCTION

Aging power plants are among the nation's largest sources of air pollution. In 1999, they contributed almost 68% of the sulfur dioxide and 23% of the nitrogen oxide emitted in the United States according to USEPA data.

When Congress amended the Clean Air Act in 1970 and 1977, older power plants-- many built in the 1950's, 1960's, and 1970's, -- were exempted from the most stringent emission standards. It was assumed that these plants would close soon and be replaced with cleaner,

newer plants. But few of these plants closed, and today these older plants produce the bulk of the nation's electricity.

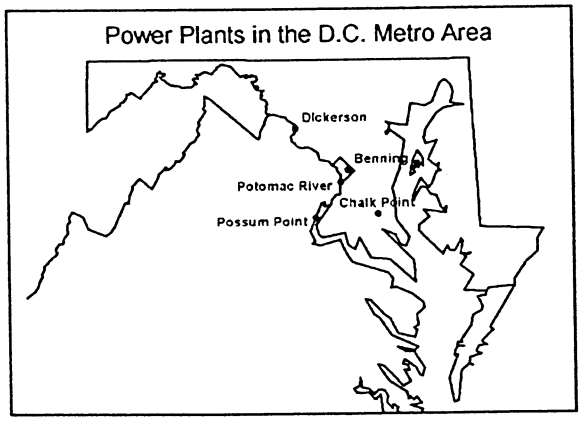
In 1996, researchers at the Harvard School of Public Health began a series of studies aimed at estimating the health impacts of air pollution from specific power plants. The principal authors of these studies, Jonathan Levy and Jack Spengler, examined impacts such as deaths, hospitalizations, asthma attacks, and other serious health outcomes. The Levy and Spengler studies generally focused on only one of many air pollutants from power plants-- fine particulate matter. Fine particulate matter (PM2.5) is composed of small soot-like particles that are a fraction of the width of a human hair. Fine particles can be directly emitted by power plants, but most form downwind as sulfur dioxide and nitrogen oxide gases react with ammonia to form sulfate and nitrate particles.

Levy and Spengler took the results from previous studies of air pollution health effects and combined them with the results of computer models that estimate pollution concentrations due to the emissions from each power plant. From this information, they were able to predict health impacts of each power plant they studied. Their earlier work was supported by Toyko Gas and Owens Corning, and their most recent studies were supported by the Clean Air Task Force with a grant from the Pew Charitable Trusts.

# RESULTS

The Harvard researchers examined five fossil-fuel power plants within 50 miles of Washington DC. These plants-- Benning, Chalk Point, Dickerson, Possum Point, and Potomac River-- burn coal as their primary fuel, with some burning oil as well. Possum Point is undergoing modifications to convert some units to burn natural gas, which will lower its emissions. The plants range in size from 480 MW to over 2200 MW. Their location is shown in Figure 1.

## FIGURE 1

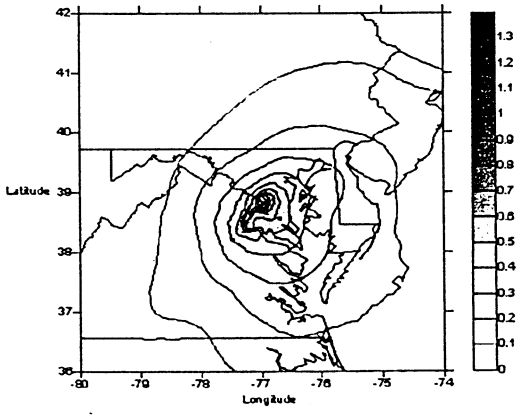



Impacts on the General Population

The five plants together contribute a maximum of 1.4 micrograms per cubic meter of fine particulate matter on an annual basis to the region around the plants. As shown in Figure 2, the concentrations from all plans combined peaked fairly close to the cluster of the plants.

# FIGURE 2

**Five Power Plants, total PM.5 (primary plus secondary), current impacts (ug/m3, annual average):**




Levy, Greco, and Spengler attribute over 250 deaths *per year* to the five plants based on their emissions in 1999. Approximately 20 of these deaths occur in Washington DC, 40 in Virginia, and 60 in Maryland. The Harvard researchers also calculate that each year, these five plants trigger approximately:

- 20,000 asthma attacks,
- 4,000 emergency room visits, and
- 300 hospitalizations.

If the plants were to cut their sulfur dioxide and nitrogen oxide emissions to levels that can be achieved using readily available pollution controls, approximately 75% of the deaths and disease attributed to the five plants could be avoided. Table 1 summarizes the health impacts for each plant for both current conditions and the benefits if the plants emitted less pollution, using the estimates from Levy et al. that do not consider differential effects on disadvantaged populations. These impacts are in a study area covering about 47 million people within a 250 mile radius of the plants.

# TABLE 1

**Attributed Plant Impacts and Benefits**

| | Premature Deaths | | Hospital Admissions | | Emergency Room Visitis | | Asthma Attacks | |
|---|---|---|---|---|---|---|---|---|
| | [illegible] | | Current Impacts | Hospital Admissions Prevented by Reduced Emissions | [illegible] | | Current Impacts | Asthma Attacks Prevented by Reduced Emissions |
| Benning | [illegible] | | 3 | 1 | [illegible] | | 180 | 99 |
| Chalk Point | [illegible] | | 110 | 82 | [illegible] | | 7,400 | 5,700 |
| Dickerson | [illegible] | | 53 | 43 | [illegible] | | 3,700 | 3,000 |
| Possum Point | [illegible] | | 57 | 44 | [illegible] | | 3,900 | 3,000 |
| Potomac River | [illegible] | | 66 | 43 | [illegible] | | 4,600 | 3,000 |

Impacts on Vulnerable Groups

Previous studies have shown that some groups are more vulnerable to air pollution than others. For example, people with lower education may be at higher risk from dying because of air pollution exposure. To better understand how the burden of these impacts are borne by groups who are especially vulnerable to air pollution, the study changed the baseline model's homogeneous population assumption to account for differences in population characteristics. Specifically, the study looked more closely at a several specific health impacts and how they affect three key groups:

- *Mortality and how deaths are distributed by educational level.* Educational attainment is a surrogate measure for a number of factors that influence mortality such as economic status, early childhood health, and supportive networks of family and friends.
- *Pediatric asthma emergency room visits and how these visits are distributed among African-American children.* African-Americans are at higher risk for asthma and for having episodes that require use of the emergency room than the general population as a whole.
- *Cardiovascular hospital admissions among the elderly and how these admissions are distributed among the elderly who have diabetes.* Diabetics are at higher risk for heart disease and hospital admissions when compared to the general population.

Accounting for susceptible groups did not significantly change the total quantity of damage, but did impact the distribution of who was harmed and who benefited if emissions from the plants are reduced.

- Although only 25% of the population studied have less than a high school education, this group suffers about half of the mortality impacts and receives about half the benefits in lives saved when emissions are reduced using readily available pollution controls.
- While 21% of the population of children studied are African-American, they account for about 64% of the pediatric asthma related emergency room visits when emissions are reduced using readily available pollution controls, and about the same percentage of the visits attributed to current power plant emissions.
- While diabetics comprised only 13% of the elderly population studied, they account for about 54% of the cardiovacular hospital admission benefits when emissions are reduced using readily available pollution controls, and about the same percentage of the admissions attributed to current power plant emissions.

Table 2 summarizes the estimates from the Levy et al. study when they used information on vulnerable groups.

# TABLE 2

**Distribution of Attributed Impacts and Benefits on Vulnerable Groups**

| | Current Impacts | Benefits from Reduced Emissions | Vulnerable Groups |
|---|---|---|---|
| Mortality | 310 | 230 | Although only 25% of the population studied have less than a high school education, this group gets half of the mortality impacts and benefits. |
| Pediatric Asthma Emergency Room Visits | 210 | 150 | While 21% of the population of children are African-American, they receive 64% of the impacts and benefits in pediatric asthma related emergency room visits. |
| Cardiovascular Hospital Admissions among the elderly | 80 | 60 | While diabetics comprised only 13% of the elderly population, they accounted for about 54% of the impacts and benefits in cardiovascular hospital admissions. |

# STUDY DESIGN

Over the last decade, a growing body of scientific studies has linked current levels of soot in our air to death and disease. These studies tracked thousands of people in cities across the nation who were exposed to different levels of soot in their air. By factoring out differences such as age, smoking, and occupation, these studies establish that people who live in areas with polluted air suffer greater health damage than people who live in areas with cleaner air.

Levy, Greco, and Spengler used these studies to determine how the risk of health damage varies as fine particle concentration changes in the air. Then using state-of-the art computer models, the Harvard researchers estimated the concentration of particulate pollution at ground-level from specific power plants on an annual basis. They calculated the number of deaths, hospitalizations, asthma attacks and other serious health impacts attributable to these plants by combining:

- Fine particulate matter concentration at ground level derived from computer modeling.
- Health risk associated with fine particulate matter concentration
- Census data showing the number people exposed in the region around the plants.

Levy, Greco, and Spengler examined the health impacts attributable to these plants under two emissions scenarios: 1) current emissions (1999), and 2) a hypothetical case assuming the plants were to reduce emissions by applying Best Available Control Technology. This second scenario assumed the plants reduced emissions to sulfur dioxide emissions down to .3 lb/MMBTU, nitrogen oxide emissions down to .15 lb/MMBTU, and .01 lb/MMBTU of PM10. Table 3 summarizes the characteristics of the plants. The Harvard researchers estimated the number of deaths, hospital admissions, emergency room visits and asthma attacks for each plant in each scenario. They considered the impacts of differences among the populations. First, the impacts on the population as a

whole were estimated assuming the population was homogeneous. However, some groups of people within the general population are especially vulnerable to air pollution's effects. These people could bear a greater share of power plant health impacts. Next the study considered heterogeneity by looking at a subset of damages and how they impacted vulnerable groups. The study evaluated three cases: 1) Mortality and how deaths are distributed by educational level, 2) Pediatric asthma emergency room visits and how these visits are distributed among African-American children, and 3) Cardiovascular hospital admissions among the elderly and how these admissions are distributed among the elderly who have diabetes.

# TABLE 3
## Plant Characteristics

| Plant | Current Owner 1. | Capacity (MW) 1. | $SO_2$ 1999 (tons) 2. | $SO_2$ Rate 1999 (lb/MMBTU) 2. | NOx 1999 (tons) 2. | NOx Rate 1999 (lb/MMBTU) 2. |
|---|---|---|---|---|---|---|
| Benning | Potomac Power Resources | 550 | 1,432 | 0.87 | 447 | 0.27 |
| Chalk Point | Mirant Corp | 2283 | 57,634 | 1.30 | 6,084 | 0.14 |
| Dickerson | Mirant Corp | 1178 | 30,641 | 1.72 | 10,956 | 0.62 |
| Possum Point | Virginia Electric Power | 1251 | 19,497 | 1.35 | 5,116 | 0.35 |
| Potomac River | Mirant Corp | 480 | 17,627 | 1.10 | 6,893 | 0.43 |

1. United States. Energy Information Administration. Form 767. Spring 2002 <http://www.eia.doe.gov/>
2. United States. Environmental Protection Agency. CEMS data in Acid Raid Scorecard. Spring 2002 <http://epa.gov/airmarkets/emissions/score00/index.html>. Table B1

Sources

Levy, Jonathan I. Briefing on Health Impacts of Power Plants: Case Studies in Massachusetts, Illinois, and Washington DC. United States Senate Environment and Public Works Committee. Senate Office Building. May 17, 2002.

Levy, Jonathan I., Susan L. Greco, and John D. Spengler. "The Influence of Population Heterogeneity on Air Pollution Risk Assessment: A Case Study of Power Plants Near Washington, DC." In press, Environmental Health Perspectives (2003).

United States. Environmental Protection Agency. EPA AIRs Data. <http://www.epa.gov/air/data/net.html>. For SO2 in 1999: 12,738,813 tons out of 18,884,520 or 67.5%. For NOx in 1999: 5,732,256 tons out of 25,394,985 or 22.5%.

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Addendum C



# The Importance of Population Susceptibility for Air Pollution Risk Assessment: A Case Study of Power Plants Near Washington, DC

*Jonathan I. Levy, Susan L. Greco, and John D. Spengler*

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In evaluating risks from air pollution, health impact assessments often focus on the magnitude of the impacts without explicitly considering the distribution of impacts across subpopulations. In this study, we constructed a model to estimate the magnitude and distribution of health benefits associated with emission controls at five older power plants in the Washington, DC, area. We used the CALPUFF atmospheric dispersion model to determine the primary and secondary fine-particulate-matter (< 2.5 μm in aerodynamic diameter) concentration reductions associated with the hypothetical application of "Best Available Control Technology" to the selected power plants. We combined these concentration reductions with concentration–response functions for mortality and selected morbidity outcomes, using a conventional approach as well as considering susceptible subpopulations. Incorporating susceptibility had a minimal effect on total benefits, with central estimates of approximately 240 fewer premature deaths, 60 fewer cardiovascular hospital admissions (CHA), and 160 fewer pediatric asthma emergency room visits (ERV) per year. However, because individuals with lower education appear to have both higher background mortality rates and higher relative risks for air-pollution–related mortality, stratifying by educational attainment implies that 51% of the mortality benefits accrue among the 25% of the population with less than high school education. Similarly, diabetics and African Americans bear disproportionate shares of the CHA and ERV benefits, respectively. Although our ability to characterize subpopulations is constrained by the available information, our analysis demonstrates that incorporation of susceptibility information significantly affects demographic and geographic patterns of health benefits and enhances our understanding of individuals likely to benefit from emission controls. *Key words:* asthma emergency department visits, cardiovascular hospital admissions, diabetes, education, mortality, particulate matter, power plant, risk assessment, susceptibility. *Environ Health Perspect* 110:1253–1260 (2002). [Online 29 October 2002]
*http://ehpnet1.niehs.nih.gov/docs/2002/110p1253-1260levy/abstract.html*

The issue of subpopulation susceptibility to fine particulate matter (< 2.5 μm in aerodynamic diameter; $PM_{2.5}$) has been given increased attention by researchers in recent years, motivated in part by the research priorities articulated by the National Academy of Sciences (*1*). Understanding patterns of susceptibility not only would help identify and protect sensitive subpopulations, but also would contribute to the understanding of mechanisms by which $PM_{2.5}$ might influence human health.

Often, air pollution policies are informed by risk assessments or benefit–cost analyses, which generally focus on the total health benefits of alternative emission control strategies (*2–5*). Because relevant susceptibility evidence is limited, differential effects on susceptible subpopulations are rarely incorporated. Typically, the same relative risks are applied to all individuals in an "at-risk" age group, and baseline rates of disease or health care use are assumed to be uniform across large geographic areas (often national averages).

However, it is likely that the effects of air pollution vary widely across subpopulations, depending on demographics, behavior patterns, income, access to health care, and other factors. Differences could exist either in relative risks (if an increment of air pollution yields a different percentage increase in different populations) or in absolute risks (if there are differences in baseline disease patterns by subpopulation, independent of air pollution). For a benefits assessment, if policy makers were concerned about distributional issues or if the ultimate valuation of benefits depended on population characteristics, the incorporation of susceptibility could potentially influence the conclusions.

One current policy issue for which information on susceptibility could be influential is the regulation of emissions from older power plants. To date, older power plants have not been required to meet the same control requirements as new sources, helping to extend the useful lifetime of older facilities (*6–8*). These facilities contribute a substantial fraction of national power sector emissions. In 1999, coal-fired power plants contributed approximately 86% of nitrogen oxide ($NO_x$) emissions and 93% of sulfur dioxide emissions from the utility sector, largely from facilities exempted from new source standards (*9*).

At the time this article was written (2001), several states (including Massachusetts, Connecticut, and Texas) had introduced multipollutant regulations or legislation to require older power plants to meet emission levels commensurate with the application of "Best Available Control Technology" (BACT; technology required under the Clean Air Act for new or modified sources in attainment areas). Pollutants considered typically included $NO_x$ and $SO_2$, as well as mercury and carbon dioxide. Multipollutant power plant legislation was also being debated at the federal level, but no bills or regulations had been passed at the time of our analysis.

From both a state and a federal perspective, the question of how the benefits of emission controls would be distributed could be important. Policy makers may be concerned about providing benefits to high-risk communities, communities near power plants, or other subpopulations. If these questions are important, population susceptibility could influence the policy choices (e.g., emission trading vs. mandatory on-site controls).

In this article, we develop a model to estimate the health benefits associated with emission reductions at older fossil-fueled power plants. We focus on both primary $PM_{2.5}$ and secondary sulfate and nitrate particles formed through emissions of $SO_2$ and $NO_x$. Here we consider a case study of all older power plants located within a 50-mile (80-km) radius of Washington, DC. We calculated three health end points—premature mortality, cardiovascular hospital admissions (CHA) in the elderly, and pediatric asthma emergency room visits (ERV)—both using conventional assumptions and then considering available evidence for differential effects on susceptible subpopulations. Our goal was both to quantify the health benefits associated with the implementation of BACT at the selected power plants and to consider whether introduction of susceptibility models might affect the interpretation of our findings.

## Case Study Setting

For this analysis, our goal was to select a geographic area that had multiple older power plants nearby and geographic heterogeneity in

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factors that might influence relative risks, baseline health status, or health care use (e.g., socioeconomic status). Washington, DC, and its surrounding suburbs provide an example of such a region. According to 1990 U.S. Census data (*10*), median household income in Washington, DC, ranged from less than $10,000 to more than $150,000 across census tracts. Washington, DC, is also quite racially divided, with few African Americans residing in the western half of the city and mostly African Americans residing in the eastern half of the city.

In addition, within a 50-mile (80-km) radius of Washington, DC, there are five fossil-fueled power plants grandfathered under the Clean Air Act—Benning, Chalk Point, Dickerson, Possum Point, and Potomac River (Table 1). The choice of these five power plants is somewhat artificial because any single regulation would not affect only these plants. However, our analysis is meant to be illustrative, and these five plants are likely the greatest contributors to heterogeneity in power-plant–related exposures in the area. Inclusion of additional power plants would increase the total benefits but decrease the relative concentration gradient across the Washington, DC, area.

## Methods

To quantify the magnitude and distribution of health benefits, we estimated the emission reductions of key pollutants, applied an atmospheric dispersion model to determine incremental concentration reductions, and derived concentration–response functions. Any such analysis involves numerous boundary decisions and contains substantial uncertainties. In this article, we focus largely on issues related to susceptible subpopulations and the resulting implications. We do not extensively address the complexities of other elements of the model, nor do we provide a formal analysis of uncertainties. We also do not consider the economic valuation dimension of a benefits assessment. Additional information about parametric uncertainties in our atmospheric model (*4,11*) and issues related to differential particle toxicity or alternative interpretations of the health evidence (*4*) can be found elsewhere.

***Quantification of emissions.*** We estimated emissions of $PM_{2.5}$ and its precursors ($NO_x$ and $SO_2$) following the model structure in our earlier analyses (*4,11*) and supported by the fact that $PM_{2.5}$ has dominated aggregate benefits in past air pollution risk assessments (*2,3*). This omits any benefits associated with ozone, air toxics, or other impact pathways from the power sector. Of note, most proposed regulations consider $NO_x$ and $SO_2$ but do not directly require controls for primary $PM_{2.5}$ (although many $NO_x$ and $SO_2$ control strategies would affect primary $PM_{2.5}$).

We used 1999 as the base year for our analysis, evaluating the concentration and health benefits that would have been obtained had lower target emission rates been achieved. This is not identical to the future benefits that might be obtained through pending regulation, because some facilities have ongoing or near-term plans for repowering or emission controls.

Emissions of $SO_2$ and $NO_x$ were taken from the 1999 acid rain program emissions scorecard from the U.S. Environmental Protection Agency (EPA) (*12*). To capture seasonality in emissions, we incorporated quarterly average emission rates when reported. When no data on seasonal emissions were available, we assumed constant emissions per unit of heat input. For filterable $PM_{2.5}$, total plant emissions were taken from the U.S. EPA National Emission Trends database (*13*). We estimated condensable $PM_{2.5}$ emissions given fuel type and sulfur content, using AP-42 air pollution emission factors from the U.S. EPA (*14*).

We selected lower target emissions to correspond to the levels proposed in multiple regulations, which correspond to the application of BACT. This resulted in target emission rates of 0.3 lb/MMBTU (million British thermal units) of $SO_2$, 0.15 lb/MMBTU of $NO_x$, and 0.01 lb/MMBTU of filterable particulate matter. Lower target condensable particulate emissions were taken from AP-42, given assumed application of control technologies. Because both Dickerson and Benning power plants have actual filterable $PM_{2.5}$ emissions less than the lower target rate, we set the lower target filterable $PM_{2.5}$ emission rate equal to actual emissions for these plants.

***Atmospheric modeling.*** We established a receptor grid covering a 400-km (250-mile) radius around Washington, DC (centered at 38.9°N, 77°W), to capture a significant fraction of total benefits without extending the dispersion modeling boundaries excessively (Figure 1). Because of our focus on spatial patterns, it was important to determine concentration reductions at small geographic scales close to the sources. We selected census tracts within 100 km of Washington, DC, because they are relatively small (generally between 2,500 and 8,000 people) and were theoretically designed to be socioeconomically homogeneous. Beyond 100 km, we used county-level resolution, resulting in a nested receptor grid with 1,908 receptors. Using 1990 Census data (*10*) (the most recent data available at the time of our study), our receptor grid contained 47 million individuals, 7 million of whom live within 100 km of Washington, DC.

We conducted our atmospheric modeling using CALPUFF (CALMET version 5.2 000602a, CALPUFF version 5.4-000602-1, CALPOST version 5.2 991104b; Earth Tech, Concord, MA). CALPUFF is a regional-scale Lagrangian puff model that has been recommended by the U.S. EPA for long-range transport modeling (*15*), given that it has been shown to be relatively unbiased at distances out to 200 km (*16*). In general, limitations in the atmospheric chemistry make the secondary pollutant estimates relatively more uncertain than the primary $PM_{2.5}$ estimates, given the nonlinearities associated with sulfate and nitrate formation.

Our methodology to generate meteorologic files for CALMET was similar to the approach in our past applications and is described in depth elsewhere (*4,11*). We combined National Oceanic and Atmospheric Administration (NOAA) prognostic model outputs with mesoscale data assimilation systems for each hour across our case study year (January 1999–January 2000). This involved combining lower-resolution upper air data (40-km grid spacing) generated through NOAA's Rapid Update Cycle (RUC2) model (*17*) with Aviation Routine Weather Report (METAR) surface observations and cloud cover data available at 15 km resolution (*18*). These data sources were combined using the Advanced Regional Prediction System (ARPS) Data Assimilation System (ADAS) and provided hourly CALMET windfields within eight vertical layers. Precipitation data were taken from all National Climatic Data Center stations within the receptor region, with CALMET

**Table 1.** Characteristics of five power plants in Washington, DC, case study (1999 data).

| Characteristics | Benning | Chalk Point | Dickerson | Possum Point | Potomac River |
|---|---|---|---|---|---|
| Initial year of commercial operation | 1968 | 1964 | 1959 | 1948 | 1949 |
| Nameplate capacity (megawatts) | 580 | 2,046 | 588 | 1,373 | 514 |
| Heat input (MMBTU) | 3,304,107 | 85,352,274 | 33,592,811 | 28,930,805 | 32,100,184 |
| Emissions, tons (% per quarter) | | | | | |
| $SO_2$ | 1,432 (2, 21, 76, 2) | 57,630 (21, 25, 31, 23) | 30,637 (30, 17, 34, 18) | 19,497 (24, 22, 32, 23) | 17,627 (22, 28, 29, 21) |
| $NO_x$ | 447 (2, 22, 74, 1) | 25,222 (20, 24, 30, 26) | 10,709 (30, 17, 34, 18) | 5,116 (25, 22, 32, 21) | 6,893 (21, 28, 30, 21) |
| $PM_{2.5}$ | 12 (2, 22, 74, 2) | 304 (21, 27, 33, 20) | 14 (30, 17, 34, 18) | 156 (23, 20, 37, 20) | 106 (21, 28, 29, 22) |

defaults used for interpolation between stations. The primary difference from our previous applications was the inclusion of 50 evenly spaced "soundings" based on columns of the ADAS data, to more accurately provide a reasonable high-resolution temperature field and subsequent planetary boundary-layer depth estimates.

In CALPUFF, we adopted recommended modeling assumptions that were used in our past applications (*4,11*). We used the MESOPUFF II chemical transformation mechanism, which is generally preferred in urban settings. Wet and dry deposition were incorporated using precipitation data and CALPUFF default deposition rates. Hourly background ozone concentrations were taken from five U.S. EPA Clean Air Status and Trends Network (CASTNET) stations spaced throughout our receptor region (Prince George's County, MD; Mercer County, NJ; Elk County, PA; Prince Edward County, VA; Gilmer County, WV), and we assumed a background ammonia concentration of 1 ppb.

For brevity's sake, in this article we do not provide sensitivity or uncertainty analyses for our atmospheric modeling. In our past analyses (*4,11*), we found total benefits to be reasonably stable given single parametric changes in CALPUFF, including the chemical conversion mechanism, background ammonia concentration, and treatment of wet and dry deposition. In addition, we concluded that any bias associated with either hypothetical CALPUFF overestimation beyond 200 km or exclusion of long-range exposures is relatively small in comparison with other model uncertainties. A comprehensive risk assessment would need to incorporate these uncertainties in an evaluation of overall model uncertainty.

***Health evidence.*** Although numerous health outcomes have been incorporated into past analyses (*2*), here we focus on a subset for which some evidence exists for differential effects on susceptible subpopulations. The choice of outcomes as well as the subpopulations considered therefore depends entirely on the current literature and is not meant to be comprehensive. Furthermore, we restricted the health evidence to epidemiologic studies conducted in the United States, because patterns of health care use and the relationship between demographics and health status likely vary across countries. Given these criteria, we evaluated premature mortality (stratified by education), CHA for the elderly (stratified by diabetic status and age), and asthma ERV for children (stratified by race and age). For each outcome, we both describe a conventional approach and construct a susceptibility model. Our goal is not to consider the complete array of susceptible subpopulations, but rather to select one example for each outcome for which epidemiologic evidence and population data exist.

***Premature mortality.*** For premature mortality, we derived a central estimate from the follow-up analysis of the American Cancer Society (ACS) cohort study (*19*). Several other cohort studies are available (*20,21*), but the ACS study has the largest and most geographically diverse population, with relative risks bounded by other studies and a statistical approach suggested by a detailed reanalysis (*22*). For all-cause mortality, the authors calculated a relative risk of 1.04 [95% confidence interval (CI), 1.01–1.08] for a 10 $\mu g/m^3$ increase in annual mean $PM_{2.5}$ concentrations (using 1979–1983 concentrations). The relative risk was slightly higher (1.06) using more recent pollution data, but we use the lower figure to be conservative and because Pope et al. (*19*) presented stratified estimates based on the 1979–1983 concentrations.

Relative risks did not vary substantially across most demographic factors except educational attainment. Educational attainment appeared to be a strong effect modifier across all causes of mortality. The relative risk for a 10 $\mu g/m^3$ increase in annual mean $PM_{2.5}$ concentrations was 1.085 (95% CI, 1.031–1.142) for individuals with less than high school education, 1.045 (95% CI, 1.004–1.087) for individuals with high school education, and 1.003 (95% CI, 0.967–1.040) for individuals with more than high school education.

There are numerous uncertainties related to the application of this stratified relative risk. The ACS cohort is somewhat more educated than the population at large, and correlated terms such as race and poverty status have not been significant in time-series mortality or hospital admissions studies (*23–25*). In addition, the statistical approach implies that we are modeling the effect of education controlling for smoking and other factors, which would ideally be included to model the influence of all risk factors correlated with educational attainment. Regardless, we use the education-stratified values to determine the implications of the reported relationship.

For background mortality rates, the standard approach is to apply county-level averages to individuals 30 or more years old [the age range considered in the ACS study (*19*)]. We used this as our baseline approach, but for our susceptibility model, we considered whether mortality rates vary as a function of education while still averaging to the reported county-level rates.

There is a strong and consistent negative relationship between socioeconomic status and all-cause mortality (*26*). Socioeconomic status can be measured by occupation, income, education, or some combination of

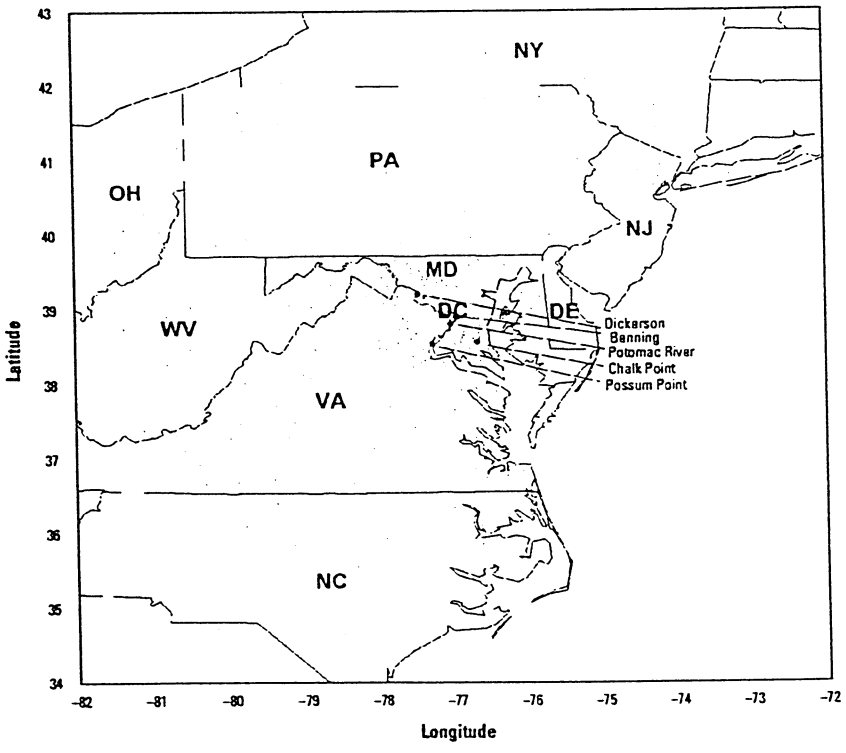


**Figure 1.** Receptor grid and power plant locations for Washington, DC, case study.

these terms. It is generally believed that both income (*27*) and educational attainment (*28*) are independent predictors of mortality, although the bases for these relationships are not well understood. Some argue that those in lower socioeconomic classes display high-risk behaviors, such as smoking, being overweight, and not exercising (*29*), producing higher mortality rates. However, only a small fraction of the increased mortality can be explained by a higher prevalence of high-risk behaviors (*30*), so there must be other contributing factors. In any case, it is clear that those in low education or income categories represent a susceptible subpopulation for all-cause mortality.

Educational attainment is a useful predictor of mortality because it typically does not change after adulthood. Additionally, this term is available for all segments of the adult population, even those not in the work force. Although it may be a proxy for other factors, various hypotheses have been presented for why lower education might be a causal factor for mortality. Education may be a marker for factors (e.g., intelligence and good health in early childhood) that allow for both educational attainment and good health in adulthood, for acquired knowledge that can be used to obtain positive health outcomes, for relative status in society, or for the development of positive social networks (*31*). The protective effect of higher education has been seen in the United States (*31*) and worldwide (*32,33*).

We selected our baseline mortality risk ratios from a study that evaluated risks for all-cause mortality as a function of both education and annual income among a cohort 25–64 years old, drawn from the National Longitudinal Mortality Study (*31*). The relationship between education and mortality was best described by a trichotomy (less than high school education, high school diploma or greater but no college diploma, or a college diploma or greater). When compared with the highest education group, the annual mortality relative risk for men was 1.7 for less than high school education and 1.5 for high school diploma or greater but no college diploma. For women, the corresponding relative risks were 1.5 and 1.2. The attenuation in women has been documented previously and can be attributed largely to the married subpopulation of women (*34*). We applied these relative risks to all individuals more than 30 years old, although there is some evidence that socioeconomic differences play less of a role in determining mortality rates among the aged (*35*).

***Cardiovascular hospital admissions.*** Several studies in the United States have evaluated the relationship between particulate matter exposure and CHA among individuals 65 or more years old (*24,25,36–43*). Most central estimates from these studies fall in the range of a 0.5–1% increase in CHA for a 10 $\mu g/m^3$ increase in daily concentrations of particulate matter < 10 μm in aerodynamic diameter ($PM_{10}$). Using a typical $PM_{2.5}$:$PM_{10}$ ratio of 60%, we would consider appropriate a central estimate of an approximate 1% increase in CHA per 10 $\mu g/m^3$ increase in daily $PM_{2.5}$ concentrations. As a baseline, we applied this percentage to the average background rate of 0.084 CHA per year per individual ≥ 65 years old (*44*).

Although numerous factors might influence either the baseline risk or the relative risk of an air-pollution–related CHA, we focused on diabetes to illustrate the influence of a risk factor that varies demographically and might influence both risks. To estimate the number of diabetic and nondiabetic CHA in a county or census tract, we considered two relationships: the risk factors for diabetes among the elderly and the differential risk for a CHA given the presence of diabetes.

In those > 65 years old, noninsulin-dependent diabetes mellitus (NIDDM) accounts for virtually all of the diabetic caseload. There are numerous risk factors for NIDDM, including age, obesity, family history, and sedentary lifestyle. Although lifestyle variables are the strongest predictors of diabetic status [accounting for as much as 90% of population attributable risk (*45*)], we cannot estimate these variables at the census tract level from publicly available data. In the absence of this information, we estimated NIDDM prevalence as a function of gender, age, and race. According to a national survey (*46*), NIDDM prevalence in individuals > 65 years old is higher among African Americans and Mexican Americans than in non-Hispanic whites, ranging from 10.9% for non-Hispanic white males 65–74 years old to 29% for Mexican-American females 65–74 years old. We applied these estimates to our study populations, despite the limitations in applying national relationships based on race to a specific geographic setting. The relationship between race and common risk factors likely varies widely across regions and within small geographic areas, a feature that is not captured by our model.

Regarding risks for a CHA, it has been well established that diabetics have an increased risk of heart disease. Several studies also indicate that diabetics are admitted to the hospital more frequently than are nondiabetics (*47,48*). Thus, it is not surprising that CHA rates are elevated in diabetic populations. According to a national diabetes surveillance report (*49*), as of 1996, the annual CHA rate was 0.20 admissions per year per diabetic 65–74 years old and 0.27 for diabetics ≥ 75 years old. In contrast, the rates for the population as a whole are 0.06 (ages 65–74 years) and 0.11 (≥ 75 years) (*44*). Using these two rates and the estimated diabetes prevalence across our study population, we can calculate the CHA rate for nondiabetics. Clearly, there are several appreciable assumptions underlying these estimates. Although we know that marked differences can exist in CHA rates among states and communities, we assume that tract-specific rates vary only as a function of the estimated number of diabetics, with CHA rates invariant for nondiabetics. This likely underestimates the degree of spatial and demographic variability in CHA rates.

On the relative risk side, a time-series study in Chicago (*38*) found a 2% increase in CHA for diabetic individuals > 65 years old for a 10 $\mu g/m^3$ increase in $PM_{10}$, versus a 0.9% increase for nondiabetics. In contrast, the studies that evaluated factors such as race, education, or poverty (*24,37,43*) found no significant effect modification for CHA relative risks. To ensure that our concentration–response function agrees with our nonstratified estimate, we assumed that a factor of two difference exists between diabetics and nondiabetics and calculated the concentration–response function given the estimated number of CHA in diabetics and nondiabetics in our study population. The result is a 0.7% increase in CHA per 10 $\mu g/m^3$ increase in $PM_{2.5}$ for nondiabetics, with a 1.5% increase for diabetics.

***Pediatric asthma ERV.*** Many studies have associated ERV for numerous respiratory and cardiovascular causes with particulate matter, but to date only two studies in the United States have considered asthma-related visits among children (defined here as ≤ 18 years old). In Seattle (*50*), an 11.6 $\mu g/m^3$ increase in $PM_{10}$ was associated with a 14% increase in asthma ERV (95% CI, 5–23%), and a 9.5 $\mu g/m^3$ increase in $PM_{2.5}$ was associated with a 15% increase. This study found the relative risk to be similar in high-use and low-use areas (a proxy for socioeconomic status). In Atlanta (*51*), a 4% increase in pediatric asthma ERV was estimated for a 15 $\mu g/m^3$ increase in $PM_{10}$ concentrations (95% CI, 0.4–7%). As in Seattle, there did not appear to be effect modification due to race or socioeconomic status. Simply pooling these two studies using a random effects model (*52*) provides a central estimate of a 0.7% increase in asthma ERV per microgram per cubic meter increase in $PM_{10}$, which we translate into an approximate 1% increase in asthma ERV per microgram per cubic meter increase in daily $PM_{2.5}$. This can be applied to a background asthma ERV rate of 0.012 for children 0–4 years old, 0.0081 for children 5–14 years old, and 0.0069 for children ≥ 15 years old (*53*).

Although the published studies did not identify susceptible subpopulations from a relative risk perspective, the background rate

of asthma ERV would be anticipated to differ widely across subpopulations. This would be a function both of trends in asthma prevalence and of patterns in health care use across populations.

The prevalence of asthma has increased substantially in recent years (*53*), with lower-income individuals and minorities disproportionately affected by the disease (*54–58*). Many of the significant predictors of childhood asthma, such as cockroach presence in the home (*59*) and maternal education (*60*), are related to socioeconomic status. Furthermore, patterns of health care use are strongly related to income. The ratio of anti-inflammatory to beta-agonist medication is lower in low-income communities and is inversely correlated with hospitalization rates (*61*), and lower-income populations lacking health insurance often use emergency services as a means of primary care. Thus, it would be expected that low-income populations would have somewhat higher pediatric asthma ERV rates.

Data on pediatric asthma ERV rates as a function of income were limited, but substantial racial differences have been documented. According to data from the National Hospital Ambulatory Medical Care Survey (*53*), across all ages, the asthma ERV rate for African Americans is nearly five times greater than that for whites (0.023 and 0.0049 per capita, respectively). No data were provided on asthma ERV rates stratified across both age and race, but a study of 3-year-olds in the United States found a racial differential of similar magnitude but with some independent effects of both race and income (*54*).

Given available information, we estimated baseline pediatric asthma ERV rates as a function of age and race, assuming the racial disparity to exist in all age groups. This encompasses differences both in prevalence and in health care use. As with our diabetes estimates, there are some substantial limitations in using only race as a predictor, because the relationship between race and asthma ERV risk factors varies by income, urban/rural status, and other factors. Regardless, the consistent relationship between race and ERV and the ability to gather racial information at the census tract level make this the best available covariate.

## Results

*Concentration reductions.* With our atmospheric dispersion model, the emission reductions at the five selected power plants would lead to annual average $PM_{2.5}$ (primary plus secondary) concentration reductions ranging from 0.009 to 0.9 μg/m³ in our receptor region (Figure 2C). By way of comparison, according to U.S. EPA AIRS data (*62*), annual average $PM_{2.5}$ concentrations in Washington, DC, were approximately 14–18 μg/m³ in 1999. The maximum annual average $PM_{2.5}$ concentration reduction is found within Washington, DC, as might be anticipated by the power plant selection criteria and the inclusion of primary $PM_{2.5}$.

The geographic distribution of benefits varies somewhat across particle types, power plants, and seasons. Annual average primary $PM_{2.5}$ concentration reductions peak closer to the plants and decrease more rapidly with distance than secondary sulfates or nitrates (Figure 2). As a result, a greater fraction of total exposure reduction (defined as the sum across receptors of the product of concentration reduction and population assigned to the receptor) occurs closer to the power plants for primary than for secondary $PM_{2.5}$ (Figure 3). However, there is tremendous variability in the distribution of total exposure reduction, caused principally by variations in source locations and pollutant type (primary vs. secondary). In addition, total exposure reduction per unit emissions displayed expected seasonal patterns, with slightly higher values for primary $PM_{2.5}$ in the winter and fall (related in part to lower mixing heights) and higher values for sulfates and lower values for nitrates in the summer due to the effect of temperature on relative conversion rates.

*Health benefits.* For premature mortality, using nonstratified relative risks and homogeneous baseline mortality rates within counties, our central estimate is that emission reductions from the five power plants would lead to 210 fewer deaths per year (Table 2). The estimated impact under the current emissions scenario is 270 deaths per year. Of the total mortality benefits, approximately 25% occur in individuals with less than high school education (identical to the proportion in the population). Approximately 16% of mortality benefits accrue within 50 km of the power plants, largely related to the substantial contribution of secondary sulfates (62%) and nitrates (19%) to total $PM_{2.5}$ exposures.

In our susceptibility model, with both baseline mortality rates and $PM_{2.5}$ relative risks stratified by educational attainment, our understanding of the affected subpopulations changes substantially (Table 2). The total mortality benefit is largely unaffected, with a slight increase associated with differences in educational attainment between the Washington, DC, area and the ACS cohort. However, 51% of the estimated mortality benefits now accrue among individuals with less than high school education, double the prediction in the homogeneous risk model.

Although stratification by education does not significantly influence the broad geographic patterns of benefits (i.e., the fraction of benefits within 50 km), at the census tract level benefits differ by as much as a factor of 13 between the models. Figure 4 depicts the geographic patterns of benefits under both the baseline and susceptibility models, focusing solely on census tracts in Washington, DC, for simplicity. Using the baseline model, the mortality risk reductions in Washington are reasonably homogeneous, ranging from 36 to 67 fewer deaths per year per million individuals > 30 years old. Under the education-stratified

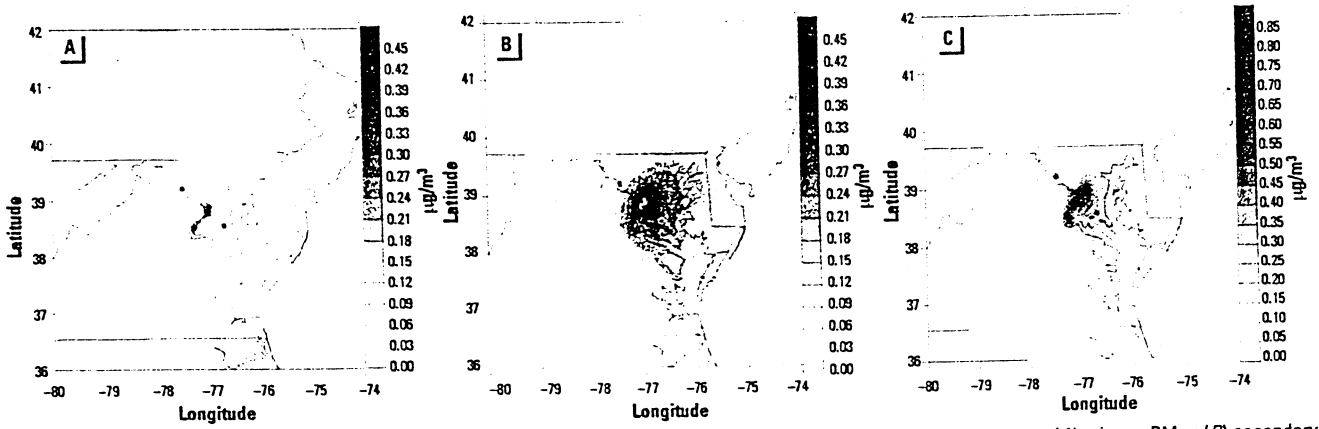


**Figure 2.** Combined concentration reductions (annual average, μg/m³) from hypothetical emission controls at five power plants: (*A*) primary $PM_{2.5}$; (*B*) secondary $PM_{2.5}$; (*C*) total $PM_{2.5}$.

model, the range broadens considerably and the distribution is more complex, with per capita benefits now varying by more than a factor of 10 across census tracts. The mortality benefits are generally increased in southeastern Washington, DC, the lowest-income area of the city.

When we consider CHA among the elderly, our baseline model estimates 59 fewer CHA per year. Although it seems counterintuitive that the mortality numbers could exceed the morbidity numbers, this is related to the limited focus on CHA because of only short-term exposures among the elderly (vs. all-cause mortality from long-term exposures among individuals ≥ 30 years old). Using a conventional model that assumes diabetics do not differ in any way from nondiabetics, 13% of the CHA benefits are estimated to occur among diabetics, whereas 80% are found among non-Hispanic whites (Table 2). The geographic distribution of CHA benefits is similar to the exposure reduction and mortality benefits, with differences reflecting the relative number of individuals 65–74 years old and ≥ 75 years old within census tracts.

As expected, incorporating the diabetes-based information has a minimal impact on aggregate benefits but dramatically alters the profile of the affected individuals (Table 2). Using this model, 54% of the CHA benefits are found among diabetics, with 76% among non-Hispanic whites. Because we have assumed that baseline CHA risk for nondiabetics does not differ as a function of race or income, the CHA estimates under the susceptibility model are closer to those from the baseline model than are those for mortality (Figure 4). However, even considering only diabetes-related susceptibility changes the census tract-level benefits by as much as 40%.

Finally, we estimate 140 fewer pediatric asthma ERV per year using our nonstratified model (38% in children 0–4 years old, 46% in children 5–14 years old). Twenty-seven percent of benefits occur in African-American children (who represent 21% of the study population). When we stratify asthma ERV risk by race, the total benefits increase to 160 fewer visits per year, with significant changes in the geographic and demographic distributions (Table 2). The census-tract–level risk reduction varies by an order of magnitude across Washington, DC, with the benefits increased by more than a factor of two in the eastern half of the city (Figure 4). The proportion of benefits among African-American children is increased to 64%, commensurate with the assumption of greater baseline asthma ERV rates.

## Discussion

Our analytical approach demonstrates two important points. First, given an interpretation of the epidemiologic evidence that assumes that ambient concentrations in the Washington, DC, area exceed any potential population threshold for $PM_{2.5}$ health effects, emission controls at older fossil-fueled power plants would provide tangible and quantifiable health benefits. Second, when we take account of susceptible subpopulations and differences in both relative risk and baseline disease rates across these populations, the small-scale geographic and demographic distributions of those benefits are strongly affected. For the example of premature mortality, if educational attainment influences both the relative risk of air pollution and the baseline mortality risk, then more than half of the mortality benefits accrue among the 25% of our study population with less than high school education. Similarly, for pediatric asthma ERV, the fact that background rates are substantially greater in African Americans implies that most ERV benefits accrue in 21% of the population, even given identical relative risks from air pollution. The relatively smaller differences found for CHA when diabetes is considered illustrates that evidence for differential effects on a relatively small fraction of the population has a smaller effect than a population-wide model.

There are clearly some barriers in both interpretation of the study findings and application of our model to other settings. One important uncertainty is related to the stratified risk models we selected. For all health outcomes, we used stratification variables (such as race) that might have independent effects on baseline health but likely are proxies for numerous socioeconomic end points. If the stratification variables represent other factors, this adds to the uncertainty in a site-specific stratified analysis.

In general, we have applied susceptibility models based on national data to a small number of states, which has multiple inherent limitations. Clearly, it would be preferable to use local health data, but data at small geographic scales for a large region are difficult to obtain and are rarely stratified across all demographic variables of interest. In addition, the reliance on national data increases the generalizability of our findings. Despite

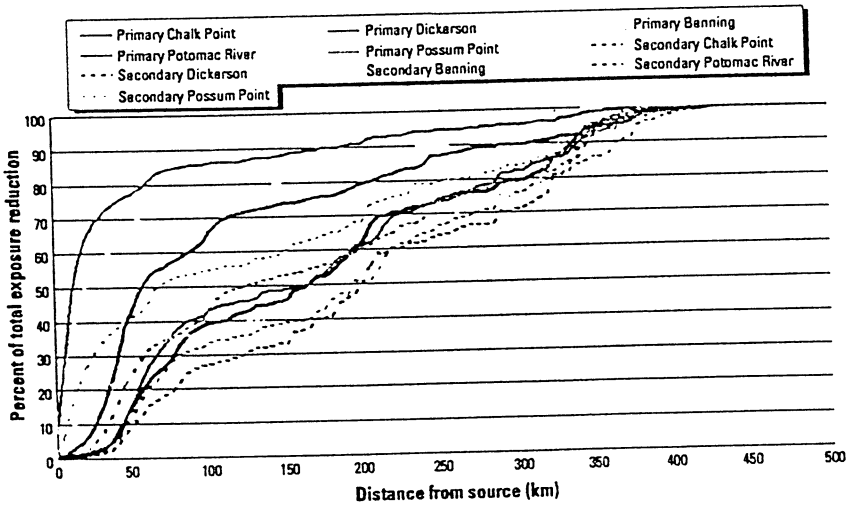


**Figure 3.** Cumulative distribution of total exposure reduction as a function of distance from the source, by power plant and pollutant type.

**Table 2.** Magnitude and distribution of health benefits associated with modeled emission reductions at five power plants near Washington, DC.

| Health outcome and stratification covariate | Baseline model (No stratification) | Full susceptibility model (Stratification by listed covariate) |
|---|---|---|
| Deaths/year | | |
| Total | 210 | 240 |
| < High school education | 52 | 120 |
| ≥ High school education | 150 | 120 |
| CHA/year | | |
| Total | 59 | 60 |
| Diabetic | 8 | 33 |
| Nondiabetic | 51 | 27 |
| Asthma ERV/year | | |
| Total | 140 | 160 |
| African American | 38 | 100 |
| Non-African American | 100 | 57 |

Data presented are rounded to two significant figures; sums may not add because of rounding.

these issues, our models demonstrate that simple assumptions about susceptibility can be influential in our understanding of health risks and benefits. The alternative is an assumption of homogeneity, which itself introduces implicit uncertainty and may contribute to biases in selected settings.

Another limitation of our study is the fact that we have devoted limited attention to uncertainty analysis, a crucial element in interpreting sensitive and complex findings. Drawing on the uncertainty analyses in our earlier work (*4,11*), most parametric changes in CALPUFF led to changes to aggregate benefits of less than a factor of two, whereas variations in concentration–response assumptions (particularly for mortality) could influence estimates by as much as a factor of five. The influence of population susceptibility is generally at the lower end of this range, even for small geographic scales. However, susceptibility information has a greater influence on the relative distribution of benefits than do other assumptions, many of which tend to affect all populations identically (e.g., the population-averaged concentration–response function). Furthermore, a broader view of areas of heterogeneity or susceptibility [e.g., assumptions regarding particle size and chemical composition, time–activity data, or physiologic factors (*63*)] could increase the importance of this evidence. Further analysis that considers the full array of uncertainties and evaluates which (if any) would be influential in policy decisions would be warranted.

In addition, although we have focused on power plants (partly because of pending regulatory decisions at the time of our analysis), the issue of susceptible subpopulations is likely more significant for motor vehicle pollution. Given that motor vehicles have low stack heights and have a strong presence in urban street canyons with high population density, it is likely that aggregate impacts would be spread over a smaller population than for power plants. If the exposed population had demographic differences from the United States average, assumptions of homogeneity would bias the risk calculations.

Finally, any assessment of impacts from a limited number of sources is somewhat impaired by the relatively small reductions when compared with baseline concentrations. This makes field validation of model results difficult and implies that an ultimate comparison of the costs and benefits of taking action would be required to determine if action is warranted.

Despite these limitations, our analysis illustrates that emission controls at older fossil-fueled power plants could lead to quantifiable concentration and health benefits and that susceptibility information informs the interpretation of those benefits. Although the individual benefits represent a small increment over baseline risks, the number of people affected because of long-range pollution transport implies aggregate benefits that are relevant for policy evaluation. As the health literature develops additional information about differences in relative and absolute risk across populations, risk assessments and benefit–cost analyses should take advantage of this information to provide more interpretable information to decision makers.

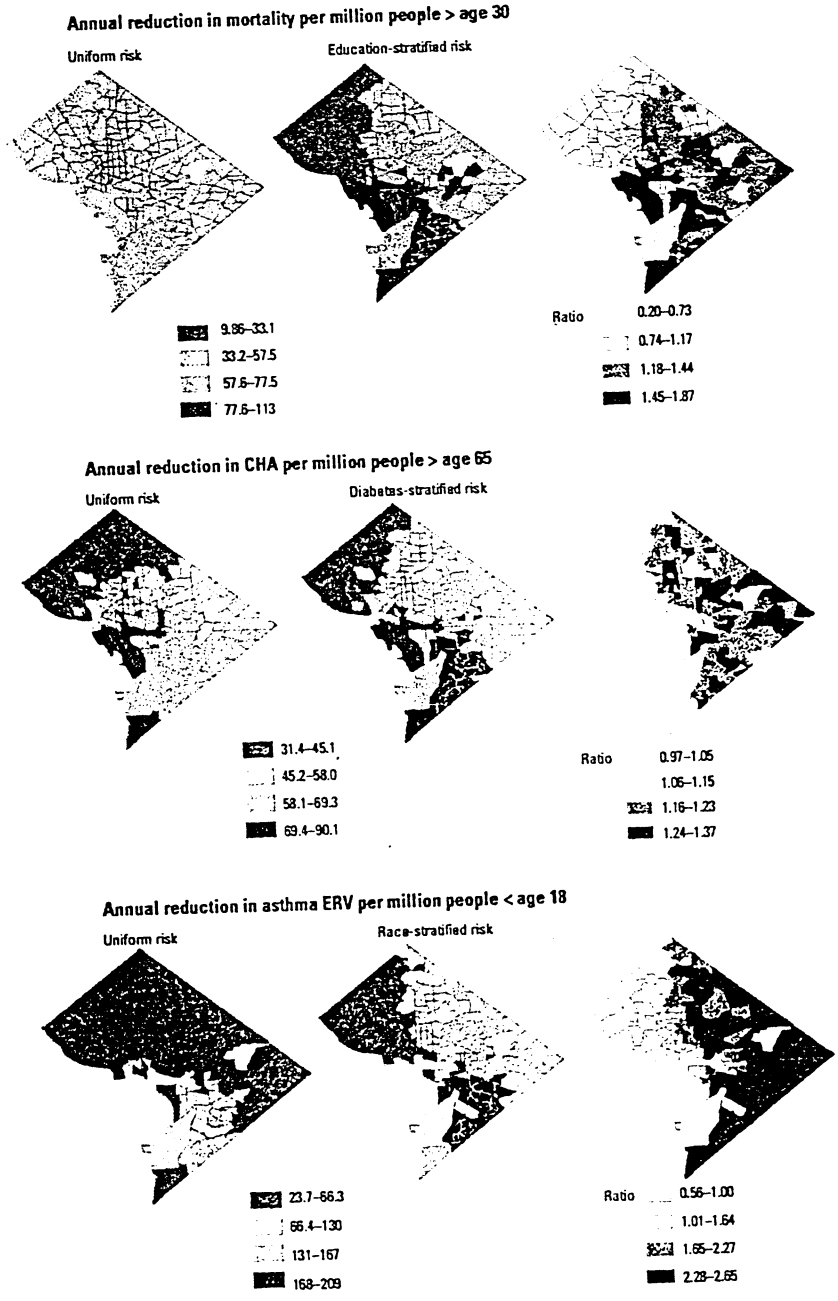


**Figure 4.** Distribution of health benefits by census tract in Washington, DC (no color indicates zero at-risk population).

## Conclusions

We have evaluated the health benefits of emission controls at five older fossil-fueled power plants in the Washington, DC, area, using conventional risk assessment assumptions and incorporating available information about susceptible subpopulations. We found that the geographic and demographic distributions of benefits differ substantially between the two approaches. If robust and causal, our susceptibility models identify subpopulations that

bear a disproportionate air pollution burden and account for a substantial fraction of the benefits of emission controls (lower-educated individuals for mortality, diabetics for CHA, and African Americans for asthma ERV). The characterization of high-risk subpopulations can help both in the interpretation of the risk assessment and in targeting future exposure assessment or epidemiologic efforts.

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# Lung Cancer, Cardiopulmonary Mortality, and Long-term Exposure to Fine Particulate Air Pollution

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**Context** Associations have been found between day-to-day particulate air pollution and increased risk of various adverse health outcomes, including cardiopulmonary mortality. However, studies of health effects of long-term particulate air pollution have been less conclusive.

**Objective** To assess the relationship between long-term exposure to fine particulate air pollution and all-cause, lung cancer, and cardiopulmonary mortality.

**Design, Setting, and Participants** Vital status and cause of death data were collected by the American Cancer Society as part of the Cancer Prevention II study, an ongoing prospective mortality study, which enrolled approximately 1.2 million adults in 1982. Participants completed a questionnaire detailing individual risk factor data (age, sex, race, weight, height, smoking history, education, marital status, diet, alcohol consumption, and occupational exposures). The risk factor data for approximately 500 000 adults were linked with air pollution data for metropolitan areas throughout the United States and combined with vital status and cause of death data through December 31, 1998.

**Main Outcome Measure** All-cause, lung cancer, and cardiopulmonary mortality.

**Results** Fine particulate and sulfur oxide–related pollution were associated with all-cause, lung cancer, and cardiopulmonary mortality. Each 10-μg/m$^3$ elevation in fine particulate air pollution was associated with approximately a 4%, 6%, and 8% increased risk of all-cause, cardiopulmonary, and lung cancer mortality, respectively. Measures of coarse particle fraction and total suspended particles were not consistently associated with mortality.

**Conclusion** Long-term exposure to combustion-related fine particulate air pollution is an important environmental risk factor for cardiopulmonary and lung cancer mortality.

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Based on several severe air pollution events,[1-3] a temporal correlation between extremely high concentrations of particulate and sulfur oxide air pollution and acute increases in mortality was well established by the 1970s. Subsequently, epidemiological studies published between 1989 and 1996 reported health effects at unexpectedly low concentrations of particulate air pollution.[4] The convergence of data from these studies, while controversial,[5] prompted serious reconsideration of standards and health guidelines[6-10] and led to a long-term research program designed to analyze health-related effects due to particulate pollution.[11-13] In 1997, the Environmental Protection Agency adopted new ambient air quality standards that would impose regulatory limits on fine particles measuring less than 2.5 μm in diameter ($PM_{2.5}$). These new standards were challenged by industry groups, blocked by a federal appeals court, but ultimately upheld by the US Supreme Court.[14]

Although most of the recent epidemiological research has focused on effects of short-term exposures, several studies suggest that long-term exposure may be more important in terms of overall public health.[4] The new standards for long-term exposure to $PM_{2.5}$ were originally based primarily on 2 prospective cohort studies,[15,16] which evaluated the effects of long-term pollution exposure on mortality. Both of these studies have been subjected to much scrutiny,[5] including an extensive independent audit and reanalysis of the original data.[17] The larger of these 2 studies linked individual risk factor and vital status data with national ambient air pollution data.[16] Our analysis uses data from the larger study and

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(1) doubles the follow-up time to more than 16 years and triples the number of deaths; (2) substantially expands exposure data, including gaseous copollutant data and new $PM_{2.5}$ data, which have been collected since the promulgation of the new air quality standards; (3) improves control of occupational exposures; (4) incorporates dietary variables that account for total fat consumption, and consumption of vegetables, citrus, and high-fiber grains; and (5) uses recent advances in statistical modeling, including the incorporation of random effects and nonparametric spatial smoothing components in the Cox proportional hazards model.

## METHODS

### Study Population

The analysis is based on data collected by the American Cancer Society (ACS) as part of the Cancer Prevention Study II (CPS-II), an ongoing prospective mortality study of approximately 1.2 million adults.[18,19] Individual participants were enrolled by ACS volunteers in the fall of 1982. Participants resided in all 50 states, the District of Columbia, and Puerto Rico, and were generally friends, neighbors, or acquaintances of ACS volunteers. Enrollment was restricted to persons who were aged 30 years or older and who were members of households with at least 1 individual aged 45 years or older. Participants completed a confidential questionnaire, which included questions about age, sex, weight, height, smoking history, alcohol use, occupational exposures, diet, education, marital status, and other characteristics.

Vital status of study participants was ascertained by ACS volunteers in September of the following years: 1984, 1986, and 1988. Reported deaths were verified with death certificates. Subsequently, through December 31, 1998, vital status was ascertained through automated linkage of the CPS-II study population with the National Death Index.[19] Ascertainment of deaths was more than 98% complete for the period of 1982-1988 and 93% complete after 1988.[19] Death certificates or codes for cause of death were obtained for more than 98% of all known deaths. Cause of death was coded according to the *International Classification of Diseases, Ninth Revision (ICD-9)*. Although the CPS-II cohort included approximately 1.2 million participants with adequate questionnaire and cause-of-death data, our analysis was restricted to those participants who resided in US metropolitan areas with available pollution data. The actual size of the analytic cohort varied depending on the number of metropolitan areas for which pollution data were available. TABLE 1 provides the number of metropolitan areas and participants available for each source of pollution data.

### Air Pollution Exposure Estimates

Each participant was assigned a metropolitan area of residence based on address at time of enrollment and 3-digit ZIP code area.[20] Mean (SD) concentrations of air pollution for the metropolitan areas were compiled from various primary data sources (Table 1). Many of the particulate pollution indices, including $PM_{2.5}$, were available from data from the Inhalable Particle Monitoring Network for 1979-1983 and data from the National Aerometric Database for 1980-1981, periods just prior to or at the beginning of the follow-up period. An additional data source was the Environmental Protection Agency Aerometric Information Retrieval System (AIRS). The mean concentration of each pollutant from all available monitoring sites was calculated for each metropolitan area during the 1 to 2 years prior to enrollment.[17]

Additional information on ambient pollution during the follow-up period was extracted from the AIRS database as quarterly mean values for each routinely monitored pollutant for 1982 through 1998. All quarterly averages met summary criteria imposed by the Environmental Protection Agency and were based on observations made on at least 50% of the scheduled sampling days at each site. The quarterly mean values for all stations in each metropolitan area were calculated across the study years using daily average values for each pollutant except ozone. For ozone, daily 1-hour maximums were used and were calculated for the full year and for the third quarter only (ie, July, August, September). While gaseous pollutants generally had recorded data throughout the entire follow-up period of interest, the particulate matter monitoring protocol changed in the late 1980s from total suspended particles to particles measuring less than 10 μm in diameter ($PM_{10}$), resulting in the majority of total suspended particle data being available in the early to mid-1980s and $PM_{10}$ data being mostly available in the early to mid-1990s.

As a consequence of the new $PM_{2.5}$ standard, a large number of sites began collecting $PM_{2.5}$ data in 1999. Daily $PM_{2.5}$ data were extracted from the AIRS database for 1999 and the first 3 quarters of 2000. For each site, quarterly averages for each of the 2 years were computed. The 4 quarters were averaged when at least 1 of the 2 corresponding quarters for each year had at least 50% of the sixth-day samples and at least 45 total sampling days available. Measurements were averaged first by site and then by metropolitan area. Although no network of $PM_{2.5}$ monitoring existed in the United States between the early 1980s and the late 1990s, the integrated average of $PM_{2.5}$ concentrations during the period was estimated by averaging the $PM_{2.5}$ concentration for early and later periods.

Mean sulfate concentrations for 1980-1981 were available for many cities based on data from the Inhalable Particle Monitoring Network and the National Aerometric Database. Recognizing that sulfate was artifactually overestimated due to glass fiber filters used at that time, season and region-specific adjustments were made.[17] Since few states analyzed particulate samples for sulfates after the early 1980s, individual states were directly contacted for data regarding filter use. Ion chromatography was used to analyze $PM_{10}$ filters and this data could be obtained from metropolitan areas across the

United States. Filters were collected for a single reference year (1990) in the middle of the 1982-1998 study period. The use of quartz filters virtually eliminated the historical overestimation of sulfate. Mean sulfate concentrations for 1990 were estimated using sulfate from AIRS, data reported directly from individual states, and analysis of archived filters.

### Statistical Analysis

The basic statistical approach used in this analysis is an extension of the standard Cox proportional hazards survival model,[21] which has been used for risk estimates of pollution-related mortality in previous longitudinal cohort studies.[15,16] The standard Cox model implicitly assumes that observations are statistically independent after controlling for available risk factors, resulting in 2 concerns with regard to risk estimates of pollution-related mortality.[22] First, if the assumption of statistical independence is not valid, the uncertainty in the risk estimates of pollution-related mortality may be misstated. Second, even after controlling for available risk factors, survival times of participants living in communities closer together may be more similar than participants living in communities farther apart, which results in spatial autocorrelation. If this spatial autocorrelation is due to missing or systematically mismeasured risk factors that are spatially correlated with air pollution, then the risk estimates of pollution-related mortality may be biased due to inadequate control of these factors. Therefore, in this analysis, the Cox proportional hazards model was extended by incorporating a spatial random-effects component, which provided accurate es-

**Table 1.** Summary of Alternative Pollution Indices*

| Pollutant (Years of Data Collection) | Units | Source of Data | Data Compilation Team† | No. of Metropolitan Areas | No. of Participants, in Thousands | Mean (SD) |
|---|---|---|---|---|---|---|
| $PM_{2.5}$ | µg/m³ | | | | | |
| 1979-1983 | | IPMN | HEI | 61 | 359 | 21.1 (4.6) |
| 1999-2000 | | AIRS | NYU | 116 | 500 | 14.0 (3.0) |
| Average | | | | 51 | 319 | 17.7 (3.7) |
| $PM_{10}$ | µg/m³ | | | | | |
| 1982-1998 | | AIRS | NYU | 102 | 415 | 28.8 (5.9) |
| $PM_{15}$ | µg/m³ | | | | | |
| 1979-1983 | | IPMN | HEI | 63 | 359 | 40.3 (7.7) |
| $PM_{15\text{-}2.5}$ | µg/m³ | | | | | |
| 1979-1983 | | IPMN | HEI | 63 | 359 | 19.2 (6.1) |
| Total suspended particles | µg/m³ | | | | | |
| 1980-1981 | | NAD | HEI | 156 | 590 | 68.0 (16.7) |
| 1979-1983 | | IPMN | HEI | 58 | 351 | 73.7 (14.3) |
| 1982-1998 | | AIRS | NYU | 150 | 573 | 56.7 (13.1) |
| Sulfate | µg/m³ | | | | | |
| 1980-1981 | | IPMN and NAD, artifact adjusted | HEI | 149 | 572 | 6.5 (2.8) |
| 1990 | | Compilation and analysis of $PM_{10}$ filters | NYU | 53 | 269 | 6.2 (2.0) |
| Sulfur dioxide | ppb | AIRS | | | | |
| 1980 | | | HEI | 118 | 520 | 9.7 (4.9) |
| 1982-1998 | | | NYU | 126 | 539 | 6.7 (3.0) |
| Nitrogen dioxide | ppb | AIRS | | | | |
| 1980 | | | HEI | 78 | 409 | 27.9 (9.2) |
| 1982-1998 | | | NYU | 101 | 493 | 21.4 (7.1) |
| Carbon monoxide | ppm | AIRS | | | | |
| 1980 | | | HEI | 113 | 519 | 1.7 (0.7) |
| 1982-1998 | | | NYU | 122 | 536 | 1.1 (0.4) |
| Ozone | ppb | AIRS | | | | |
| 1980 | | | HEI | 134 | 569 | 47.9 (11.0) |
| 1982-1998 | | | NYU | 119 | 525 | 45.5 (7.3) |
| 1982-1998‡ | | | NYU | 134 | 557 | 59.7 (12.8) |

*$PM_{2.5}$ indicates particles measuring less than 2.5 µm in diameter; $PM_{10}$, particles measuring less than 10 µm in diameter; $PM_{15}$, particles measuring less than 15 µm in diameter; $PM_{15\text{-}2.5}$, particles measuring between 2.5 and 15 µm in diameter; µg/m³, micrograms per cubic meter; ppb, parts per billion; ppm, parts per million; IPMN, Inhalable Particle Monitoring Network; AIRS, Aerometric Information Retrieval System [Environmental Protection Agency]; and NAD, National Aerometric Database.
†HEI indicates data were compiled by the Health Effects Institute reanalysis team, which was previously published.[17] NYU indicates data were compiled at the New York University School of Medicine, Nelson Institute of Environmental Medicine (K.I. and G.D.T.).
‡Daily 1-hour maximums were used. Values were calculated only for the third quarter (ie, July, August, September).

timates of the uncertainty of effect estimates. The model also evaluated spatial autocorrelation and incorporated a nonparametric spatial smooth component (to account for unexplained spatial structure). A more detailed description of this modeling approach is provided elsewhere.[22]

The baseline analysis in this study estimated adjusted relative risk (RR) ratios for mortality by using a Cox proportional hazards model with inclusion of a metropolitan-based random-effects component. Model fitting involved a 2-stage process. In the first stage, survival data were modeled using the standard Cox proportional hazards model, including individual level covariates and indicator variables for each metropolitan area (without pollution variables). Output from stage 1 provided estimates of the metropolitan-specific logarithm of the RRs of mortality (relative to an arbitrary reference community), which were adjusted for individual risk factors. The correlation between these values, which was induced by using the same reference community, was then removed.[23] In the second stage, the estimates of adjusted metropolitan-specific health responses were related to fine particulate air pollution using a linear random-effects regression model.[24] The time variable used in the models was survival time from the date of enrollment. Survival times of participants who did not die were censored at the end of the study period. To control for age, sex, and race, all of the models were stratified by 1-year age categories, sex, and race (white vs other), which allowed each category to have its own baseline hazard. Models were estimated for all-cause mortality and for 3 separate mortality categories: cardiopulmonary (*ICD*-9 401-440 and 460-519), lung cancer (*ICD*-9 162), and all others.

Models were estimated separately for each of the 3 fine particle variables, $PM_{2.5}$ (1979-1983), $PM_{2.5}$ (1999-2000), and $PM_{2.5}$ (average). Individual level covariates were included in the models to adjust for various important individual risk factors. All of these variables were classified as either indicator (ie, yes/no, binary, dummy) variables or continuous variables. Variables used to control for tobacco smoke, for example, included both indicator and continuous variables. The smoking indicator variables included: current cigarette smoker, former cigarette smoker, and a pipe or cigar smoker only (all vs never smoking) along with indicator variables for starting smoking before or after age 18 years. The continuous smoking variables included: current smoker's years of smoking, current smoker's years of smoking squared, current smoker's cigarettes per day, current smoker's cigarettes per day squared, former smoker's years of smoking, former smoker's years of smoking squared, former smoker's cigarettes per day, former smoker's cigarettes per day squared, and the number of hours per day exposed to passive cigarette smoke.

To control for education, 2 indicator variables, which indicated completion of high school or education beyond high school, were included. Marital status variables included indicator variables for single and other vs married. Both body mass index (BMI) values and BMI values squared were included as continuous variables. Indicator variables for beer, liquor, and wine drinkers and nonresponders vs nondrinkers were included to adjust for alcohol consumption. Occupational exposure was controlled for using various indicator variables: regular occupational exposure to asbestos, chemicals/acids/solvents, coal or stone dusts, coal tar/pitch/asphalt, diesel engine exhaust, or formaldehyde, and additional indicator variables that indicated 9 different rankings of an occupational dirtiness index that has been developed and described elsewhere.[17,25] Two diet indices that accounted for fat consumption and consumption of vegetables, citrus, and high-fiber grains were derived based on information given in the enrollment questionnaire.[18] Quintile indicator variables for each of these diet indices were also included in the models.[18]

In addition to the baseline analysis, several additional sets of analysis were conducted. First, to more fully evaluate the shape of the concentration-response function, a robust locally weighted regression smoother[26] (within the generalized additive model framework[27]) was used to estimate the relationship between particulate air pollution and mortality in the second stage of model fitting. Second, the sensitivity of the fine particle mortality risk estimates compared with alternative modeling approaches and assumptions was evaluated. Standard Cox proportional hazards models were fit to the data including particulate air pollution as a predictor of mortality and sequentially adding (in a controlled forward stepwise process) groups of variables to control for smoking, education, marital status, BMI, alcohol consumption, occupational exposures, and diet.

In addition, to evaluate the sensitivity of the estimated pollution effect while more aggressively controlling for spatial differences in mortality, a 2-dimensional term to account for spatial trends was added to the models and was estimated using a locally weighted regression smoother. The "span" parameter, which controls the complexity of the surface smooth, was set at 3 different settings to allow for increasingly aggressive fitting of the spatial structure. These included a default span of 50%, the span that resulted in the lowest unexplained variance in mortality rate between metropolitan areas, and the span that resulted in the strongest evidence (highest $P$ value) to suggest no residual spatial structure. The risk estimates and SEs (and thus the confidence intervals) were estimated using generalized additive modeling[27] with S-Plus statistical software,[28] which provides unbiased effect estimates, but may underestimate SEs if there is significant spatial autocorrelation and significant correlations between air pollution and the smoothed surface of mortality. Therefore, evidence of spatial autocorrelation was carefully evaluated and tested using the Bartlett test.[29] The correlations of residual mortality

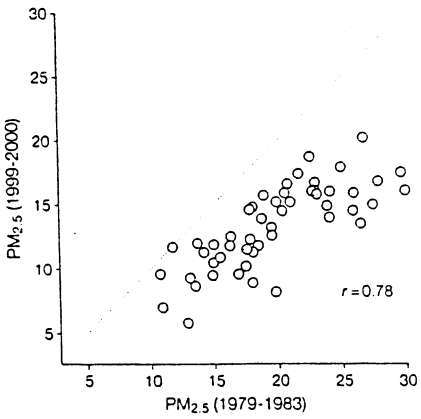
with distance between metropolitan areas were graphically examined.

Analyses were also conducted of effect modification by age, sex, smoking status, occupational exposure, and education. Finally, models were fit using a variety of alternative pollution indices, including gaseous pollutants. Specifically, models were estimated separately for each of the pollution variables listed in Table 1, while also including all of the other risk factor variables.

## RESULTS

Fine particulate air pollution generally declined in the United States during the follow-up period of this study. FIGURE 1 plots mean $PM_{2.5}$ concentrations for 1999-2000 over mean $PM_{2.5}$ concentrations for 1979-1983 for the 51 cities in which paired data were available. The concentrations of $PM_{2.5}$ were lower in 1999-2000 than in 1979-1983 for most cities, with the largest reduction observed in the cities with the highest concentrations of pollution during 1979-1983. Mean $PM_{2.5}$ levels in the 2 periods were highly correlated ($r=0.78$). The rank ordering of cities by relative pollution levels remained nearly the same. Therefore, the relative levels of fine particle concentrations were similar whether based on measurements at the beginning of the study period, shortly following the study period, or an average of the 2.

**Figure 1.** Mean Fine Particles Measuring Less Than 2.5 μm in Diameter ($PM_{2.5}$)




Mean $PM_{2.5}$ concentrations in micrograms per meters cubed for 1999-2000 are plotted along with concentrations for 1979-1983 for the 51 metropolitan areas with paired pollution data. The dotted line is a reference 45°-equality line.

As reported in TABLE 2, all 3 indices of fine particulate air pollution were associated with all-cause, cardiopulmonary, and lung cancer mortality, but not mortality from all other causes combined. FIGURE 2 presents the nonparametric smoothed exposure response relationships between cause-specific mortality and $PM_{2.5}$ (average). The log RRs for all-cause, cardiopulmonary, and lung cancer mortality increased across the gradient of fine particulate matter. Goodness-of-fit tests indicated that the associations were not significantly different from linear associations ($P>.20$).

The fine particle mortality RR ratios from various alternative modeling approaches and assumptions are presented in FIGURE 3. After controlling for smoking, education, and marital status, the controlled forward stepwise inclusion of additional covariates had little influence on the estimated associations with fine particulate air pollution on cardiopulmonary and lung cancer mortality. As expected, cigarette smoking was highly significantly associated with elevated risk of all-cause, cardiopulmonary, and lung cancer mortality ($P<.001$). Estimated RRs for an average current smoker (men and women combined, 22 cigarettes/day for 33.5 years, with initiation before age 18 years) were equal to 2.58, 2.89, and 14.80 for all-cause, cardiopulmonary, and lung cancer mortality, respectively. Statistically significant, but substantially smaller and less robust associations, were also observed for education, marital status, BMI, alcohol consumption, occupational exposure, and diet variables. Although many of these covariates were also statistically associated with mortality, the risk estimates of pollution-related mortality were not highly sensitive to the inclusion of these additional covariates.

Figure 3 also demonstrates that the introduction of the random-effects component to the model resulted in larger SEs of the estimates and, therefore, somewhat wider 95% confidence intervals. There was no evidence of statistically significant spatial autocorrelation in the survival data based on the Bartlett test ($P>.20$) after controlling for fine particulate air pollution and the various individual risk factors. Furthermore, graphical examination of the correlations of the residual mortality with distance between metropolitan areas did not reveal significant spatial autocorrelation (results not shown). Nevertheless, the incorporation of spatial smoothing was included to further investigate the robustness of the estimated particulate pollution effect. Effect estimates were not highly sensitive to the incorporation of spatial smoothing to account for regional clustering or other spatial patterns in the data.

FIGURE 4 presents fine particle air pollution–related mortality RR ratios after stratifying by age, sex, education, and smoking status, and adjusting for all other risk factors. The differences across age and sex strata were not generally consistent or statistically significant. However, a consistent pattern emerged from this stratified analysis: the association with particulate pollution was stronger for both cardiopulmo-

**Table 2.** Adjusted Mortality Relative Risk (RR) Associated With a 10-μg/m³ Change in Fine Particles Measuring Less Than 2.5 μm in Diameter

| | Adjusted RR (95% CI)* | | |
|---|---|---|---|
| Cause of Mortality | 1979-1983 | 1999-2000 | Average |
| All-cause | 1.04 (1.01-1.08) | 1.06 (1.02-1.10) | 1.06 (1.02-1.11) |
| Cardiopulmonary | 1.06 (1.02-1.10) | 1.08 (1.02-1.14) | 1.09 (1.03-1.16) |
| Lung cancer | 1.08 (1.01-1.16) | 1.13 (1.04-1.22) | 1.14 (1.04-1.23) |
| All other cause | 1.01 (0.97-1.05) | 1.01 (0.97-1.06) | 1.01 (0.95-1.06) |

*Estimated and adjusted based on the baseline random-effects Cox proportional hazards model, controlling for age, sex, race, smoking, education, marital status, body mass, alcohol consumption, occupational exposure, and diet. CI indicates confidence interval.

nary and lung cancer mortality for participants with less education. Also, for both cardiopulmonary and lung cancer mortality, the RR estimates were higher for nonsmokers.

FIGURE 5 summarizes the associations between mortality risk and air pollutant concentrations listed in Table 1. Statistically significant and relatively consistent mortality associations existed for all measures of fine particulate exposure, including $PM_{2.5}$ and sulfate particles. Weaker less consistent mortality associations were observed with $PM_{10}$ and $PM_{15}$. Measures of the coarse particle fraction ($PM_{15-2.5}$) and total suspended particles were not consistently associated with mortality. Of the gaseous pollutants, only sulfur dioxide was associated with elevated mortality risk. Interestingly, measures of $PM_{2.5}$ were associated with all-cause cardiopulmonary, and lung cancer mortality, but not with all other mortality. However, sulfur oxide pollution (as measured by sulfate particles and/or sulfur dioxide) was significantly associated with mortality from all other causes in addition to all-cause, cardiopulmonary, and lung cancer mortality.

## COMMENT

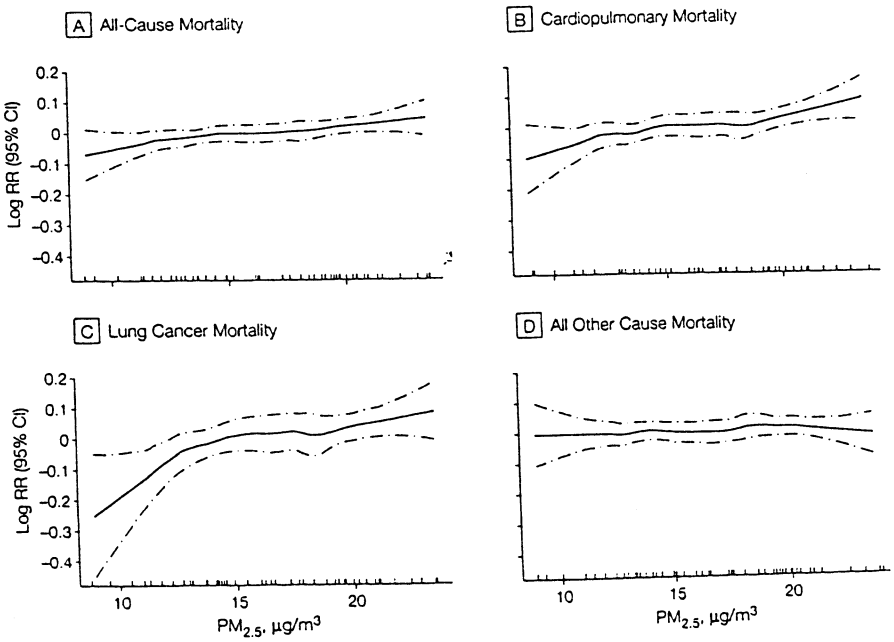
This study demonstrated associations between ambient fine particulate air pollution and elevated risks of both cardiopulmonary and lung cancer mortality. Each 10-μg/m$^3$ elevation in long-term average $PM_{2.5}$ ambient concentrations was associated with approximately a 4%, 6%, and 8% increased risk of all-cause, cardiopulmonary, and lung cancer mortality, respectively, although the magnitude of the effect somewhat depended on the time frame of pollution monitoring. In addition, this analysis addresses many of the important questions concerning the earlier, more limited analysis of the large CPS-II cohort, including the following issues.

First, does the apparent association between pollution and mortality persist with longer follow-up and as the cohort ages and dies? The present analysis more than doubled the follow-up time to more than 16 years, resulting in approximately triple the number of deaths, yet the associations between pollution and mortality persisted.

Second, can the association between fine particulate air pollution and increased cardiopulmonary and lung cancer mortality be due to inadequate control of important individual risk factors? After aggressively controlling for smoking, the estimated fine particulate pollution effect on mortality was remarkably robust. When the analysis was stratified by smoking status, the estimated pollution effect on both cardiopulmonary and lung cancer mortality was strongest for never smokers vs former or current smokers. This analysis also controlled for education, marital status, BMI, and alcohol consumption. This analysis used improved variables to control for occupational exposures and incorporated diet variables that accounted for total fat consumption, as well as for consumption of vegetables, citrus, and high-fiber grains. The mortality associations with fine particulate air pollution were largely unaffected by the inclusion of these individual risk factors in the models. The data on smoking and other individual risk factors, however, were obtained directly by questionnaire at time of enrollment and do not reflect changes that may have occurred following enrollment. The lack of risk factor follow-up data results in some misclassification of exposure, reduces the precision of control for risk factors, and constrains our ability to differentiate time dependency.

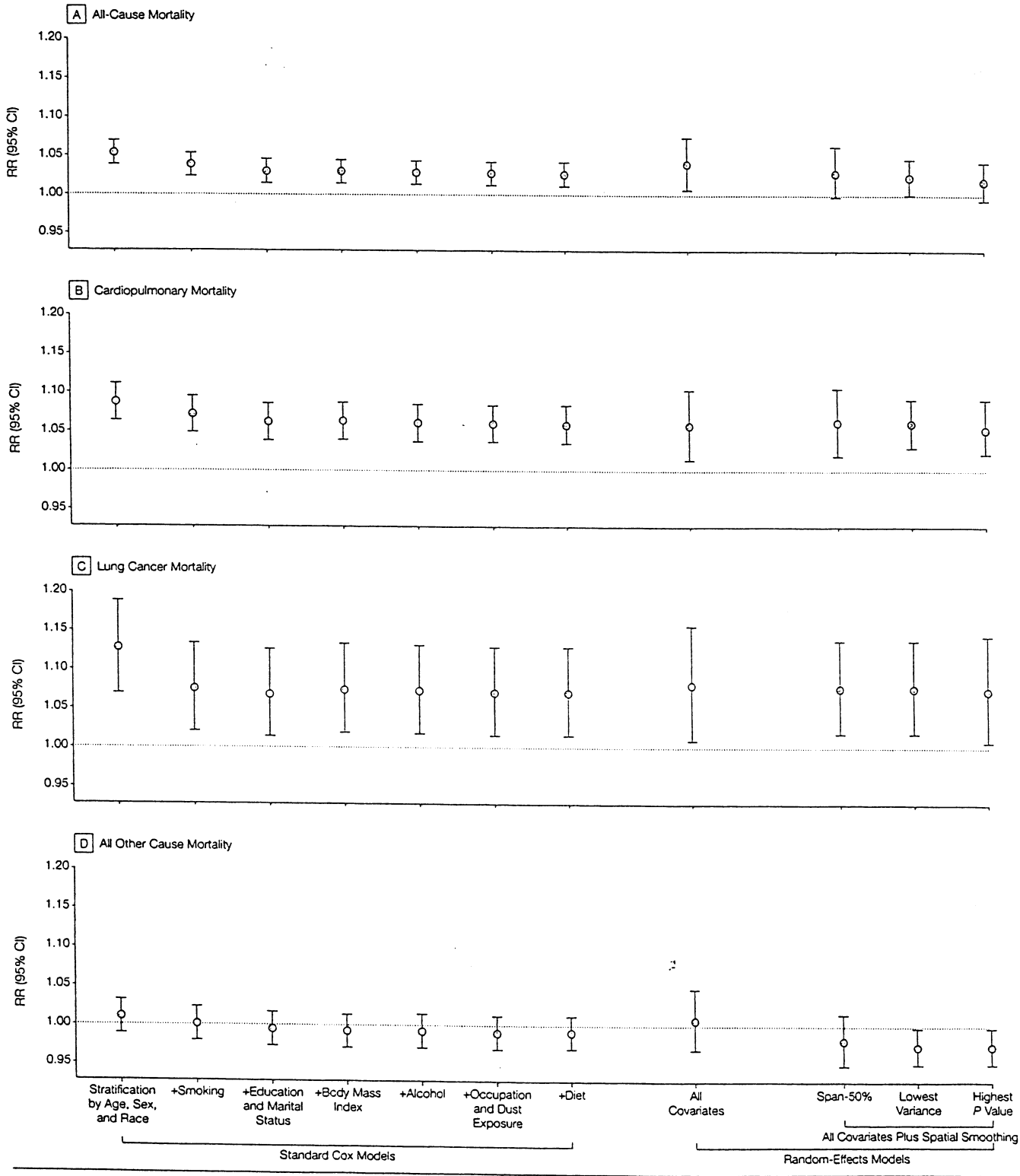
Third, are the associations between fine particulate air pollution and mortality due to regional or other spatial differences that are not adequately controlled for in the analysis? If there are unmeasured or inadequately modeled risk factors that are different across locations, then spatial clustering will occur. If this clustering is independent or random across metropolitan areas, then the spatial clustering can be modeled by adding a random-effects component to the Cox proportional hazards model as was done in our analysis. The clustering may not be independent or random across metropolitan areas due to inadequately measured or modeled

**Figure 2.** Nonparametric Smoothed Exposure Response Relationship




Vertical lines along x-axes indicate rug or frequency plot of mean fine particulate pollution; $PM_{2.5}$, mean fine particles measuring less than 2.5 μm in diameter; RR, relative risk; and CI, confidence interval.

**Figure 3.** Mortality Relative Risk (RR) Ratio Associated With 10-μg/m³ Differences of $PM_{2.5}$ Concentrations




Data presented are for 1979-1983 for the different causes of death, with various levels of controlling for individual risk factors, and using alternative modeling approaches. The 3 models with spatial smoothing allow for increasingly aggressive fitting of the spatial structure. Plus sign indicates model included previous variables (ie, smoking included stratification by age, sex, and race); $PM_{2.5}$, mean fine particles measuring less than 2.5 μm in diameter; and CI, confidence interval.

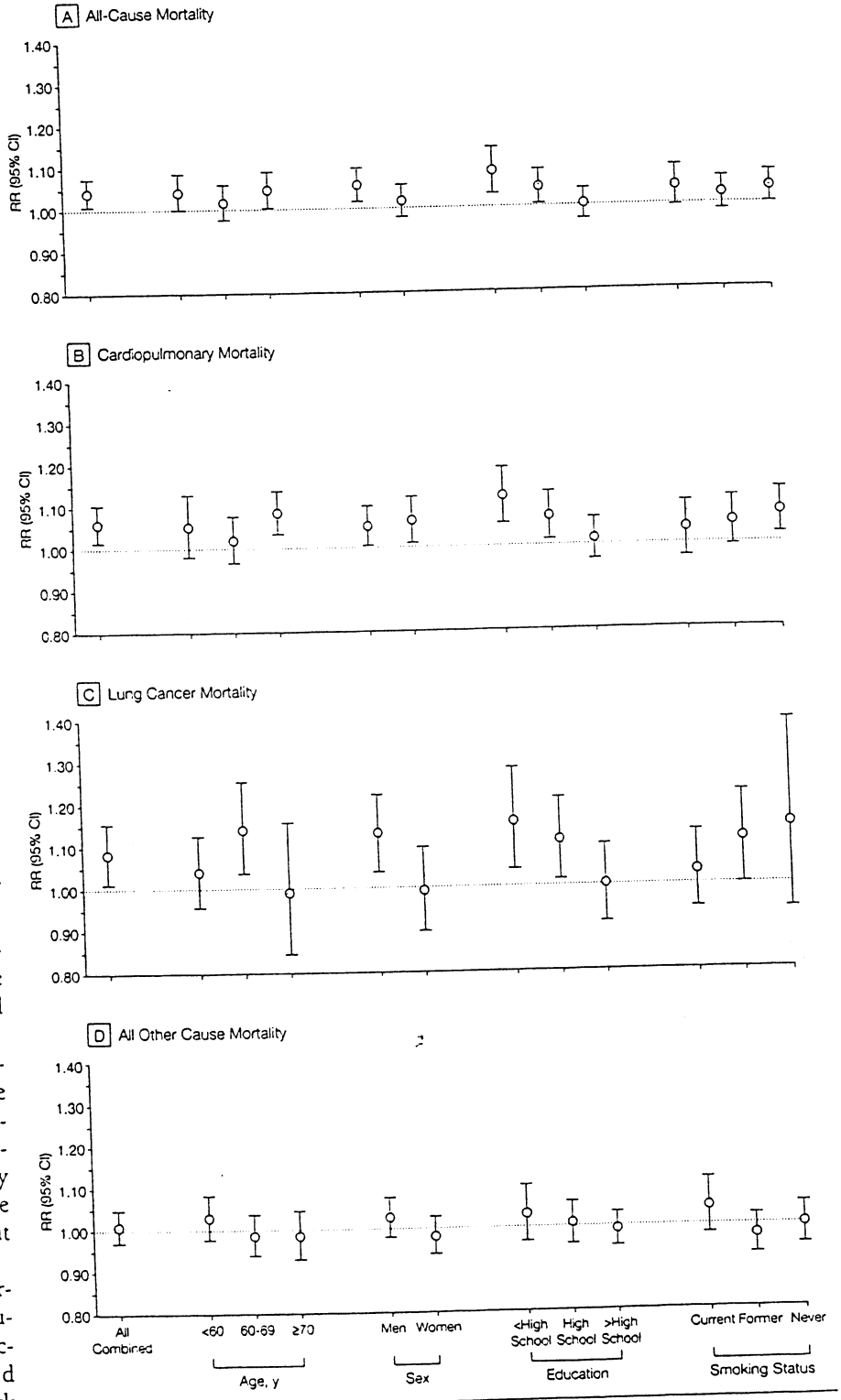
risk factors (either individual or ecological). If these inadequately measured or modeled risk factors are also spatially correlated with air pollution, then biased pollution effects estimates may occur due to confounding. However, in this analysis, significant spatial autocorrelation was not observed after controlling for fine particulate air pollution and the various individual risk factors. Furthermore, to minimize any potential confounding bias, sensitivity analyses, which directly modeled spatial trends using nonparametric smoothing techniques, were conducted. A contribution of this analysis is that it included the incorporation of both random effects and nonparametric spatial smoothing components to the Cox proportional hazards model. Even after accounting for random effects across metropolitan areas and aggressively modeling a spatial structure that accounts for regional differences, the association between fine particulate air pollution and cardiopulmonary and lung cancer mortality persists.

Fourth, is mortality associated primarily with fine particulate air pollution or is mortality also associated with other measures of particulate air pollution, such as $PM_{10}$, total suspended particles, or with various gaseous pollutants? Elevated mortality risks were associated primarily with measures of fine particulate and sulfur oxide pollution. Coarse particles and gaseous pollutants, except for sulfur dioxide, were generally not significantly associated with elevated mortality risk.

Fifth, what is the shape of the concentration-response function? Within the range of pollution observed in this analysis, the concentration-response function appears to be monotonic and nearly linear. However, this does not preclude a leveling off (or even steepening) at much higher levels of air pollution.

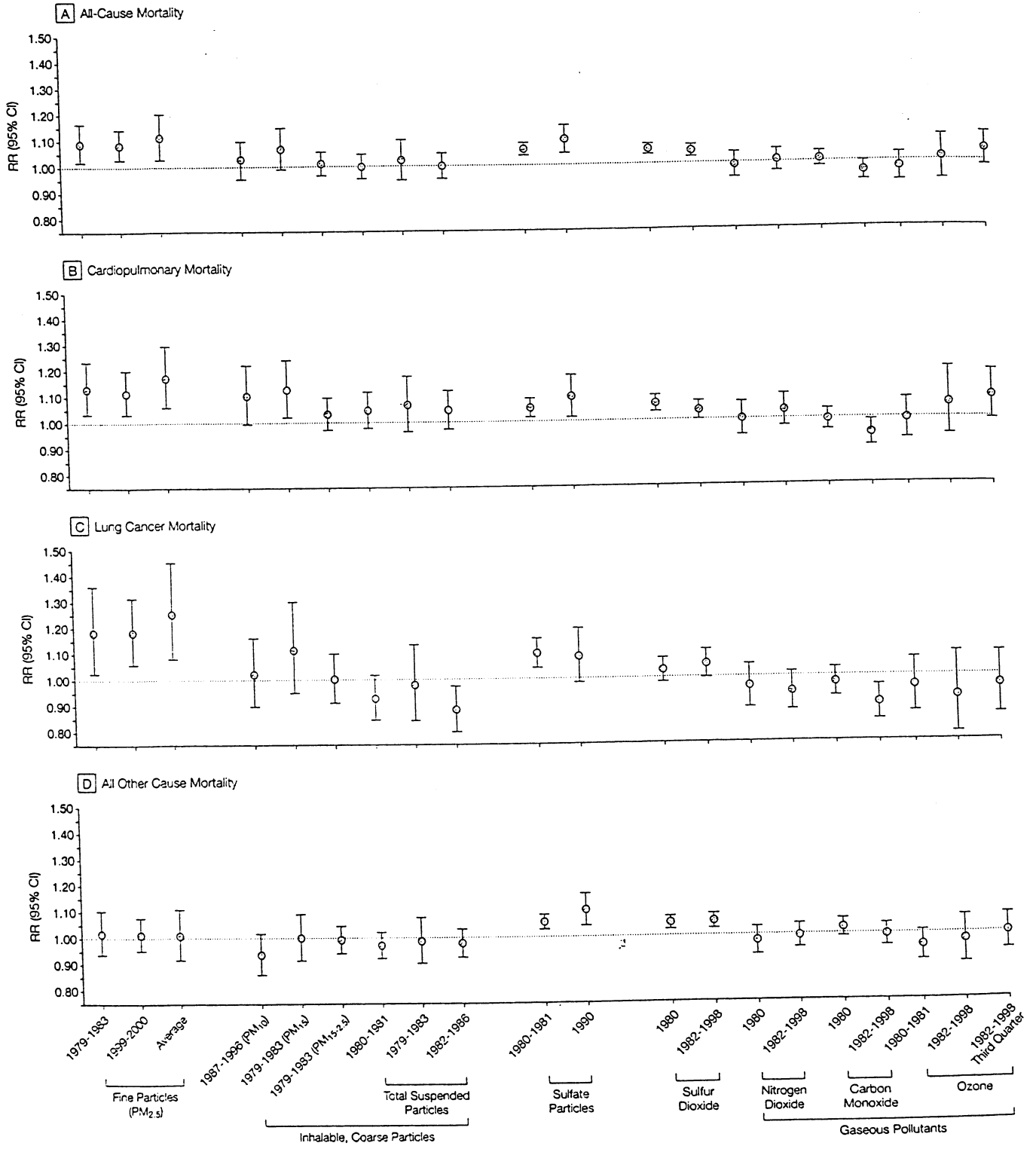
Sixth, how large is the estimated mortality effect of exposure to fine particulate air pollution relative to other risk factors? A detailed description and interpretation of the many individual risk factors that are controlled for in the analysis goes well beyond the scope of

**Figure 4.** Adjusted Mortality Relative Risk (RR) Ratio Associated With 10-µg/m³ Differences of $PM_{2.5}$ Concentrations




Data presented are for 1979-1983 for the different causes of death stratified by age, sex, education, and smoking status. $PM_{2.5}$ indicates mean fine particles measuring less than 2.5 µm in diameter; CI, confidence interval.

**Figure 5.** Adjusted Mortality Relative Risk (RR) Ratio Evaluated at Subject-Weighted Mean Concentrations




$PM_{2.5}$ indicates particles measuring less than 2.5 μm in diameter; $PM_{10}$, particles measuring less than 10 μm in diameter; $PM_{15}$, particles measuring less than 15 μm in diameter; $PM_{15-2.5}$, particles measuring between 2.5 and 15 μm in diameter; and CI, confidence interval.

this report. However, the mortality risk associated with cigarette smoking has been well documented using the CPS-II cohort.[16] The risk imposed by exposure to fine particulate air pollution is obviously much smaller than the risk of cigarette smoking. Another risk factor that has been well documented using the CPS-II cohort data is body mass as measured by BMI.[30] The Word Health Organization has categorized BMI values between 18.5-24.9 kg/m$^2$ as normal; 25-29.9 kg/m$^2$, grade 1 overweight; 30-39.9 kg/m$^2$, grade 2 overweight; and 40 kg/m$^2$ or higher, grade 3 overweight.[31] In the present analysis, BMI values and BMI values squared were included in the proportional hazards models. Consistent with previous ACS analysis,[30] BMI was significantly associated with mortality, optimal BMI was between approximately 23.5 and 24.9 kg/m$^2$, and the RR of mortality for different BMI values relative to the optimal were dependent on sex and smoking status. For example, the RRs associated with BMI values between 30.0 and 31.9 kg/m$^2$ (vs optimal) would be up to approximately 1.33 for never smokers. Based on these calculations, mortality risks associated with fine particulate air pollution at levels found in more polluted US metropolitan areas are less than those associated with substantial obesity (grade 3 overweight), but comparable with the estimated effect of being moderately overweight (grade 1 to 2).

In conclusion, the findings of this study provide the strongest evidence to date that long-term exposure to fine particulate air pollution common to many metropolitan areas is an important risk factor for cardiopulmonary mortality. In addition, the large cohort and extended follow-up have provided an unprecedented opportunity to evaluate associations between air pollution and lung cancer mortality. Elevated fine particulate air pollution exposures were associated with significant increases in lung cancer mortality. Although potential effects of other unaccounted for factors cannot be excluded with certainty, the associations between fine particulate air pollution and lung cancer mortality, as well as cardiopulmonary mortality, are observed even after controlling for cigarette smoking, BMI, diet, occupational exposure, other individual risk factors, and after controlling for regional and other spatial differences.

**Author Contributions:** *Study concept and design:* Pope, Burnett, Krewski, Thurston.
*Acquisition of data:* Thun, Calle, Krewski, Ito, Thurston.
*Analysis and interpretation of data:* Pope, Burnett, Krewski, Thurston.
*Drafting of the manuscript:* Pope, Burnett, Ito, Thurston.
*Critical revision of the manuscript for important intellectual content:* Pope, Thun, Calle, Krewski, Thurston.
*Statistical expertise:* Pope, Burnett, Krewski.
*Obtained funding:* Pope, Thun, Thurston.
*Administrative, technical, or material support:* Pope, Calle, Krewski, Ito, Thurston.
*Study supervision:* Pope, Krewski.
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February 28, 2003

Ms. Elizabeth Chimento
1200 N. Pitt Street
Alexandria, VA 22314

Dear Ms. Chimento,

Regarding the four samples that you shipped to Dr. Mark Badger in June of 2002 (**Three Dust Samples and a Coal**), I have finally completed an evaluation of these materials using reflected light microscopy and a final report is attached. If there are questions or comments please call me directly at (814) 865-6543.

Thank you.

Sincerely,

Gareth Mitchell
Research Associate

Enclosure: Report

# A PETROGRAPHIC EVALUATION

of

**Three Dust Samples and a Coal**

for

Ms. Elizabeth Chimento
1200 N. Pitt Street
Alexandria, VA 22314

by

The Coal & Organic Petrology Laboratories
The Energy Institute
The Pennsylvania State University
104 Academic Projects Bldg
University Park, PA 16802

February 28, 2003

# PETROGRAPHIC EVALUATION

of

## Three Dust Samples and a Coal


Executive Summary

Three samples of dust accumulated over a period prior to June 25th, 2002 from different locations around Ms. Chimento's residence (1200 North Pitt Street, Alexandria, VA 22314) were evaluated using reflected-light optical microscopy to establish the nature, relative composition and source of the dust. Ms. Chimento's residence is located near the Merant Plant, Potomac Generating Station in Alexandria, Virginia and it is of some interest to know whether the dust may be coming from the plant or elsewhere. Because the Merant Plant is coal-fired a sample of coal from along the rail road tracks leading to the plant was obtained for comparison with any coal or coal-derived materials found in the dust samples.

Basically dust samples were composed of <100 μm diameter rock or mineral fragments (agglomerates) and coal particles, as well as minor amounts of fly-ash, living plant tissues and unidentifiable materials. Although the presence of coal in the dust samples was unexpected, accumulation occurred about equally from the front and back and 1st and 2nd floor of the residence. Furthermore, evaluation of the reflected light properties and relative composition of coal particles found in all of the dust samples corresponded exactly to the spillage coal found along the rail road tracks servicing the Merant Plant. Consequently, if there are no other sources using coal in the area and unit trains of coal do not past through the area to other locations, then it is clear that a significant amount of the dust accumulated on the window ledges and deck of Ms. Chimento's residence last June came from operations at the Merant Plant. An exact source of coal dust from plant operations can not be determined, but may include rail road car tops, dumping operations, stock piling and reclaiming, transportation, crushing operations or all of the above.

From my understanding, these findings represent a departure from past thinking which was directed primarily on stack emissions (fly-ash) as the root cause of dust accumulation instead of a raw materials handling source. Some fly-ash materials were found in all dust samples, but an insignificant amount compared to coal dust. Although it was not possible to determine the rate of dust accumulation or potential losses of coal from the plant, it may be of interest to personnel at the Merant Plant both from a financial point of view (valuable coal is being lost) as well as a service to the community.

## Introduction

From conversations with Ms. Chimento and Dr. Mark Badger, it was my understanding that the neighborhood very near to the Merant Plant, Potomac Generating Station in Alexandria, Virginia suffers from some degree of dust accumulation. The question of interest to Ms. Climento was whether the dust may be coming from the coal-fired power plant? Arrangements were made to obtain samples of freshly accumulated dust from the neighborhood, as well as a coal sample from along the rail road tracks leading to the plant. The coal sample may be helpful in determining the type of carbonaceous fly-ash particles that could be expected from plant operations.

Four samples were provided to Dr. Badger in June of 2002 and were turned over to Gareth Mitchell in July of 2002 for petrologic evaluation. Petrology is concerned with the origin, occurrence, structure and history of rocks or materials and is a special technique applied by the Coal & Organic Petrology Laboratories. In this investigation, reflected-light optical microscopy was employed to determine the nature and content of the material comprising three dust samples and a coal spillage sample from along the rail road tracks that services the Merant Plant.

## Procedures

Dust samples were collected during a period prior to June 25$^{th}$, 2002 from three locations at Ms. Climento's residence, including samples #1) a second floor rear window ledge, #2) the back deck (2$^{nd}$ floor) and #3) the first floor window ledge (street side). Over an undetermined amount of time dust was allowed to accumulate on waxed paper at these locations, then the samples were carefully wrapped, placed in individual plastic bags and shipped as collected. A fourth sample consisting of six, ~25 mm diameter particles of coal were obtained from rail road tracks beside the Merant Plant.

When the waxed paper containing the dust was unfolded, very small particles of a black material sparsely covered the surface. Approximately less than 0.1 mg of sample was retrieved from dust samples #1 and #2 and slightly more from dust sample #3. Particles from each paper were lightly scrapped or tapped into a 1.0 mL flat-bottom plastic vial (with lid). A small amount of a cold-setting epoxy resin was dripped into each tube; particles were wetted with epoxy, placed in a vacuum to force out air bubbles and spun in a centrifuge to concentrate the particles at the bottom of each tube. After hardening, the plastic tube was removed and the epoxy cylinder was cut in half to expose the sample. The halves were glued side-by-side in the bottom of a steel mold and more epoxy was added to make a 25 mm diameter cylinder suitable for polishing and microscopy.

The coal sample (all six particles together) was crushed to pass a 20 mesh sieve

(having 0.85 mm openings), dried in an oven for 6 hours at 50°C and divided to about 50 g using a riffle. The coal was wetted with epoxy and placed into two 25 mm diameter steel molds with end caps and pressed under 4000 psi pressure in accordance with the ASTM D2797 method for the preparation of petrographic briquettes. After hardening overnight, the coal cylinders were removed from the steel molds and prepared for polishing.

Polishing followed the same procedure for all samples, although a little more care was necessary with the dust samples. Samples were placed in a holder and ground on an automatic lap using both 400 and 600 grit abrasive paper, then polished in two stages using first 0.3 micron alumina slurry on high nap cloth and 0.05 micron alumina slurry on a silk to form a flat, level surface suitable for reflected light microscopy. After polishing, the samples were allowed to dry at least overnight over a desiccant.

All samples were inspected using a Zeiss Universal research reflectance microscope with a vertical illuminator capable of delivering polarized, white-light to the polished surface at 625X magnification (resolution of 1 to 2 μm). Immersion oil of 1.515 index of refraction was placed on the polished surfaces in a manner to form an interface between the sample and microscope objective. The change in index of refraction improves contrast among the different materials allowing for improved identification. During this inspection the types of particles, size, shape and approximate composition were evaluated and a decision was made regarding what, if any, analytical procedure could be initiated. The observation of small coal particles in all of the dust samples suggested that constituents should be photographed to compare with the coal sample. This was done using the Zeiss Universal with 100 ASA TMAX black & white film. In addition, the measurement of mean maximum vitrinite reflectance was employed to establish whether there may be any correspondence between the coal sample collected from along the rail road tracks and coal particles found in the dust samples.

To more fully understand the observations and analyses to be provided in this report some background information is necessary. The organic (carbonaceous) fraction of coal usually is derived from plant remains which have been altered first by bacteria and fungi at the point of deposition and then by temperature, time and pressure as the remains become buried deep in the earth. Coal *type* can be determined by accounting for the different constituents or **macerals** that are derived from the different layers of plant debris.

In general, coals are composed of three maceral groups (each containing many different recognizable components) including, **vitrinite, liptinite** and **inertinite** as shown in Plate IA and IB. Usually vitrinite-group macerals serve as the matrix for most coal, was derived from the alteration of woody tissues by bacteria and fungi in the original peat swamp, and results in a material that possesses thermoplastic properties in bituminous coals. Liptinite-group macerals constitutes a collection of materials composed of waxes.

resins, seed coats and lipids, which were highly resistant to decay in the original peat swamp. Generally, liptinite macerals are highly reactive in thermal processes, contributing much to low molecular weight volatile matter and little to the carbonaceous residue. Inertinite macerals represent a group of materials that were highly altered in the original peat swamp, either by thermal or biological oxidation. They are typically high in carbon and oxygen content, are generally inert with respect to thermoplasticity and are often recognizable in the residues from thermal processes, i.e., fly ash, metallurgical coke, etc.

Another characteristic of coal is the ***rank*** that it has attained through out burial by being exposed to elevated temperatures for significant periods of time. A common classification of coal rank is provided by the ASTM D388 standard that identifies coals as lignite, subbituminous, bituminous or anthracite. Using this system a coal's rank can be determined by measured values of calorific value or fixed carbon and volatile matter and agglomerating character. Another technique that has been used to define a coal's rank is the measure of the amount of light reflected from the polished surface of vitrinite. This technique is particularly valuable when there is insufficient sample to determine rank by standard techniques.

Measurement of the mean maximum vitrinite reflectance was conducted on polished samples following the appropriate ASTM procedure (D2798). Analysis was conducted using a Leitz MPV2 research microscope photometer system at 625X magnification using polarized white-light and oil immersion. The photometer system was calibrated using a series of glass standards of know reflectance. The procedure requires the identification of individual particles of vitrinite in a grid-like fashion across the polished surface. Each particle was brought into focus, and while the incident light reflecting from the surface is directed to the photometer, the particle (stage) is rotated 360° and a maximum reflectance reading recorded. To determine the mean maximum reflectance of a coal 100 individual readings are recorded and the mean value and distribution of reflectance readings reported. The quality of the polished surface, the presence of other macerals, additives or weathered coal particles can have a profound influence on the recorded values. When performed in accordance with the ASTM standard, maximum reflectance values obtained by a given laboratory are repeatable to 0.02% actual reflectance.

Mean maximum vitrinite reflectance was performed in accordance with the above procedure for coal sample #4. However, because of the size, composition and sparsity of coal particles in the dust samples only a small sampling of particles was possible. These results are given in Figures 1 and 2.

Results

Inspection of the crushed coal sample under an optical microscope suggested that

it may be a blend of two different high volatile A bituminous coals that are very similar in rank (see Plate I). This observation was based upon appearance, maceral associations and the relatively broad distribution of reflectance readings (ranging from 0.70 % to 1.12 %) as shown in Figure 1. The higher-reflecting coal particles tended to have a greater amount of fine size inertinite (micrinite) and the liptinite macerals appeared gray rather than black, whereas the low-reflecting coal particles exhibited thicker bands of vitrinite and were characterized by fairly high inertinite and liptinite content. See the comparison in Plate IC. Although some of the lower reflecting coal particles exhibited features of weathering (Plate ID), it was not possible to determine where the weathering occurred, i.e., along the rail road tracks, at the mine, during transit and storage, etc.

In general, all of the dust samples were composed of very nearly the same constituents in approximately the same relative order of concentration by volume. The most common component of the dust samples was aggregates of individual minerals, such as aluminosilicate clay-size particles (Plate IIA), fragments of quartz and/or carbonate (Plate IIIA), some iron oxide (rust, Plate IVB), and many mineral particles that remain unidentified. Because most of these particles were angular they did not appear to have been derived directly from thermal processes like stack emissions, but were probably normal airborne particles. The next most common component was fine, angular, non-thermally reacted coal particles (see Plate IIB, IIIA and IVA). Some particles, like that illustrated in Plate IIC, appeared to have been thermally altered and devolatilized. As seen in the photomicrographs and in Figure 2, coal particles found in the dust samples were very similar in composition and reflectance to the coal collected from along the rail road tracks. Finally, the last two components of the dust samples observed in much lower concentration included fly-ash like char particles (balloon-shaped or collapsed particles with relatively thin porous walls of high-reflecting and anisotropic carbon) and tissues from living plants (Plate IIID). Fly-ash particles like those seen in Plate IID, IIIC, IVC & D often exhibited a granular texture in crossed-polarized light which is characteristic of the 1-2 micron size anisotropic texture inherited from the rapid heating of a bituminous coal.

Because of the small amount of each dust sample and the difficulty of ensuring a representative sampling, no attempt was made to quantify the concentration of the major particle types observed beyond what has been given. However, owing to the fact that mineral components have a significantly higher density than carbonaceous materials, by weight the mineral components would constitute most of the sample followed very closely by coal particles. The occurrence of other materials like fly-ash, plant tissues and unidentifiable materials, although persistent was not significant. In addition, the two-dimensional cross-section of particles was measured along the shortest dimension to give some idea of particle size for each of the components. In general, the mean diameter of mineral particles was greatest at 65 μm, coal particles averaged about 45 μm and mean fly-ash particle diameter was 30 μm. Measured particle sizes ranged from below 10 to 100 μm and can be considered to be very fine sand to coarse silt.

## Summary

Using a petrographic technique to investigate the nature of the contents of dust samples collected last June at Ms. Chimento's residence has revealed a significant presence of coal dust on a volume basis. While it is not possible to determine the magnitude of the coal dust problem in the neighborhood from this investigation, it appeared to be sufficiently common in June 2002 that accumulation occurred at about equal amounts on two sides of a house and between the first and second floor. Also, the fact that the reflectance of coal particles (vitrinite matrix) in all of the dust samples had the same distribution as spillage coal along the rail road tracks servicing the Merant Plant (and if there is no other source using coal in the general vicinity) suggests that the coal fines may be coming from the coal delivery, handling or preparation part of plant operations.

## Recommendations

These findings represent a departure from past thinking, in that past efforts have focused primarily on stack emissions (fly-ash) as the root cause of dust accumulation, whereas this investigation suggests a raw materials handling source. Some fly-ash materials were found in all dust samples, but a relatively insignificant amount compared to coal dust. If there are no other sources using coal in the area and unit trains of coal do not pass through the area to other locations, then it is clear that a significant amount of the dust accumulated on window ledges and deck last June came from operations at the nearby Merant Plant.

From this study, it is not possible to determine the exact source of coal dust, i.e., from rail road car tops, dumping operations, stock piling and reclaiming, transportation, crushing operations or all of the above. Also it is not possible to determine the rate of dust accumulation or really the potential losses of coal from plant operations from this study, but personnel at the Merant Plant may be interested in these findings. Getting a handle on these losses may be beneficial from a financial point of view (valuable coal is being lost) as well as a service to the community.

## Bibliography

1. Annual Book of ASTM Standards (1999), "Gaseous Fuels; Coal and Coke", vol. 05.05, 650 pp.
2. Taylor G.H., Teichmuller, M., Davis, A., Diessel, C.F.K., Littke, R. and Robert, P. (1998), "Organic Petrology", Gebruder Borntraeger, Berlin, 704 pp.
3. Bailey, J. (1992), The Origin of Unburnt Combustibles in Coal, Unpub. Ph.D Thesis, University of Newcastle, New South Wales, Australia, 383 pp.

## Description of Photomicrographs

### Plate I – Sample #4, Spillage coal from along rail road tracks

A. A coal particle that illustrates the diverse nature of coal and showing examples of the vitrinite- (v), liptinite- (l) and inertinite- (i) group macerals. Notice how vitrinite serves as the particle matrix for the other maceral groups or can be found as individual layers free of other macerals.
B. A coal particle having a high concentration of inertinite (i) and with minor amounts of vitrinite (v) and liptinite (l).
C. Comparison of the higher- (left) and lower- (right) reflecting coal particles found in Sample #4 which suggests that two different high volatile A bituminous coals may have been sampled from the tracks.
D. The uneven and darkened edge of this coal particle is characteristic of weathering.

### Plate II – Sample #1, Dust from back deck window ledge 2nd floor

A. Granular texture, poor polish, relatively low reflectance and internal reflection are characteristic features of aluminosilicate clay particles.
B. Edge of fairly large coal particle containing high reflecting inertinite (i) as found in this dust sample.
C. This appears to be a remnant of a coal particle that has been heated to a point at which volatile matter is given off and the vitrinite matrix has become thermoplastic. Note the rounded vacuoles in the particle interior.
D. Combustion char particle (crassinetwork) from a high rank bituminous coal that has developed a submicron anisotropic texture during thermal processing.

### Plate III – Sample #2, Dust from 2nd floor back deck

A. A small coal particle (left) beside a clay particle (right) bearing fragments of quartz and carbonate.
B. A high vitrinite containing coal particle exhibiting collapsed cell structure (thin black lines represent areas between cell walls).
C. A typical thick-walled cenosphere (crassisphere) derived from coal combustion. Note the high reflecting wall structure has many small gas vacuoles in addition to the one large pore near the center.
D. Cell wall tissue from a living plant.

**Plate IV – Sample #3, Dust from front 1st floor window ledge (street side)**

A. The edge of a relative large coal particle with a high inertinite content and very similar to coal sample #4 as seen in Plate IB.
B. Several rust particles found in this dust sample and generally composed of iron oxide that is partially hydrated (lower reflecting edge).
C. A typical coal combustion char particle (crassinetwork) exhibiting a granular texture as a result of optical anisotropy.
D. Another classic thick-walled cenosphere (crassisphere) derived from coal combustion.

Figure 1 – Mean Maximum Vitrinite Reflectance and Distribution of Coal Particles Collected from Along Rail Road Tracks Beside the Merant Plant

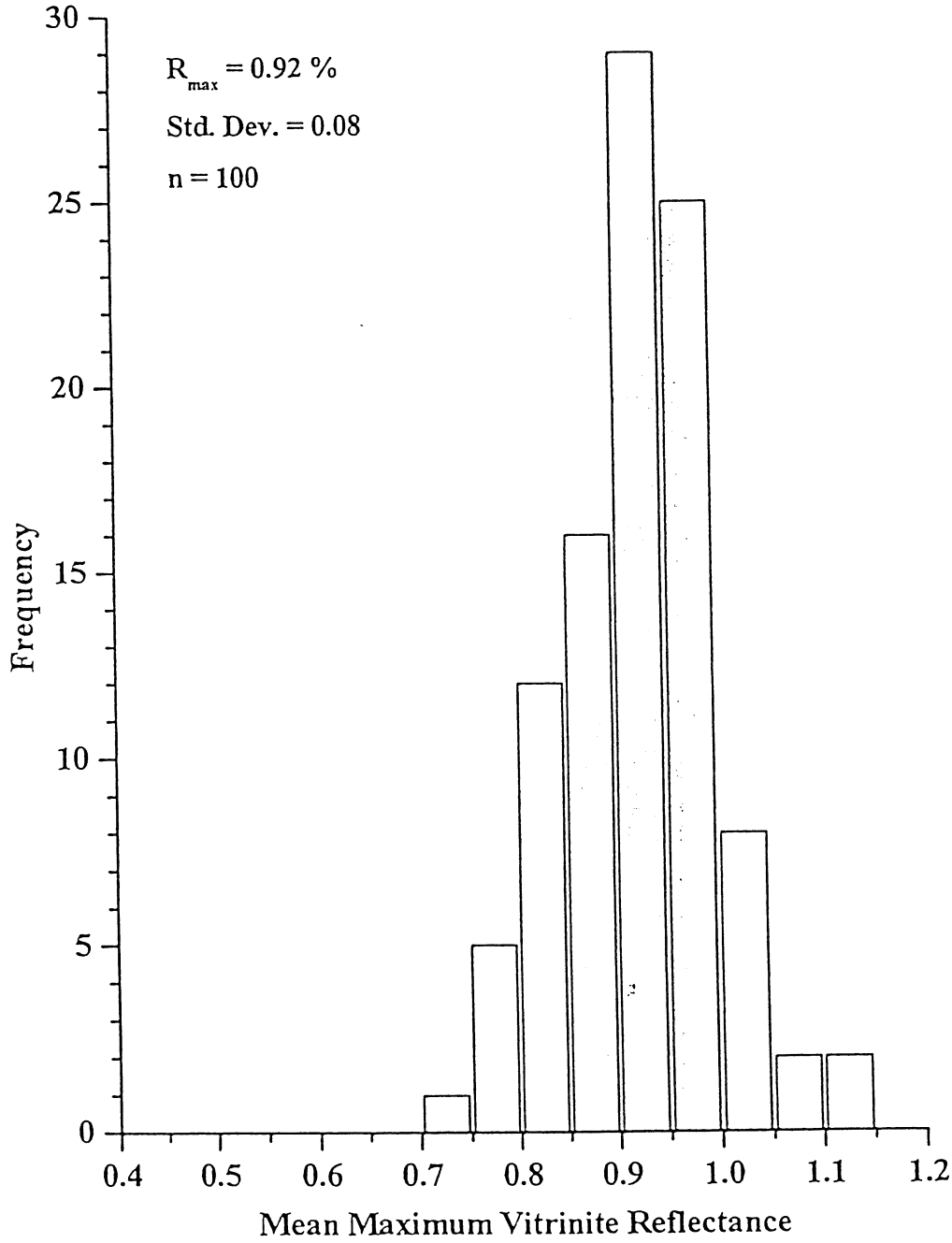
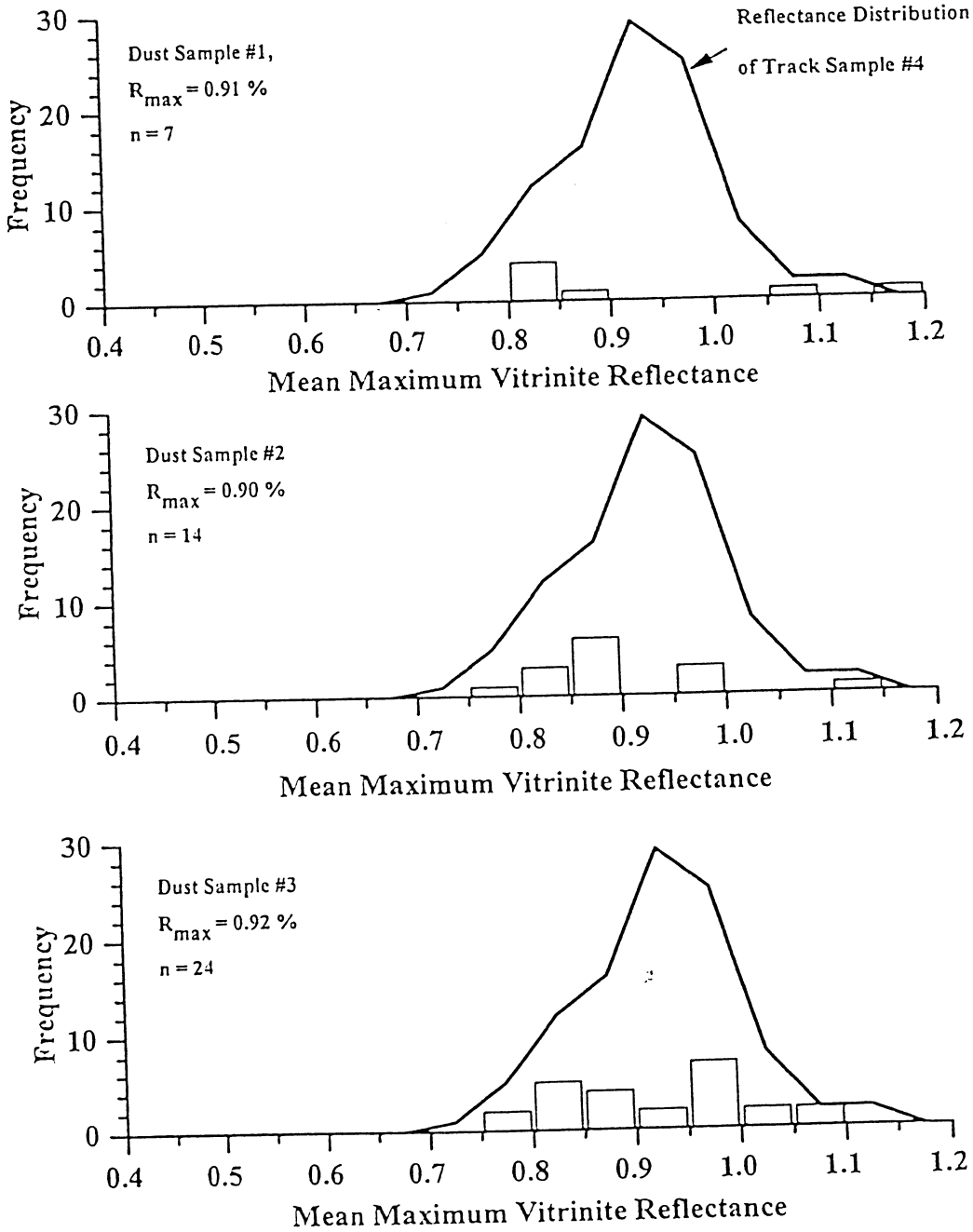


Figure 2 – Maximum Reflectance Readings on Coal Particles Found in Three Dust Samples Compared with Coal Found Beside the Rail Road Tracks



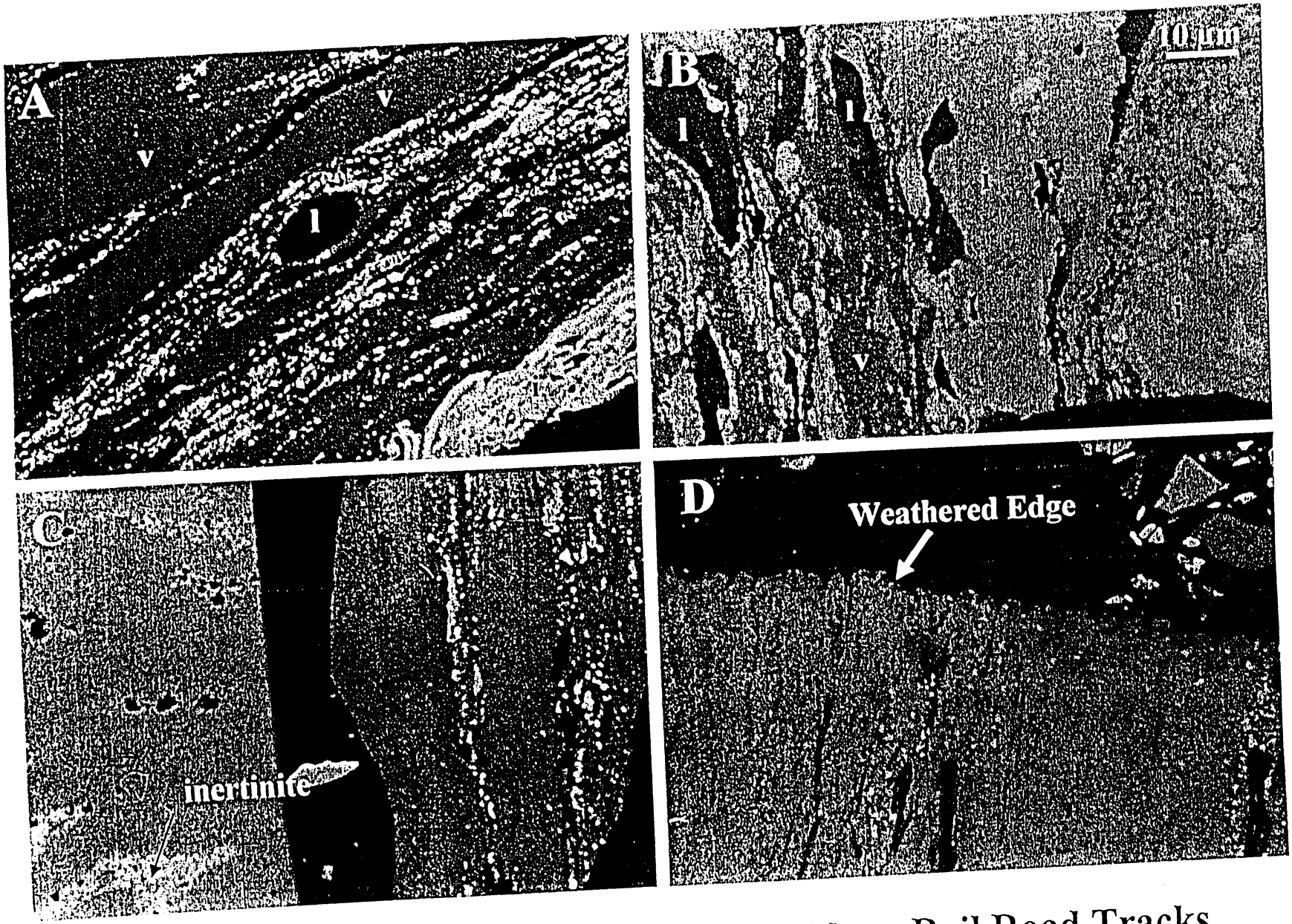



Plate I – Sample #4, Spillage Coal From Along Rail Road Tracks

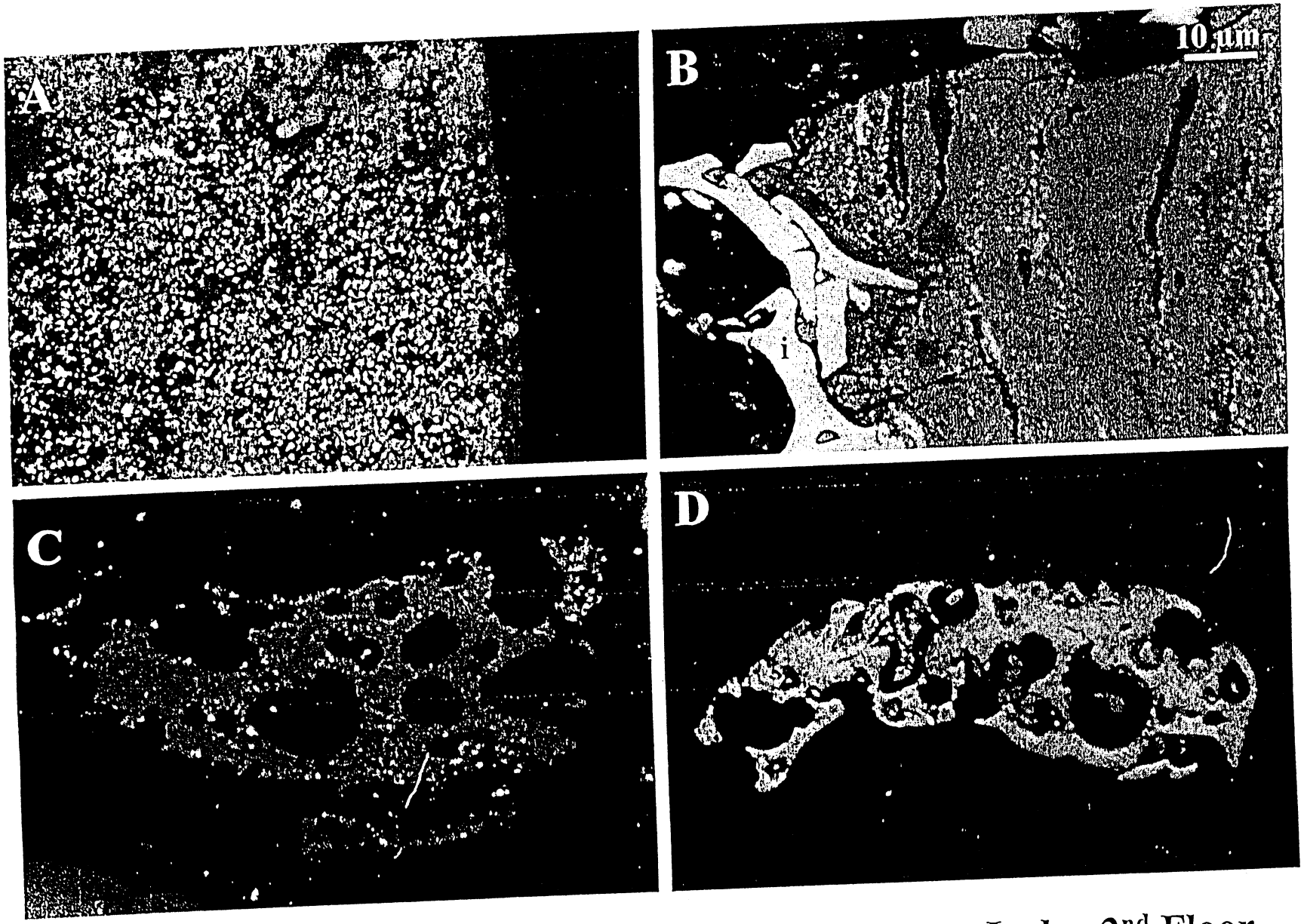



Plate II – Sample #1, Dust From Back Deck Window Ledge 2nd Floor

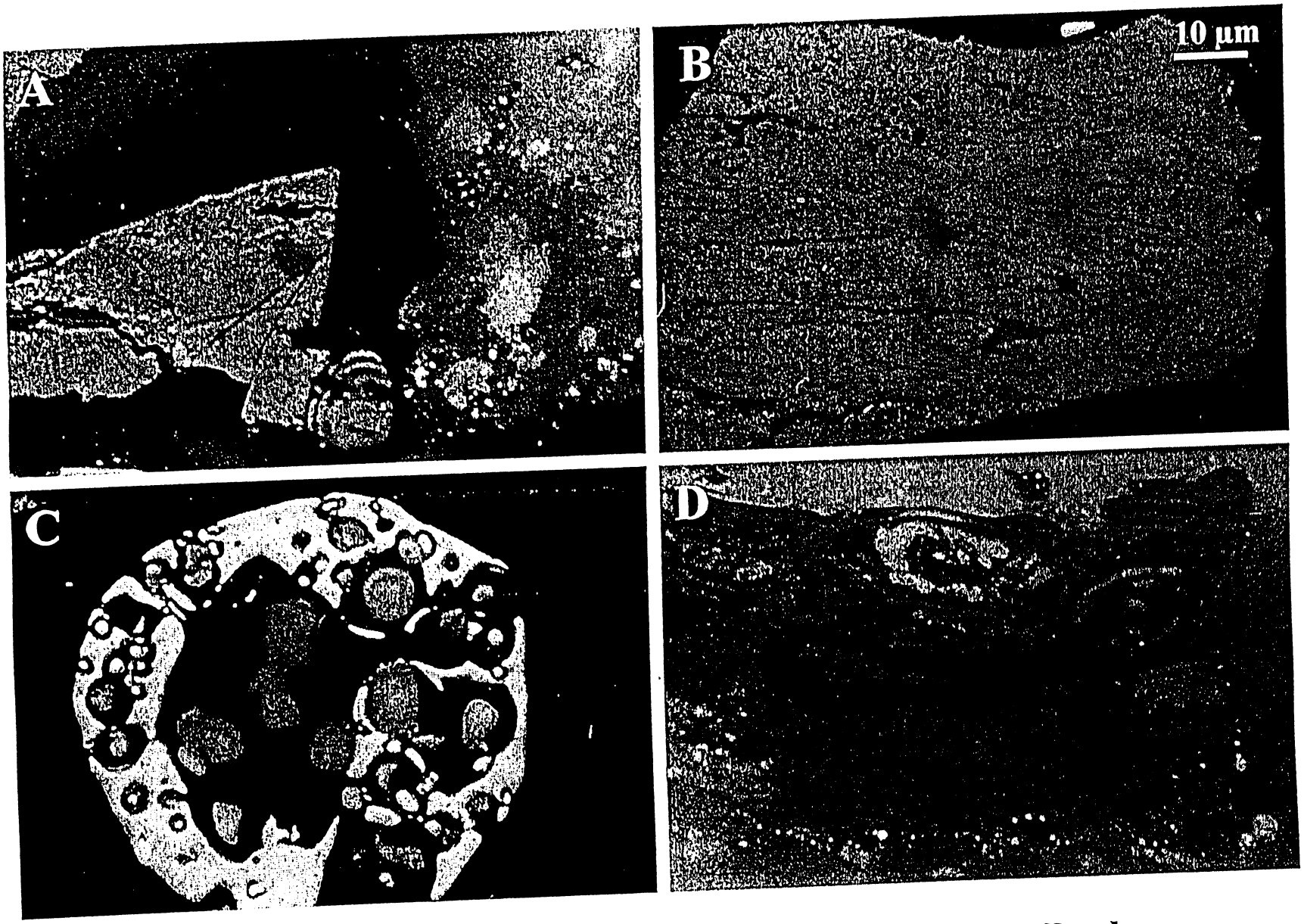



Plate III – Sample #2, Dust From 2nd Floor Back Deck

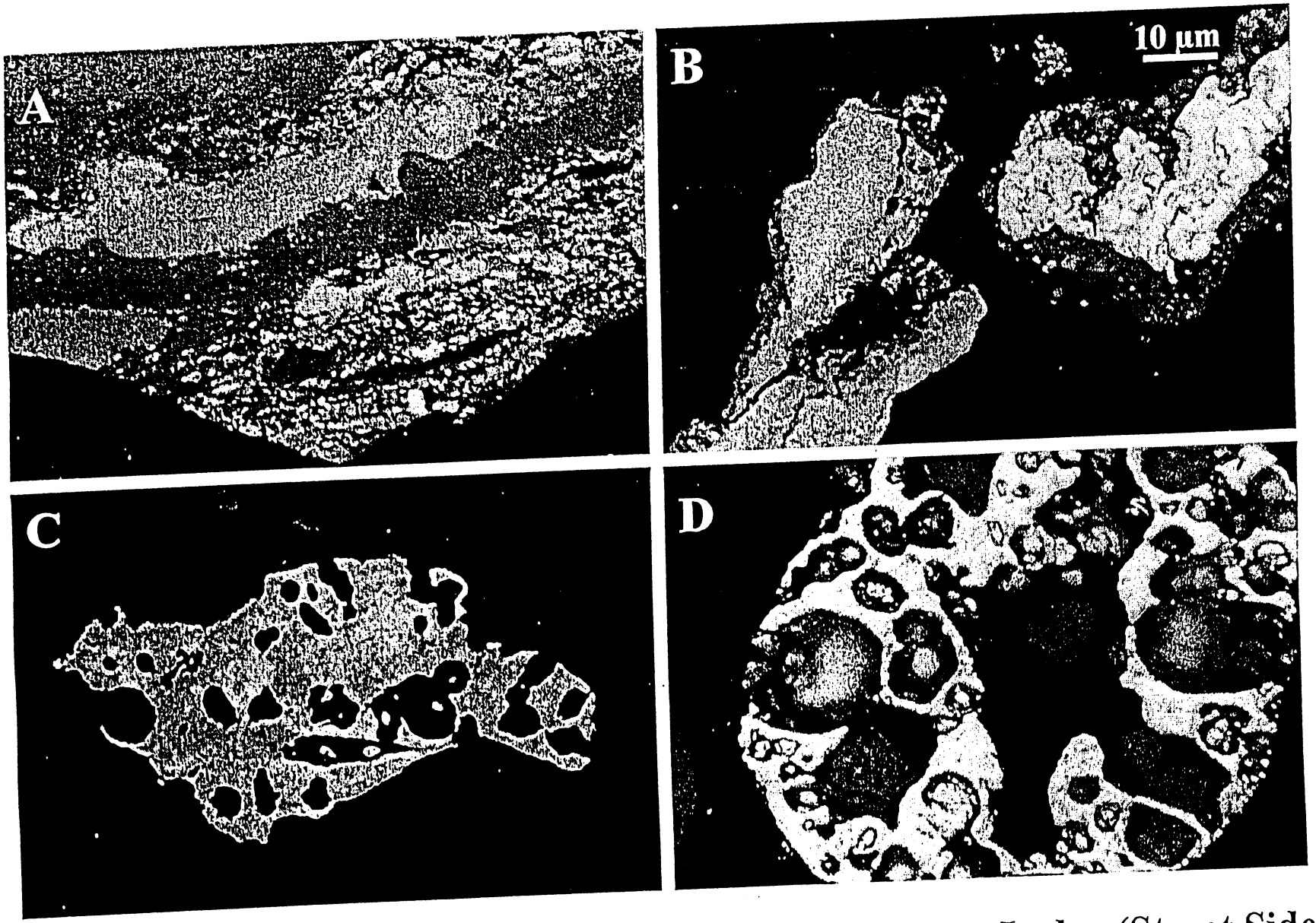



Plate IV – Sample #3, Dust From Front 1st Floor Window Ledge (Street Side)

# COMMONWEALTH OF VIRGINIA
# DEPARTMENT OF ENVIRONMENTAL QUALITY

## MEMORANDUM

**TO:** Charles D. Forbes (DEQ/NVRO)

**FROM:** Kelly Lease, Air Compliance Inspector, NVRO

**SUBJECT:** Analysis of Dust Samples Taken at 1200 and 1202 Pitt Street, Alexandria, Virginia

**DATE:** June 10, 2003

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The purpose of this memo is to present the analytical results for dust samples collected in response to citizen complaints regarding particulate emissions from Mirant's Potomac River Generating Station, a coal-fired power plant located within the City of Alexandria. Dust samples were collected from areas in the front and rear of residences at 1200 and 1202 Pitt Street in Alexandria at the request of Ms. Elizabeth Chimento, a resident of 1200 Pitt Street. Her residence is approximately one-quarter mile from the plant.

The samples were collected on April 22, 2003, by Ms. Kelly Lease and Mr. David Hartshorn with the Department's Northern Virginia Regional Office (NVRO). In addition to Ms. Chimento, Mr. Poul Hertel, also an Alexandria resident, was present during the sampling event. The samples were delivered to the Department's Office of Air Quality Assessment, with an unbroken chain of custody.

The samples were visually examined using a polarizing light microscope at 150 times magnification. The results of this examination are attached. To summarize these results and follow up discussions with the analyst of record, uncombusted coal dust was estimated to constitute up to 50 percent of each sample. Coal combustion products were also present, including partially combusted coal and partially fused ash, at approximately 10 percent of each sample. These combustion products appeared to have not been exposed to high temperature. It should be noted that this analysis has the following limitations: a) a particle count was not performed; and b) as black particles, coal dust stands out against lighter particles of other materials. This can result in over-estimating their relative proportion within the sample. The remainder of the constituents in the sample were either biological (e.g., pollen, fungus, algae, stellate hairs) or materials common to urban environments (e.g., asphalt, mineral particulate, rubber, fibers, paint, wood dust).

In reviewing these results, it is important to note that these results cannot be considered quantitative. The sampling methodology used (i.e., simply brushing dust from surfaces into vials) does not support defensible conclusions regarding constituent concentrations in ambient air, nor when materials found in the sample were generated. Furthermore, no chemical analysis was performed on the samples. Simply put, NVRO can make no formal determination regarding the compliance status of the Potomac River Generating Station as a result of this sampling, but the results warrant further discussions with Mirant and closer monitoring of the facility. To this end, and with your approval, NVRO's compliance inspection strategy for the Potomac River Facility will be modified to focus attention on coal-handling procedures and particulate emission control features at the facility. A meeting with Mirant will be scheduled to discuss this report and review the operating records and current conditions at the plant. At this time, the pertinent dust control regulations (9 VAC 5-40-90 - Standard for Fugitive Dust/Emissions) will be reviewed.

cc: Carolyn Stevens (DEQ Office of Air Quality Assessment)
Lalit Sherma (Alexandria Health Department)
Debra Knight (Mirant Mid-Atlantic, LLC)

**Virginia Dept. of Environmental Quality**
**Office of Air Quality Assessment**
**Microscopic Analysis Form**

**Submittal No.** M0498

**Submitted by:** Kelly Lease

**Date sample received:** 5/14/03

**Sample description:** Twelve vials containing sample material collected from 1200 and 1202 Pitt Street in Alexandria, VA, in response to a citizen complaint

**Analytical results:** (Include date of completion of analysis and signature of responsible party)

**Identified:**

1. Vial labeled "Glass Table Deck": Sample moderate to large amounts of asphalt, coal dust, mineral particulate (quartz, biotite, calcite, clay, etc), pollen, fungus, assorted fibers, rubber, coal combustion products
2. Vial labeled "1200 Front Window": Sample contained moderate to large amounts of asphalt, pollen, coal dust, mineral particulate; smaller amounts of algae, paint, partially fused ash, rubber
3. Vial labeled "1202 Window Ledge": Sample contained moderate to large amounts asphalt, mineral particulate, pollen, stellate hairs, coal dust; smaller amounts of coal combustion products, fungus
4. Vial labeled "1202 Entry": Sample contained moderate to large amounts of asphalt, pollen, mineral particulate, coal dust, wood dust, stellate hairs; smaller amounts of assorted fibers, paint, coal combustion products, rubber, partially fused ash
5. Vial labeled "1202" Entryway": Same as #4, including some fungus
6. Vial labeled "1200 Entryway": Same as #4
7. Vial labeled "1200 E Entry": Same as #4
8. Vial labeled "1202 Front Window": Sample contained mostly pollen; also contained asphalt, mineral particulate, coal dust, stellate hairs, paint, coal combustion products, fungus
9. Vial labeled "1200 Side Window": Sample contained mostly pollen; also contained asphalt, coal dust, mineral particulate, rubber, coal combustion products
10. Vial labeled "Rear Windowsill": Sample contained mostly pollen; also contained small amounts of coal dust, coal combustion products, stellate hairs

Two samples, "1200 Front Window" and "Rear Windowsill", were collected on tape, and could not be analyzed. Most of the coal contained in the samples did not appear to have been exposed to any combustion process.

C. M. Stevens
5/30/03

COMMONWEALTH OF VIRGINIA
STATE AIR POLLUTION CONTROL BOARD
REGULATIONS FOR THE CONTROL AND ABATEMENT OF AIR POLLUTION

9 VAC 5 CHAPTER 40.
EXISTING STATIONARY SOURCES.

PART II.
Emission Standards.

ARTICLE 1.
Visible Emissions and Fugitive Dust/Emissions (Rule 4-1).


9 VAC 5-40-60. Applicability and designation of affected facility.
9 VAC 5-40-70. Definitions.
9 VAC 5-40-80. Standard for visible emissions.
9 VAC 5-40-90. Standard for fugitive dust/emissions.
9 VAC 5-40-100. Monitoring.
9 VAC 5-40-110. Test methods and procedures.
9 VAC 5-40-120. Waivers.


9 VAC 5-40-60. Applicability and designation of affected facility.

A. The affected facilities to which the provisions of this article apply are the following:

1. Each source of visible emissions; and

2. Each source of fugitive dust/emissions.

B. The provisions of this article apply throughout the Commonwealth of Virginia.

9 VAC 5-40-70. Definitions.

A. For the purpose of these regulations and subsequent amendments or any orders issued by the board, the words and terms shall have the meaning given them in subsection C of this section.

B. As used in this article, all terms not defined here shall have the meaning given them in 9 VAC 5 Chapter 10 (9 VAC 5-10-10 et seq.), unless otherwise required by context.

C. Terms defined.

"Fugitive dust" means particulate matter composed of soil or other materials, or both, of natural origin. Fugitive dust may include emissions from haul roads, wind erosion of exposed surfaces and storage piles and other activities in which the material is

either removed, stored, transported or redistributed.

"Fugitive emissions" means emissions which are generated by industrial or other activities and which do not pass through a stack, chimney, vent or other functionally equivalent opening, but which may escape from openings (such as windows, doors, ill-fitting closures or poorly maintained equipment) or material handling equipment.

"Opacity" means the degree to which emissions reduce the transmission of light and obscure the view of an object in the background, expressed as a percentage.

"Six-minute period" means any one of the 10 equal parts of one hour or a one-hour period, as may be applicable.

9 VAC 5-40-80. Standard for visible emissions.

Unless specified otherwise in this part, no owner or other person shall cause or permit to be discharged into the atmosphere from any affected facility any visible emissions which exhibit greater than 20% opacity, except for one six-minute period in any one hour of not more than 60% opacity. Failure to meet the requirements of this section because of the presence of water vapor shall not be a violation of this section.

9 VAC 5-40-90. Standard for fugitive dust/emissions.

No owner or other person shall cause or permit any materials or property to be handled, transported, stored, used, constructed, altered, repaired or demolished without taking reasonable precautions to prevent particulate matter from becoming airborne. Such reasonable precautions may include, but are not limited to, the following:

1. Use, where possible, of water or chemicals for control of dust in the demolition of existing buildings or structures, construction operations, the grading of roads or the clearing of land.

2. Application of asphalt, water, or suitable chemicals on dirt roads, materials stockpiles and other surfaces which may create airborne dust; the paving of roadways and maintaining them in a clean condition.

3. Installation and use of hoods, fans and fabric filters to enclose and vent the handling of dusty materials. Adequate containment methods shall be employed during sandblasting or other similar operations.

4. Open equipment for conveying or transporting materials likely to create objectionable air pollution when airborne shall be covered or treated in an equally effective manner at all times when in motion.

5. The prompt removal of spilled or tracked dirt or other materials from paved streets and of dried sediments resulting from soil erosion.

9 VAC 5-40-100. Monitoring.

A. Unless otherwise approved by the board, all continuous monitoring systems required by this article shall be installed, calibrated, maintained and operated in accordance with applicable requirements in 9 VAC 5-40-40 and 9 VAC 5-40-41.

B. Each owner required to install a continuous monitoring system shall provide notifications and reports and maintain records and monitoring results in accordance with the requirements of 9 VAC 5-40-50.

C. In cases where the requirements of 9 VAC 5-40-40 and 9 VAC 5-40-41 are not appropriate for a particular source type, the owner shall comply with other procedures acceptable to the board.

9 VAC 5-40-110. Test methods and procedures.

The provisions of 9 VAC 5-40-20 A 2 apply to determine compliance with the standard prescribed in 9 VAC 5-40-80.

9 VAC 5-40-120. Waivers.

A. A waiver from the opacity emission limitation in 9 VAC 5-40-80 may be granted by the director, provided that a technical decision is reached that the plume opacity observations made in accordance with 9 VAC 5-40-20 and 9 VAC 5-40-110 are not representative of the pollutant loading of the plume.

B. Upon granting the above waiver, the director shall require one or more alternate source surveillance methods, which may include, but are not limited to, the following:

1. Requiring the owner to install, calibrate, maintain and operate systems for continuously monitoring and recording emissions of specified pollutants in accordance with 9 VAC 5-40-40 and 9 VAC 5-40-100.

2. Requiring the owner to conduct, at specified intervals, emission tests for measuring emissions of specified pollutants in accordance with 9 VAC 5-40-30.

3. Establishing an opacity emission limitation for the facility based on a correlation between tests of visible and other specified pollutant emissions.

C. The waiver may be granted for an indefinite period of time; however, approval may be withdrawn by the director:

1. For failure to adhere to any terms or conditions of the waiver;

2. If the affected facility is found to be in violation of any applicable emission standard; or

3. For failure to conduct or adhere to any alternate source surveillance method required for waiver approval.

HISTORICAL NOTES:

Derived from: Rule 4-1 of Part IV of VR 120-01 (§ 120-04-0101 through § 120-04-0107)

Effective Date: March 17, 1972
Promulgated: March 17, 1972
Amended: August 9, 1975
Amended: October 6, 1978
Amended: August 3, 1979
Amended: October 5, 1979
Amended: January 1, 1985
Amended: February 1, 2003

REG\VAC\401

HEALTH EFFECTS INSTITUTE

# STATEMENT

## Synopsis of the Particle Epidemiology Reanalysis Project

### BACKGROUND

Epidemiologic work conducted over several decades has suggested that long-term residence in cities with elevated ambient levels of air pollution from combustion sources is associated with increased mortality. Subsequently, two prospective cohort studies, the Six Cities Study (as reported in Dockery et al 1993) and the American Cancer Society (ACS) Study (as reported in Pope et al 1995) estimated that annual average all-cause mortality increased in association with an increase in fine particles (all particles less than 2.5 μm in median aerodynamic diameter [$PM_{2.5}$]).

As part of the Six Cities Study, Dockery and colleagues (1993) had prospectively followed a cohort of 8,111 adult subjects in northeast and midwest United States for 14 to 16 years beginning in the mid-1970s. The authors found that higher ambient levels of fine particles and sulfate ($SO_4^{2-}$) were associated with a 26% increase in mortality from all causes when comparing the most polluted to the least polluted city, and that an increase in fine particles was also associated with increased mortality from cardiopulmonary disease. The relative risks in all-cause mortality were associated with a difference (or range) in ambient fine particle concentrations of 18.6 μg/m$^3$ and a difference of ambient sulfate concentrations of 8.0 μg/m$^3$, comparing the least polluted city to the most polluted city.

In the much larger ACS Study, Pope and colleagues (1995) followed 552,138 adult subjects in 154 US cities beginning in 1982 and ending in 1989 (3 cities did not overlap between the 151 and 50 cities studied, resulting in a total of 154 cities). Again, higher ambient levels of fine particles were associated with increased mortality from all causes and from cardiopulmonary disease in the 50 cities for which fine particle data were available (sampled from 1979 to 1983). Higher ambient sulfate levels were associated with increased mortality from all causes, cardiopulmonary disease, and lung cancer in the 151 cities for which sulfate data were available (sampled from 1980 to 1982). The difference between all-cause mortality in the most-polluted city and the least-polluted city was 17% and 15% for fine particles and sulfate, respectively (with a range of 24.5 μg/m$^3$ for fine particles and of 19.9 μg/m$^3$ for sulfate).

Both of these studies came under intense scrutiny in 1997 when the EPA used the results to support new National Ambient Air Quality Standards for fine particles and to maintain the standards for particles less than 10 μm in median aerodynamic diameter ($PM_{10}$) already in effect. Members of Congress and industry, the scientific community and others interested in regulation of air quality scrutinized the studies' methods and their results. Some insisted that any data generated using federal funding should be made public. Others argued that these data had been gathered with assurances of confidentiality for the individuals who had agreed to participate and that the concept of public access to federally funded data did not take into account the intellectual property rights of the investigators and their supporting institutions. To address the public controversy, Harvard University and the ACS requested that the Health Effects Institute organize an independent reanalysis of the data from these studies. Both institutions agreed to provide access to their data to a team of analysts to be selected by HEI through a competitive process.

### APPROACH

To conduct the reanalysis, the HEI Board of Directors, with support from the EPA, industry, Congress, and other stakeholders, appointed an Expert Panel chaired by Dr Arthur Upton from the University of Medicine and Dentistry of New Jersey and former Director of the National Cancer

This Statement, prepared by the Health Effects Institute, is a summary of a research project conducted by the Reanalysis Team, led by Dr Daniel Krewski at the University of Ottawa. The following Special Report contains the detailed Investigators' Report (Summary, Introduction, and Parts I and II), Commentary on the project prepared by a special panel of the Institute's Health Review Committee, and Comments on the Reanalysis Project by the Original Investigators (Drs Douglas W Dockery, C Arden Pope III et al).

Institute. The Expert Panel selected competitively a Reanalysis Team—led by Dr Daniel Krewski of the University of Ottawa—and oversaw all aspects of the team's work. They were assisted in their oversight efforts by a broad-based Advisory Board of knowledgeable stakeholders and scientists who, in the project's early stages, provided extensive advice to the Expert Panel on the key questions to be analyzed. The final results of the Reanalysis Team were intensively and independently peer reviewed by a Special Panel of the HEI Health Review Committee, which was chaired by Dr Millicent Higgins of the University of Michigan.

The overall objective of what became the Particle Epidemiology Reanalysis Project was to conduct a rigorous and independent assessment of the findings of the Six Cities and ACS Studies of air pollution and mortality. This objective was met in two parts. In *Part I: Replication and Validation*, the Reanalysis Team sought to replicate the original studies via a quality assurance audit of a sample of the original data and to validate the original numeric results. In *Part II: Sensitivity Analyses*, they tested the robustness of the original analyses to alternate risk models and analytic approaches.

## RESULTS AND IMPLICATIONS

### PART I: REPLICATION AND VALIDATION

- An extensive audit of the study population data for both the Six Cities and ACS Studies and of the air quality data in the Six Cities Study revealed the data to be of generally high quality with a few exceptions. In both studies, a few errors were found in the coding and inclusion of certain subjects; when those subjects were included in the analyses, they did not materially change the results as originally reported. Because the air quality data used in the ACS Study could not be audited, a separate air quality database was constructed for the sensitivity analyses described in Part II.
- The Reanalysis Team was able to replicate the original results in both studies using the same data and statistical methods as used by the Original Investigators. The Reanalysis Team confirmed the original point estimates: For the Six Cities Study, they reported the relative risk of mortality from all causes associated with an increase in fine particles of 18.6 $\mu g/m^3$ as 1.28, close to the 1.26 reported by the Original Investigators. For the ACS Study, the relative risk of mortality from all causes associated with an increase in fine particles of 24.5 $\mu g/m^3$ was 1.18 in the reanalysis, close to the 1.17 reported by the Original Investigators.

### PART II: SENSITIVITY ANALYSES

Once the original results of the studies had been validated, the Reanalysis Team sought to test an array of different models and variables to determine whether the original results would remain robust to different analytic assumptions.

- First, the Reanalysis Team used the standard Cox model used by the Original Investigators and included variables in the model for which data were available from both original studies but had not been used in the published analyses (eg, physical activity, lung function, marital status). The Reanalysis Team also designed models to include interactions between variables. None of these alternative models produced results that materially altered the original findings.
- Next, for both the Six Cities and ACS Studies, the Reanalysis Team sought to test the possible effects of fine particles and sulfate on a range of potentially susceptible subgroups of the population. Although different subgroups did show some variation in their estimated effects, the results were not statistically significant with one exception. The estimated effects of fine particles did appear to vary with educational level; the association between an increase in fine particles and mortality tended to be higher for individuals without a high school education than for those who had completed high school or for those with more than a high school education.
- In the ACS study, the Reanalysis Team tested whether the relationship between ambient concentrations and mortality was linear. They found some indications of both linear and nonlinear relationships, depending upon the analytic technique used, suggesting that the

issue of concentration-response relationships deserves additional analysis.

- In the Six Cities Study where data were available, the Reanalysis Team tested whether effect estimates changed when certain key risk factors (smoking, body mass index, and air pollution) were allowed to vary over time. One of the criticisms of both original studies has been that neither analyzed the effects of change in pollutant levels over time. In general, the reanalysis results did not change when smoking and body mass index were allowed to vary over time. The Reanalysis Team did find for the Six Cities Study, however, that when the general decline in fine particle levels over the monitoring period was included as a time-dependent variable, the association between fine particles and all-cause mortality dropped substantially, but the effect continued to be positive and statistically significant.
- Using its own air quality dataset constructed from historical data to test the validity of the original ACS air quality data, the Reanalysis Team found essentially the same results.
- Any future analyses using the sulfate data should take into account the impact of artifactual sulfate. Sulfate levels with and without adjustment differed by about 10% for the Six Cities Study. Both the original ACS Study air quality data and the newly constructed dataset contained sulfate levels inflated by approximately 50% due to artifactual sulfate. For the Six Cities Study, the relative risks of mortality were essentially unchanged with adjusted or unadjusted sulfate. For the ACS Study, adjusting for artifactual sulfate resulted in slightly higher relative risks of mortality from all causes and cardiopulmonary disease compared with unadjusted data. The relative risk of mortality from lung cancer was lower after the data had been adjusted.
- Because of the limited statistical power to conduct most sensitivity analyses for the Six Cities Study, the Reanalysis Team conducted the majority of its sensitivity analyses using only the ACS Study dataset with 154 cities. In that dataset, when a range of city-level (ecologic) variables (eg, population change, measures of income, maximum temperature, number of hospital beds, water hardness) were included in the analyses, the results generally did not change. Two exceptions were that associations for both fine particles and sulfate were reduced when city-level measures of population change or sulfur dioxide were included in the model.
- A major contribution of the Reanalysis Project is the recognition that both pollutant variables and mortality appear to be spatially correlated in the ACS Study dataset. If not identified and modeled correctly, spatial correlation could cause substantial errors in both the regression coefficients and their standard errors. The Reanalysis Team identified several methods for dealing with this, all of which resulted in some reduction in the estimated regression coefficients. The full implications and interpretations of spatial correlations in these analyses have not been resolved and appear to be an important subject for future research.
- When the Reanalysis Team sought to take into account both the underlying variation from city to city (random effects) and the spatial correlation between cities, only sulfur dioxide as a city-level variable continued to decrease the originally reported associations between mortality and fine particles or sulfate. This effect was more pronounced for sulfate.
- When the Reanalysis Team conducted spatial analyses of sulfur dioxide, the association between sulfur dioxide and mortality persisted after adjusting for sulfate, fine particles, and other variables.
- As a result of these extensive analyses, the Reanalysis Team was able to explain much of the variation between cities, but some unexplained city-to-city variation remained.

## CONCLUSIONS

The Reanalysis Team designed and implemented an extensive and sophisticated series of analyses that included a set of new variables, all the gaseous copollutants, and the first attempts to apply spatial analytic methods to test the validity of the data and the results from the Six Cities Study and the ACS Study. Overall, the reanalyses assured the quality of the original data, replicated

the original results, and tested those results against alternative risk models and analytic approaches without substantively altering the original findings of an association between indicators of particulate matter air pollution and mortality.

At the same time, the reanalyses did extend and challenge our understanding of the original results in several important ways.

- The Reanalysis Team identified a possible modifying effect of education on the relation between air quality and mortality in that estimated mortality effects increased in the subgroup with less than high school education.
- The use of spatial analytic methods suggested that, when the analyses controlled for correlations among cities located near one another, the associations between mortality and fine particles or sulfate remained but were diminished.
- An association between sulfur dioxide and mortality was observed and persisted when other possible confounding variables were included; furthermore, when sulfur dioxide was included in models with fine particles or sulfate, the associations between these pollutants (fine particles and sulfate) and mortality diminished.

In reviewing these results, the Special Panel of the HEI Health Review Committee identified the following factors to consider when interpreting the results from the Reanalysis Team.

- The inherent limitations of using only six cities, understood by the Original Investigators, should be taken into account when interpreting results of the Six Cities Study.
- The Reanalysis Team did not use data adjusted for artifactual sulfate for most alternative analyses. When they did use adjusted sulfate data, relative risks of mortality from all causes and cardiopulmonary disease increased. This result suggests that more analyses with adjusted sulfate might result in somewhat higher relative risks associated with sulfate.
- Findings from spatial analyses applied to the ACS Study data need to be interpreted with caution; the spatial adjustment may have overadjusted the estimated effect for regional pollutants such as fine particles and sulfate compared with the effect estimates for more local pollutants such as sulfur dioxide.
- After the Reanalysis Team completed its spatial analyses, residual spatial variation was still noticeable; this finding suggests that additional studies might further refine our understanding of the spatial patterns in both air pollution and mortality.
- No single epidemiologic study can be the basis for determining a causal relation between air pollution and mortality.

In conclusion, the Reanalysis Team interpreted their findings to suggest that increased relative risk of "mortality may be attributed to more than one component of the complex mix of ambient air pollutants in urban areas in the United States". The Review Panel concurs. In the alternative analyses of the ACS Study cohort data, the Reanalysis Team identified relatively robust associations of mortality with fine particles, sulfate, and sulfur dioxide, and they tested these associations in nearly every possible manner within the limitations of the datasets. Future investigations of these issues will enhance our understanding of the effect of combustion-source air pollutants (eg, fine particles, sulfate, and sulfur dioxide) on public health.

HEI

STATEMENT

Synopsis of Research Report 110

HEALTH EFFECTS INSTITUTE

# Effects of Metals Bound to Particulate Matter on Human Lung Epithelial Cells

## INTRODUCTION

Inhaled particulate matter has been associated with both acute and chronic health effects. Concerns about these effects derive primarily from epidemiologic studies that associate short-term increases in particle concentration with increases in daily morbidity and mortality from respiratory and cardiovascular diseases. Over the past decade much research has been directed toward identifying plausible mechanisms linking particulate matter and pathophysiologic effects. Although progress has been made, many critical aspects are not understood. Thus, studies of the properties of particles that might induce pathologic effects are critical to establishing the mechanisms of particulate matter toxicity and to producing information necessary to target regulation of the sources that generate the most toxic particles.

Studies using laboratory animals have implicated metals associated with particulate matter in adverse health effects. Coal-fired power plants produce particulate residues called *fly ash*. Coal contains metals that vaporize during combustion and then condense on the surface of the ash. Inhaled coal fly ash could be a health hazard because metals solubilized from fly ash within lung cells may cause toxic reactions.

## APPROACH

Dr Ann Aust and colleagues at Utah State University, the University of Utah, the University of California, Davis, and Ford Motor Company hypothesized that transition metals (metals that can participate in possibly toxic oxidative reactions) associated with particulate matter are released within lung epithelial cells and catalyze the formation of reactive oxygen species. Reactive oxygen species can stimulate epithelial cells to produce inflammatory mediators that contribute to lung inflammation and injury. The investigators focused their study on coal fly ash that was produced in the laboratory and separated into four size fractions. (They also performed experiments using particles from gasoline and diesel exhaust, natural soils, and ambient Utah air.) This multifaceted study focused mainly on the ability of iron (the major transition metal in coal fly ash) to produce reactive oxygen species and inflammatory mediators in cultured lung epithelial cells.

## RESULTS AND INTERPRETATION

This study was performed by experienced investigators with demonstrated excellence in the area of metal-catalyzed oxidative stress and particle-associated injury. The study was of high scientific quality, was well conceived and executed, and adds substantially to our knowledge of the biologic properties of particles.

Aust and colleagues found that more iron was released from the smaller particles than from larger ones. They confirmed that soluble extracts of coal fly ash generated reactive oxygen species in vitro and that transition metals were likely responsible. Further, the smallest particles, which were rich in iron, were the most active. The investigators then examined the effects of coal fly ash on human lung epithelial cells in culture. First, they demonstrated that coal fly ash particles entered the cells and stimulated synthesis of the protein ferritin. Ferritin binds iron and is produced in response to increasing iron levels; thus, its presence indicates that iron was released intracellularly and that iron was available to provoke an inflammatory response by forming reactive oxygen species. The investigators obtained indirect evidence for formation of intracellular reactive oxygen species by demonstrating that lung epithelial cells exposed to coal fly ash synthesized the inflammatory mediator interleukin-8. Ferritin and interleukin-8 production were stimulated to a greater degree by smaller

particles than by larger ones. Thus, the investigators provided a plausible connection among the intracellular release of a transition metal from particles, formation of reactive oxygen species, and lung inflammation.

These findings may be important. To confirm their in vitro results, Aust and colleagues will measure ferritin levels in lung tissue and fluids from rats exposed to coal fly ash. The current results that smaller particles had greater effects supports the epidemiologic studies on the adverse effects of fine and ultrafine particles. Other components or properties of particles have also been proposed to cause lung injury; therefore, there may be multiple mechanisms by which inhaled particles produce adverse health effects. Further research to identify particle characteristics (and sources) responsible for particulate matter toxicity is important for developing increasingly effective and appropriate air quality regulations, as noted in HEI Perspectives, *Understanding the Health Effects of Components of the Particulate Matter Mix: Progress and Next Steps.*

# Particle Characteristics Responsible for Effects on Human Lung Epithelial Cells

Ann E Aust, James C Ball, Autumn Hu, JoAnn S Lighty, Kevin R Smith, Ann M Straccia, John M Veranth, and Willie C Young

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