

1
2 BEFORE THE
PUBLIC SERVICE COMMISSION OF WISCONSIN

3
4 Application of Wisconsin Electric Power Company; Wisconsin)
5 Energy Corporation; and W.E. Power, LLC; for a Certificate of) Docket No. 05-CE-130
6 Public Convenience and Necessity for Construction of Three)
7 Large Electric Generation Facilities, the Elm Road Generating)
8 Station, and Associated High Voltage Transmission)
9 Interconnection Facilities to be Located in Milwaukee and)
10 Racine Counties)

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12 _____
13 DIRECT TESTIMONY OF LINDA S. ERDREICH, PATRICK A. HESSEL AND
14 WILLIAM H. BAILEY ON BEHALF OF APPLICANTS
15 _____

16 **Qualifications**

17 **Q. Dr. Erdreich, what is your business address and position?**

18 A. I am a Senior Managing Scientist at Exponent. My office is at 408 Lexington Avenue,
19 New York, NY 10170.

20 **Q. Please describe your educational and business experience.**

21 A. I earned a Ph.D. in Epidemiology and a M.S. in Biostatistics and Epidemiology from the
22 University of Oklahoma Health Sciences Center. I received a B.A. in Biological
23 Sciences and a M.Ed. in Science Education from Temple University. I am a Fellow of
24 the American College of Epidemiology. Throughout my career I have been responsible
25 for assessing the health impact of environmental agents, including chemicals, from
26 scientific research. I worked for several years at the Environmental Protection Agency's
27 (EPA), National Center for Environmental Assessment, where I conducted research to
28 develop methods for setting exposure limits for chemicals to protect human health.
29 These methods have been applied, for example, to the development of standards for

1 hazardous air pollutants and drinking water pollutants. I have worked in scientific
2 research and consulting for 14 years, and during that time, developed standards,
3 evaluated health impacts of co-generation plants, waste sites, various chemicals and
4 physical agents. I have been an adjunct associate professor in the Department of
5 Environmental and Community Medicine at the Robert Wood Johnson Medical School in
6 New Jersey since 1992. I held a similar position at the University of Cincinnati in Ohio
7 from 1982 to 1989.

8 My curriculum vita and those of Dr. Bailey and Dr. Hessel are provided at
9 Exhibit____(Panel-1).

10 **Q. Dr. Hessel, what is your business address and position?**

11 A. I am a Senior Managing Scientist at Exponent. My office address is 2 North Riverside
12 Plaza, Chicago, IL 60606.

13 **Q. Dr. Hessel, can you summarize your educational background?**

14 A. I completed a Bachelor of Science degree in Natural Science from the University of
15 Wisconsin in 1974, a Master of Science degree in Environmental Health from the
16 University of Minnesota in 1978, and a Doctorate in Epidemiology from the University
17 of Pennsylvania in 1982.

18 **Q. Can you describe the work you have done since that time?**

19 A. Following my Ph.D., I worked for five and a half years at the Epidemiology Research
20 Unit in Johannesburg, South Africa, where I was a Senior Epidemiologist. Our research
21 focus was on the respiratory health of mine workers, especially in relation to dust in the
22 workplace. Our research topics included silicosis (a lung disease caused by inhaling
23 silica dust), chronic obstructive pulmonary disease, asbestos-related diseases and lung

1 cancer.

2 I then went to the University of Alberta in Edmonton, Canada. I was a professor
3 in the Department of Public Health Sciences from 1987 through 2002. I was the Director
4 of the Epidemiology Program in the Department and also directed the Epidemiology Unit
5 at the Alberta Asthma Centre.

6 My research focused on occupational lung diseases and asthma. We studied
7 occupational lung diseases among farmers, sawmill workers, workers manufacturing
8 oriented strand board, oil and gas workers and construction workers, among others. The
9 research on asthma was centered on measuring the burden of asthma in children and
10 adults, assessing interventions to improve asthma control, and examining risk factors for
11 asthma. Risk factors that were examined included indoor and outdoor factors as well as
12 genetic influences.

13 **Q. Dr. Bailey, what is your business address and position?**

14 A. My business address is Exponent, 420 Lexington Avenue, Suite 408, New York, NY
15 10170 and I am a Principal Scientist and Director of Exponent's New York office.

16 **Q. What is your educational background?**

17 A. I have a B.A. from Dartmouth College (1966), an M.B.A. from the University of Chicago
18 (1969) and a Ph.D in Neuropsychology from the City University of New York (1975).
19 After completing the requirements for my Ph.D degree I was awarded a two-year
20 postdoctoral fellowship in neurochemistry from the National Institutes of Health at The
21 Rockefeller University.

22 **Q. Please summarize your academic appointments.**

23 A. I have been a visiting research scientist at the Cornell University Medical College since

1 1986. I also have been a visiting lecturer at Rutgers University, the University of Texas
2 (San Antonio), and the Harvard School of Public Health. From 1983-1987, I was head of
3 the Laboratory of Neuropharmacology and Environmental Toxicology at the New York
4 State Institute for Basic Research. For the nine previous years, I was an Assistant
5 Professor and Postdoctoral Fellow in Neurochemistry at The Rockefeller University.

6 **Q. What are your research interests?**

7 A. After many years of research in the laboratory, I have been focusing on the assessment of
8 human exposure to a variety of physical and chemical agents and potential health risks.
9 Some of my research has been directed towards the use of Monte Carlo and other
10 probabilistic methods to characterize uncertainties pertaining to exposure impacts and the
11 determination of “safe” environmental exposures. I am currently directing research
12 projects on the effects of electrical charge on the deposition of aerosols in the respiratory
13 tract and an epidemiologic evaluation of air pollution impacts on community health from
14 mobile sources.

15 **Q. Have you served as an advisor to national scientific and governmental agencies?**

16 A. Yes. I have been an advisor to the National Institutes of Health, the National Science
17 Foundation, the National Institute of Occupational Safety and Health, the U.S.
18 Department of Energy, and the National Institute of Environmental Health Agencies. I
19 have also worked with the International Agency for Research in Cancer and a working
20 group that advises the World Health Organization on risk assessment, perception, and
21 communication.

22 **Q. What is Exponent?**

23 A. Exponent is an engineering and scientific consulting firm that provides solutions to

1 complex technical problems. Our multidisciplinary team of scientists, physicians, and
2 engineers performs in-depth research and analysis in more than 70 technical disciplines.

3 Our clients include a wide range of manufacturers, utilities, insurers, industry
4 groups, government agencies, venture capital companies, and law firms. They request
5 our services to:

- 6 • Assess potential problems related to products, people, property, or the
7 environment
- 8 • Investigate, analyze, and prevent failures and accidents
- 9 • Evaluate compliance with regulatory requirements
- 10 • Assist with product development or product recall
- 11 • Provide technical support during dispute resolution.

12 We offer clients the scientific expertise needed to understand important issues and
13 make sound strategic decisions.

14 **Task and Scope**

15 **Q. What was Exponent asked to do in this case?**

16 A. Exponent was asked to advise Wisconsin Electric on the status of scientific health
17 research relating to the assessment of potential health impacts associated with air
18 emissions from three proposed generating units at the Elm Road Generating Station
19 (ERGS). To provide a broad interdisciplinary perspective, Exponent scientists have
20 pooled their expertise in epidemiology and exposure and risk assessment to address this
21 topic.

22 **Q. What specific emissions did you consider?**

23 A. The combustion process liberates a variety of substances into the air. Our primary focus
24 was on particulate matter (PM) and sulfur dioxide (SO₂) because these are the emissions

1 of coal-fueled power plants that have generated some controversy. We also examined
2 data on oxides of nitrogen (NO_x).

3 **Emission and Exposure Considerations**

4 **Q. What are the sources of PM?**

5 A. PM is a mixture of particles produced by combustion sources including mobile sources
6 (cars, buses, trucks, train, ships), construction equipment, factories, power generators,
7 indoor heating, including wood burning stoves and fireplaces, and burning vegetation.
8 Non-combustion sources include construction sites, roads, and agriculture. These sources
9 produce dusts, pollen, spores, and plant and animal debris. Some components of PM are
10 formed secondarily from gaseous emissions. For example, sulfur dioxide (SO₂) and
11 nitrogen dioxide (NO₂) from motor vehicles, power generation, and other industrial
12 processes undergo chemical changes in the air to form sulfates and nitrates, which are
13 components of PM. The chemical composition and physical properties of various
14 fractions of a given sample of PM are likely to differ from place to place because the
15 fractions may have different sources.

16 **Q. What are the relative contributions of each of these sources to PM emissions?**

17 A. The EPA estimates that mobile sources contribute 62.6% of PM₁₀ in Milwaukee County,
18 and area sources contribute 30.7%. Area sources include industrial processes, waste
19 disposal and treatment, and various stationary sources of fuel combustion. Mobile
20 sources are also an important source of PM₁₀ in Racine County at 55.1%. Point sources
21 in Milwaukee County such as industrial processes and power plants contribute another
22 6.6% from PM_{2.5}. Table 1 summarizes this information for both counties. The relative
23 contributions of mobile sources to levels of PM₁₀, SO₂, and NO₂ are similar across

1 counties. There is a point source category termed “external combustion boilers,” which
 2 includes power plants. The group contributes 4.0% of PM_{2.5} Milwaukee County and
 3 0.13% of PM_{2.5} in Racine County (EPA, 2003).

4
 5 **Table 1. Relative sources of PM and NO₂ in Milwaukee and Racine counties**
 6 (in percent)

	PM ₁₀		PM _{2.5}		NO ₂	
	Milwaukee	Racine	Milwaukee	Racine	Milwaukee	Racine
Mobile Sources	62.6	55.1	56.2	49.5	52.0	70.2
Area Sources	30.7	39.3	30.8	34.3	18.2	19.6
Point Sources	6.6	5.5	12.8	16.1	29.6	10.0

7 Source: USEPA National Emissions Trends (NET) database (2002). [<http://www.epa.gov/air/data/netdb.html>]

8
 9 **Q. What are the point sources of SO₂ emissions in Wisconsin?**

10 A. The sources of these emissions in Milwaukee and Racine counties include 27 different
 11 types of public and private establishments, according to EPA’s emissions inventory
 12 database.

13 The five highest sources of SO₂ are (in order): 1) electric, gas, and sanitary
 14 services; 2) manufacturers of stone, clay, glass, and concrete products; 3) manufacturers
 15 of leather and leather products; 4) primary metal industries; and 5) food and kindred
 16 producers. External combustion boilers contribute 77% of the SO₂ in Milwaukee country
 17 and 3% in Racine County.

18 In addition, the airborne levels of these substances within the state are affected by
 19 more distant regional sources.

20 **Q. Has the EPA developed standards for air quality that are designed to protect**
 21 **against adverse effects of PM, SO₂, and NO₂ on public health?**

22 A. Yes. Under the Clean Air Act, the EPA developed the National Ambient Air Quality

1 Standards (NAAQS) to limit the concentrations of these substances as well as carbon
2 monoxide (CO), ozone, and lead in the air over annual and shorter averaging periods.

3 **Q. What is the goal of the NAAQS?**

4 A. The goal of the NAAQS is to identify pollutants that “may reasonably be anticipated to
5 endanger public health and welfare,” and to issue air quality criteria for them. These
6 criteria are science-based guidelines that serve as the basis for setting permissible
7 exposure levels.

8 **Q. Are the limits set by NAAQS at concentration levels where adverse effects are
9 anticipated?**

10 A. No. The primary standards are established to protect public health, including the health
11 of “sensitive” populations such as asthmatics, children, and the elderly. Primary
12 standards incorporate a margin of safety to help ensure that the limits are adequate to
13 protect public health. “The margin of safety requirement was intended to address
14 uncertainties associated with inconclusive scientific and technical information available
15 at the time of standard setting, as well to provide a reasonable degree of protection
16 against hazards that research has not yet identified” (USEPA, 1997).

17 **Q. In allowing for an adequate margin of safety for exposures to PM_{2.5}, did the EPA
18 consider arguments that epidemiological studies may point to health effects at levels
19 below the proposed standard?**

20 A. Yes, but in setting the NAAQS annual limit for PM_{2.5}, the EPA noted that lower
21 “standards would result in commensurate reductions in health risks only if, in fact, there
22 is a continuum of health risks down to the lower end of the ranges of air quality observed
23 in the key epidemiological studies, and only if the reported associations are, in fact,

1 causally related to PM_{2.5} at the lowest concentrations measured.” The EPA also pointed
2 out “quantitative risk estimates [from epidemiology studies] include significant
3 uncertainty and, therefore, should not be viewed as demonstrated health impacts”
4 (USEPA, 1997).

5 **Q. Has the EPA developed standards for air quality that are designed to protect**
6 **against adverse effects of sulfates on public health?**

7 A. No. However, sulfates, as well as nitrates and other fine particulates, are components of
8 the PM mixture. Therefore, SO₂ and NO₂ from all sources contribute to the formation of
9 particulates.

10 **Q. Will the proposed ERGS generating units comply with the NAAQS?**

11 A. Wisconsin Electric’s air permit application filed with the Wisconsin Department of
12 Natural Resources documents that maximum achievable control technologies applied to
13 the ERGS will minimize emissions and that these emissions would not exceed the
14 NAAQS limits (Burns and McDonnell, 2002). The application also summarizes how the
15 proposed units will comply with Prevention of Significant Deterioration, National
16 Emission Standards for Hazardous Air Pollutants, and other federal and Wisconsin state
17 standards.

18 **Q. How was compliance with the NAAQS determined?**

19 A. The emissions from the proposed generating units and their dispersion under
20 meteorological conditions during a representative four-year period were modeled to
21 determine the maximum concentrations of emissions within an area of 50 km x 50 km.
22 These calculated levels were then combined with background levels to determine
23 maximum expected concentrations.

1 **Q. Are these calculated values useful for describing the exposures of populations to**
 2 **emissions from these generating units?**

3 A. No. The purpose of the calculations is to determine compliance with a standard, not to
 4 characterize exposures where people live. For example, in the permit application, the
 5 maximum expected annual concentration for PM₁₀ produced by the proposed ERGS is
 6 7.98 µg/m³, and occurs within the property boundary. However, in population areas
 7 about three miles from the plant, the maximum expected concentration is lower (0.26
 8 µg/m³) (Burns and McDonnell, 2002).

9 **Q. Will the ERGS contribute to the PM, SO₂, and NO_x exposures of people in**
 10 **Wisconsin?**

11 A. Yes, to a limited extent. The greatest likelihood of exposure would be expected in large
 12 urban areas. Therefore, we calculated the increase in the exposures of population centers
 13 in Milwaukee and Racine due to ERGS operation and compared it to ambient
 14 (background) levels.

15 **Table 2. Predicted contribution of ERGS to annual average levels of PM, SO₂, and**
 16 **NO_x at population centers^{1, 2}**

Pollutant	Background levels ³	Increment contributed by ERGS (µg/m ³)	Percent change from background	Percent of NAAQS Limit from:	
				background	background plus ERGS
Milwaukee					
PM ₁₀	27.0	0.26	<1.0	54.0	54.5
SO ₂	9.2	1.20	13.0	11.5	13.0
NO _x ⁴	31.0	2.34	7.5	31.0	33.4
Racine					
PM ₁₀	27.0	0.26	<1.0	54.0	54.5
SO ₂	7.9	1.20	15.0	9.9	11.4
NO _x ⁴	31.0	1.24	4.0	31.0	32.0

¹ For PM₁₀ and SO₂, three miles or more from ERGS.

² Burns and McDonnell, 2002. At the time this testimony's was submitted, this modeling is being updated.

³ Wisconsin DNR, 2002

⁴ Highest in populated areas within 35 miles.

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2 **Q. How does the increase in annual PM₁₀, SO₂, and NO_x from ERGS compare to**
3 **existing levels in Milwaukee and Racine?**

4 A. Table 2 illustrates that the ERGS operation would produce small increases in the levels of
5 PM₁₀, SO₂, and NO_x in population centers. The table also shows that these increases,
6 when added to background levels, are well below the NAAQS limit for these substances.

7 **Q. Will ERGS have an effect on PM_{2.5}?**

8 A. Since PM_{2.5} is a fraction of the projected annual PM₁₀ levels shown in Table 2, the effect
9 will be very small. In addition, Wisconsin Energy has provided the results of preliminary
10 modeling of peak 24-hour PM_{2.5} levels during the summer of 2010 for conditions
11 resulting in high ozone and PM_{2.5} levels. Keeping in mind the uncertainties in the
12 modeling of PM_{2.5}, these results show that on most days the ERGS will not increase
13 PM_{2.5} levels over a base case scenario. For those days on which the levels are projected
14 to increase, the increase would be hardly measurable, substantially less than 1 µg/m³.
15 The operation of ERGS is also not projected to increase the number of days on which the
16 NAAQS standard for PM_{2.5} is exceeded under the base case scenario.

17 **Data and Approach for Assessing Air Quality Health Risks**

18 **Q. What kind of data do scientists use to assess potential health risks associated with**
19 **air quality?**

20 A. The data are similar to that used to assess potential health risks of other exposures.
21 We evaluate data from epidemiology studies, which relate the health experience of
22 human populations to their personal characteristics and exposures. We also use
23 experimental studies, in which the effects of defined exposures on humans, animals, or

1 cells and tissues are observed in the laboratory.

2 **Q. Why is it important to consider both epidemiology and experimental studies in**
3 **making judgments about health?**

4 A. The two types of studies provide different but complementary information. Both
5 epidemiology studies and experimental studies have drawbacks, but taken together the
6 strengths and limitations balance one another. For example, epidemiological studies of
7 humans include people of varying health conditions and backgrounds, but cannot obtain
8 precise measurements of exposure. Laboratory studies of animals can precisely measure
9 exposures under controlled conditions, but laboratory animals are not humans. When we
10 consider both types of studies, we get more information than if we relied on only one
11 type.

12 **Q. What is the goal of epidemiology studies?**

13 A. Epidemiology studies examine the characteristics of people and their exposures to
14 determine what affects human health and what increases the risk of disease.
15 Epidemiology studies have shown us the risks of smoking, links between animal fat and
16 risk of heart disease, factors that cause certain types of cancer, and the benefits of fruits
17 and vegetables. The objective of environmental epidemiology is to measure and evaluate
18 the associations between exposures to environmental factors (e.g., air pollutants) and
19 health outcomes (e.g., lung disease).

20 **Q. What is the goal of experimental studies?**

21 A. Experimental studies are designed to examine the effect of specific exposures on animals
22 or isolated cells and tissues to determine whether, under highly controlled conditions, the
23 exposure in question is capable of having a biological effect. Exposure levels in these

1 studies are usually higher than those found in human exposure situations and the studies
2 are conducted under conditions where all other known variables are controlled and
3 measured in all study groups. The effects of a specific exposure can be studied in relative
4 isolation from other variables, which is one of the advantages of laboratory research.
5 This means that when the effect of an exposure is studied, the outcome is less likely to be
6 the result of an extraneous factor or systematic bias. The extrapolation from animal
7 results to humans, however, is complicated by differences in anatomy and physiology
8 between animals and humans.

9 **Q. How are the data from epidemiologic and experimental studies evaluated?**

10 A. Assessing health risks involves a thorough review of each study and a comprehensive
11 assessment of the relevant data, including the overall strength, limitations, and
12 uncertainties of each study. All data considered may not be of equal importance. One
13 epidemiology study or even scores of epidemiology studies that are of weaker design
14 may provide only limited evidence for an association between an exposure and a disease.

15 A single study is rarely definitive. No study is perfect or provides all the answers.
16 Repeating a study is important because a reproducible result increases confidence that the
17 observation is correct and not the result of unforeseen error or chance occurrence. But,
18 multiple studies with the same limitations in design or exposure assessment may not
19 necessarily increase one's certainty very much. For example, some cross-sectional
20 studies of air pollution have measured exposure and mortality on the aggregate
21 (population) level, using fixed monitoring sites and mortality rates for the area, to draw
22 inferences about the causes of mortality among populations living in different cities. This
23 is called an ecological analysis and it is recognized that results of studies that use

1 aggregate exposure data and mortality rates may not accurately estimate risks for the
2 individuals in the study.

3 **Q. So, is it appropriate to make judgments about health risks based solely on**
4 **epidemiology studies?**

5 A. Generally no. Epidemiology studies can provide statistical evidence for relationships
6 between exposures and disease, which we then use to develop hypotheses for testing in
7 studies with more powerful designs. Epidemiology studies are highly relevant because
8 they focus on people in their normal daily environment. A primary advantage of
9 epidemiology is that it provides information on people. However, the exposure being
10 studied is usually not under the control of the investigator nor is the diet, genetics,
11 environment, and other exposures, all of which may be related to health. Therefore, all
12 epidemiology studies are to some degree subject to errors that arise from lack of strict
13 control over other variables. Uncontrolled variables may under some circumstances
14 confound the results, that is, produce associations that do not represent a cause and effect
15 relationship.

16 Because of such recognized limitations, epidemiology studies by themselves generally do
17 not provide a sufficient basis to support conclusions about causation. That is why the
18 assessment of health risk also must rely on data from toxicological studies in animals,
19 studies in cells and tissues, and experimental clinical studies.

20 **Q. Can you explain the procedures for assessing scientific data in more detail?**

21 A. The process for systematically evaluating health risks relies on a variety of data,
22 including toxicological and epidemiologic studies, is called health risk assessment. The
23 first step, called *hazard assessment*, includes a comprehensive review of the scientific

1 evidence to determine whether the chemical under investigation can pose a risk to health.
2 Because most chemicals or medicines, even oxygen, can be toxic if the dose is
3 sufficiently high, a *dose-response assessment* is essential to ascertain under what
4 intensity of dose, or exposure, an agent may be toxic.¹ These two steps, hazard
5 assessment and dose-response assessment, help determine how the health outcomes or
6 response are related to the level of exposure. The *exposure assessment* for a specific
7 geographic location or source determines whether the emissions are sufficiently high to
8 produce ambient concentrations that have been shown to affect health. The final step is
9 *risk characterization*, which summarizes the health impacts at the individual and
10 population level that might occur from the exposure from the specific source, based on
11 the determination of hazard and exposure response calculations.

12 *Risk characterization* also includes discussion of the uncertainties in
13 characterizing the public health impact (NRC, 1983).

14 **Q. Is this process similar to that used to develop the National Ambient Air Quality**
15 **Standards (NAAQS)?**

16 A. This approach is used by the EPA's National Center for Environmental Assessment to
17 develop regulatory limits, or "standards." The hazard identification and dose response
18 assessment provide the basis for the standards that limit exposures to contaminants in our
19 environment or the basis for deciding whether a specific chemical or source poses a
20 health risk.

21 The development of standards may also include "risk management" or policy

¹ Epidemiology studies of substances in the environment often cannot obtain measurements of individual dose. Therefore, epidemiologists are generally forced to rely on measures of environmental concentrations to estimate exposure, therefore the term exposure-response.

1 decisions in view of the uncertainties about benefits and costs that often exist. There are,
2 of course, also statutory and policy requirements that affect how the risk assessment
3 process is applied and implemented. For a specific site or facility such as the ERGS,
4 exposure assessment and risk characterization are applied.

5 **Epidemiology Studies**

6 **Q. What do the epidemiology studies indicate about effects of air pollution in general?**

7 A. There is little doubt that air pollution at sufficiently high levels poses a threat to public
8 health and increases mortality. A classic example is London's "Killer Fog" of 1952,
9 which is believed to have caused a 350% increase in the death rate during a four-day
10 period. PM levels were estimated to have exceeded $4,500 \mu\text{g}/\text{m}^3$, hundreds of times
11 higher than ambient levels in the United States.

12 Over one hundred epidemiology studies have examined associations between
13 fluctuations in daily mortality or morbidity (illness) and short-term fluctuations in
14 concentrations of one or more air quality indicators. These studies are called time-series
15 analyses because mortality was evaluated in the same general population (e.g., a city)
16 serially, over a period of time. Many of these studies report a link between higher levels
17 of PM and higher daily mortality.

18 Long-term cohort studies follow a specific group of people, measure exposure in
19 the area where they live, and assess mortality or health outcomes after several years have
20 elapsed. Fewer than a half a dozen such studies have considered exposures to PM or
21 SO_2 . Four cohort studies have considered $\text{PM}_{2.5}$ exposure in the U.S.

22 **Q. Is everyone at equal risk of health effects from air pollution?**

23 A. We know that persons with certain respiratory conditions are more sensitive to some

1 environmental exposures, including those regulated by the NAAQS. Regarding
2 mortality, some evidence suggests that deaths associated with fluctuations in particulate
3 air pollution occur in individuals who are already frail, usually as a result of advanced
4 heart and/or lung disease. If this is the case, short-term fluctuations in air pollutant levels
5 would largely reflect short-term changes in the time of death (e.g., a few hours or days)
6 as a result of higher concentrations of air pollution. Other evidence suggests that the
7 reduction in life expectancy may be more than a few days (Samet et al, 2000a; Zanobetti
8 et al, 2002).

9 **Q. Are there limitations to short-term studies?**

10 A. A major concern is that many of these studies use an ecological design, that is, they do
11 not assess either exposure or disease risk in each individual, but in the aggregate.
12 Another limitation is that short-term studies may include only the impact of potentially
13 minor fluctuations in the timing of death among people whose health is already severely
14 compromised.

15 Another concern has recently emerged. A computer program widely used in the
16 analysis of data from studies of air pollution and health has been found to lead to
17 systematic errors in risk estimates. The use of the default settings in the program has
18 been shown to overestimate the association between health effects and air pollution
19 (Dominici et al, 2002). Compared to a more precise method for analyzing these data the
20 strengths of the associations are overstated and the relative importance of various air
21 pollutants with regard to mortality is misrepresented (Dominici et al, 2002; Steib et al,
22 2003). The researchers who worked on the recent National Mortality, Morbidity and Air
23 Pollution Study (NMMAPS) first discovered this problem. NMMAPS assessed daily

1 mortality and fluctuations in PM₁₀ in the 90 largest cities in the United States (Samet et
2 al, 2000b). After adjustment for the statistical error, the statistical model indicated that
3 the association of mortality with an average daily increase of 10 µg/m³ PM₁₀ was reduced
4 from 0.41% to 0.27%. To date, the overstatement of the magnitude of the associations
5 appears to have occurred in a large number of other studies as well and the reanalysis of
6 those studies are still in progress (Steib et al, 2003).

7 **Cohort Studies of Mortality In Relation to Air Quality**

8 **Q. Why are epidemiology studies of the cohort design particularly informative for**
9 **evaluating associations between air quality and health?**

10 A. There are several reasons to focus on long-term (cohort) studies. One reason is that long-
11 term studies include both short-term (acute) and long-term (chronic) effects (McMichael
12 et al, 1998; Kunzli et al 2001; Peters and Pope, 2002; Rabl, 2003). The most informative
13 cohort studies are those that assess both exposure and risk in each individual in the study
14 and assess cumulative or average exposure over the time period prior to the event
15 (mortality) when exposure could impact disease processes. Another reason to focus on
16 cohort studies is that in general, they are methodologically stronger than ecological
17 studies and cross-sectional studies.

18 **Q. Can you briefly describe the cohort studies that have considered PM or SO₂?**

- 19 A. Four cohorts studies have been conducted in the U.S.:
- 20 • The Six Cities Cohort Study collected data on 8,000 randomly selected adults
21 identified in six cities in the U.S. in 1974. Mortality was assessed 14 to 16 years
22 later. The study focused on PM_{2.5} and sulfates (Dockery et al, 1993).
 - 23 • The American Cancer Society Study collected data on 552,138 people in 151

1 large metropolitan areas that had sulfate measurements, and 50 areas that had data
2 for PM_{2.5} (Pope et al, 1995). The study had over seven years of follow-up time
3 and was later updated to include 16 years of follow-up (Pope et al, 2002).

- 4 • The Adventists Health Study of Smog (AHSMOG) was a study of a cohort of
5 5,900 non-smokers in California and had 15 years of follow-up (1977-1992)
6 (Abbey et al, 1999). This study included PM₁₀, and a follow-up study included
7 PM_{2.5} (McDonnell et al, 2000).
- 8 • The Veterans Cohort Study evaluated 50,000 veterans who had been diagnosed
9 with hypertension and followed them for more than 21 years. Individuals who
10 had been diagnosed with hypertension would presumably be more sensitive to
11 cardiovascular health effects from air pollutants, increasing the sensitivity of the
12 study. This study analyzed 14 different pollutants (Lipfert et al, 2000).

13 **Q. Does each of these studies have strengths as well as limitations?**

14 A. Yes.

15 **Q. What are the strengths of each of these studies?**

16 A. These studies have several strengths and advantages, to varying degrees. Each of these
17 studies measured some form of PM, most measured PM_{2.5} and sulfates. Each followed a
18 relatively large population and assessed disease after a long time period. These studies
19 provide some information on individual risk factors not related to air pollution, to account
20 for the possible confounding effect of other factors that increase the risk of mortality for
21 cardiopulmonary disease, such as age, body weight and cigarette smoking.

22 **Q. Are there limitations in each of these studies?**

23 A. Yes, these studies have several limitations, each of which leads to uncertainty in the

1 results. First, the assessment of exposure to PM and other substances in these
2 epidemiology studies was not based on measurements of individual exposure. The
3 exposure assigned to each individual was a surrogate for their personal exposure based
4 upon measurements taken at fixed monitors outdoors, often in the central region of a
5 large metropolitan area. This is important because when individual exposure is not
6 correctly assessed, it leads to a known source of error in epidemiology studies called
7 exposure misclassification.

8 **Q. What kind of errors may result from exposure misclassification?**

9 A. The errors that result from exposure misclassification depend on the patterns of
10 misclassification. It could lead to underestimation or overestimation of the association,
11 depending on the number of exposure groups and the patterns of misclassification among
12 the groups. Recently, several scientists involved in air pollution research have shown
13 that misclassification of exposures to air pollutants can change the shape of the exposure-
14 response curve. Specifically, misclassification can obscure a threshold if one exists. A
15 threshold is the level of a pollutant below which no relationship is seen between the
16 pollutant and the health outcome. This is important because some scientists have
17 suggested that there is no threshold for the effects of air pollutants, especially PM. This
18 is addressed further below.

19 **Q. Are there other limitations that affect the results of the studies?**

20 A. Yes, except for one study (Lipfert et al, 2000), the number of air pollutants measured is
21 only a fraction of the total number of pollutants that could, or should, have been
22 addressed. The problem of identifying the role of single components when each is
23 associated with multiple other air quality components is particularly difficult. This can

1 introduce confounding because, for example, PM_{2.5} is reported to be correlated with other
2 pollutants that are related to health. None of the long-term studies of PM_{2.5} assessed the
3 effect of carbon monoxide, ozone, or nitrogen dioxide (NO₂), for example. If a
4 component of air pollution, X, is measured, and it is correlated with an unmeasured
5 component, Y, then a statistical link between a disease and component X could arise from
6 a causal link with X, Y or any other correlate. Meteorological conditions, such as wind
7 or inversion conditions tend to affect the concentration of all substances in the air in the
8 same way. Combustion sources, including motor vehicles and power plants, are sources
9 of multiple pollutants as well. Altogether, this makes the task of identifying the
10 individual components of ambient air responsible for the observed associations
11 particularly difficult.

12 Another limitation is that in all of these cohort studies, the individual data on
13 disease risk factors such as smoking, body weight, and alcohol consumption were
14 collected only at the time of entry into the study. Cohort epidemiology studies ideally
15 consider average lifetime exposure or cumulative measures of exposures for potential
16 confounding factors such as cigarette smoking. This data deficiency introduces
17 uncertainty because these individual risk factors may have changed over time, and are
18 strong predictors of mortality risk.

19 **Q. Can you summarize the results reported in the epidemiology studies you reviewed?**

20 A. Positive statistical associations have been reported for higher levels of PM_{2.5} after
21 adjusting for several established risk factors for cardiopulmonary disease (e.g., age,
22 gender, body weight). The reanalyses of the American Cancer Society and Six Cities
23 studies reported results similar to the original studies (Krewski et al, 2000). However, in

1 the study of veterans with hypertension, where an increased risk might be expected,
2 cardiopulmonary mortality was not increased due to exposure to PM_{2.5} (Lipfert et al,
3 2000). The risk estimates in the AHSMOG study were elevated for all cause mortality
4 for PM₁₀ and for PM_{2.5}, but included a margin of error that suggested the increase may be
5 only chance (Abbey et al, 1999; McDonnell et al, 2000).

6 Positive statistical associations were reported for sulfates in the American Cancer
7 Society and Six Cities studies, though neither reported results for SO₂. The update of the
8 American Cancer Society study reported associations between all cause and
9 cardiopulmonary mortality and mean SO₂ (Pope et al 2002). Lipfert et al and Abbey et al
10 did not find an association between all cause or cardiopulmonary mortality and SO₂. The
11 cohort of Adventist non-smokers (McDonnell et al, 2000) did not find an association
12 either, other than increases likely due to chance. SO₂ was associated with lung cancer in
13 females but not males.

14 However, subsequent analyses of the American Cancer Society data illustrated
15 how dependent correlational study results are upon the choice of model and the
16 assumptions underlying the statistical analysis. The model used in the original American
17 Cancer Society analysis assumed that the characteristics of the subjects were
18 independent, i.e., not clustered by location, or spatially independent. Subsequent
19 analyses of sulfate data showed spatial autocorrelation did affect the results. When its
20 influence was removed, a much larger uncertainty in the estimated association between
21 sulfate and mortality became apparent and the association was no longer statistically
22 significant (Burnett et al, 2001).

23 **Q. Can you summarize results reported for NO₂?**

1 A. The American Cancer Society, Six Cities, and Adventist studies reported no evidence for
2 an association of NO₂ with all cause or cardiopulmonary mortality. Lipfert et al reported
3 some evidence for an association between NO₂ and mortality in some time periods, but
4 overall, the results were mixed.

5 **Q. Was the PM_{2.5} or SO₂ from coal-fueled power plants measured in these studies?**

6 A. No. The studies made no attempt to isolate the sources of these exposures. Many other
7 sources besides coal-fueled power plants produce PM_{2.5}. (See Table 1) Combustion
8 sources of PM_{2.5} besides power plants are industries, motor vehicles, burning vegetation,
9 and indoor heating. Non-combustion sources of PM include dusts, pollen, spores, and
10 plant and animal debris. Hence, epidemiology studies, which assess health, based on
11 levels measured in the area where the people live, would include exposures from a
12 variety of unspecified outdoor sources of PM_{2.5}.

13 **Q. If a causal relationship between ambient levels of PM or other substances in the air
14 and cardiorespiratory mortality were to be confirmed, would the risk be uniformly
15 distributed across the population?**

16 A. No. If the relationships were shown to be causal, the PM or SO₂ effects would be limited
17 to persons with pre-existing cardiorespiratory disease or advanced age, not healthy
18 children and adults.

19 **Q. How can we relate such levels of potential health risk to other exposures or
20 conditions that we might be more familiar with, if exposures do not similarly affect
21 persons of all ages in the population?**

22 A. If these relationships are shown to be causal, the best method is to look at reductions in
23 life spans, i.e., the loss of life expectancy (LLE).

1 **Q. Has this method been applied to the evaluation of potential health impacts of PM?**

2 A. Yes, Leksell and Rabl (2001) and Rabl (2003) estimated the LLE for exposure to
3 particulate matter (PM_{2.5} and PM₁₀) using data from the study by Pope et al (1995). The
4 LLE was based upon mortality from cardiopulmonary disease following long-term PM
5 exposures. Their calculations of LLE corrected the exposure assessment of Pope et al to
6 reflect changes in exposure to PM over time.

7 **Q. Can you give us some perspective – again assuming that there is a causal**
8 **relationship between ambient levels of PM and other substances, and cardio**
9 **respiratory mortality – on the estimated reduction in lifespan for such exposures,**
10 **compared to other health related activities?**

11 A. Certainly. For other health related activities such as smoking, lack of exercise, alcohol
12 and obesity, the loss of life expectancy is measured in years, whereas, if you accept
13 causality, loss of life expectancy for exposures to PM or other substances, would be
14 measured in hours or minutes.

15 **Interpretation of Cohort Studies of Mortality and Air Pollution**

16 **Q. How do epidemiologists interpret this variety of results?**

17 A. Epidemiologists typically follow a widely used set of criteria often called “Hill’s
18 Criteria” that serve as guidance for reaching a decision about cause and effect
19 (USDHEW, 1964). These criteria are widely applied in the collective evaluation of
20 statistical associations reported in epidemiologic studies. Table 3 summarizes these
21 criteria and shows that the more closely the data meet these criteria, the more convincing
22 the evidence that observed statistical associations indicate cause and effect.

1

2

Table 3. Hill’s Criteria for Causation

Strength	The stronger the associations between the disease and the exposure or characteristic in question, the more persuasive the evidence.
Consistency	Consistent results across a number of different studies are more convincing than isolated observations.
Dose-response	If an increase in exposure results in an increase in the amount or severity of disease, it is more likely that the exposure is causally related to the disease.
Biological plausibility	Epidemiologic results are much more convincing if there is a known biological mechanism that can explain the effect.
Temporality	The data must provide evidence of an appropriate time period between exposure and the health outcome. The exposure must occur before the observed health outcome, with sufficient time for any induction period related to the disease.

3

4 **Q. Did you apply these criteria to determine whether the results of these epidemiology**
5 **studies indicate that PM_{2.5}, SO₂, or NO₂ causes mortality to increase?**

6 A. Yes. More often than not, these studies reported a statistical association between
7 mortality and long-term exposures to PM_{2.5} and sulfates, which are formed from chemical
8 reactions with SO₂, but measured as part of PM_{2.5}. When examined more closely, other
9 factors however, casts doubt as to whether the positive associations with PM_{2.5} reported
10 in these studies are free from bias and confounding. NO₂ was not reliably associated with
11 mortality in these studies. The possible role of other unmeasured components of ambient
12 air, such as ozone, carbon monoxide, or organic chemicals cannot be fully evaluated.

13 Causality is uncertain if bias and confounding cannot be ruled out in epidemiology

1 studies (IARC, 2002).

2 **Q. Aside from these uncertainties from potential bias and confounding, what does the**
3 **strength of the association indicate?**

4 A. These statistical associations are relatively weak. Weak associations may be correct, but
5 they are more easily explained by biases in the study or confounding exposures than
6 strong associations. Weak associations may be affected by unmeasured risk factors, bias,
7 confounding in the study design, or error in individual exposure assessment. For
8 example, in two of the cohorts previously mentioned, mortality risk was increased in
9 those with less education and there was no association in those whose education included
10 more than high school (Pope et al, 1995; Lipfert et al, 2000; Pope et al, 2002). Education
11 is not known to be linked with exposure to air pollution thus, it is not a likely confounder
12 and this observation is not consistent with the idea of a role of air pollution in mortality.

13 **Q. Do the cohort epidemiology studies describe an exposure-response pattern?**

14 A. This can be examined only in studies that report statistical associations at different levels
15 of exposure. As discussed above, some studies do not report associations between
16 exposure to PM_{2.5} and mortality. For earlier years of the study, the Six Cities study
17 reports higher associations for the city with the highest concentration of PM_{2.5} compared
18 to the city with the lowest concentration and variation between the mortality rates of
19 cities by level of PM_{2.5} (Dockery et al, 1993). The American Cancer Society study
20 reports an exposure-response relationship between mean annual PM_{2.5} levels and
21 mortality rates after adjusting for age, sex and race.

22 **Q. You mentioned biological plausibility as another criteria epidemiologists consider**
23 **when assessing a body of epidemiology studies . What is the experimental evidence**

1 **for adverse effects of exposure to PM or to PM_{2.5}?**

2 A. There are studies in laboratory animals and controlled studies of people in the laboratory
3 that have evaluated effects of exposure to PM. Effects have been observed that may be
4 relevant to increased cardiopulmonary mortality for PM, however, the doses used in some
5 experimental studies are very high, so this information may have limited relevance to
6 human experience at much lower, ambient levels.

7 Many studies have indicated that there are differences in biological responses to different
8 types of particulates and even to different size fractions. More work is needed to identify
9 the size fractions of PM that are toxic and whether the toxicity of PM varies according to
10 its source. No specific mechanism has been identified to support the epidemiologic
11 observation of increased mortality, however, several hypotheses are currently being
12 tested (EPA, 1997).

13 **Q. Can you summarize this evidence with regard to cause and effect from PM_{2.5} and**
14 **SO₂, based on the Hill's criteria?**

15 A. To assess cause and effect, we considered the Hill's criteria and other factors that affect
16 the reliability of the statistical associations in the epidemiology studies. First, the criteria
17 for causality regarding strength and consistency are not met for effects of PM_{2.5} and SO₂,
18 as discussed above. While there were similar results in two of the epidemiology studies,
19 other studies present some puzzling inconsistencies. The laboratory data indicate some
20 toxicity at high doses. However, the toxicity of mixtures may vary depending on the
21 components and measuring selected pollutants correlated with others that may affect
22 health can cause misleading results. Each of the cohort epidemiology studies described
23 measured only a few of the many components of air pollution, and these components may

1 be correlated (Lipfert et al, 2000; Klemm et al, 2000). Given the correlations, the causal
2 component of a mortality increase remains unclear (Lipfert and Wyzga, 1995).

3 **Q. Why is it important to examine the composition of the air pollution exposures in**
4 **epidemiology studies?**

5 A. One of the goals of epidemiology is to find opportunities to improve health by identifying
6 exposures that can be prevented. If we know which components of air pollution are
7 related to health outcomes, it will be possible to better target efforts to reduce these
8 components.

9 **Q. In your summary of the epidemiology studies you stated that no consistent**
10 **associations were reported between SO₂ and mortality at the historical**
11 **environmental levels or with lower levels associated with modern coal-fueled power**
12 **plants. What do experimental studies tell us regarding effects of SO₂?**

13 A. Acute exposure to moderate to high concentrations of SO₂ is known to increase airway
14 resistance (making it more difficult to breathe) in humans and laboratory animals. Animal
15 data are generally consistent with those from clinical studies, which indicate that no
16 significant constriction of airways (bronchoconstriction) develops in healthy subjects
17 exposed to usual ambient levels of SO₂. The human experimental data regarding SO₂
18 exposure provide a relatively clear *threshold* for effects on lung function of about 0.1
19 ppm (262 µg/m³) for asthmatics, particularly when the subjects are also engaged in
20 increased physical activity, and at higher levels for healthy subjects. This level is below
21 24-hour average SO₂ levels nationwide, but short-term peak exposures above this level
22 may occur.

23 **Q. What is meant by the term “threshold”?**

1 A. A threshold is the lowest exposure level that produces a biological response, or health
2 outcome. A threshold is a well-known biological phenomenon that is more reliably
3 identified in toxicological studies in which responses are observed at varying levels of
4 exposure. This contrasts with epidemiologic studies, which rely on less direct measures
5 of exposures to the study population. Knowledge of thresholds is important in
6 establishing allowable exposure concentrations, i.e., setting standards.

7 **Q. Do the epidemiology studies that you reviewed tell us whether or not there is a**
8 **threshold for PM_{2.5}?**

9 A. In the latest report of the American Cancer Society study, the exposure-response curves
10 plotted do not show evidence of a threshold for PM_{2.5}. In these curves, the confidence
11 intervals were broader at lower exposure levels, reflecting greater uncertainty (Pope et al,
12 2002). However, it is premature to interpret these data from a single study as indicating
13 an absence of a threshold.

14 **Q. What is the reason for the reluctance to accept these data as evidence that there is**
15 **no threshold?**

16 A. The reason is based on specific limitations in exposure assessment in these studies. The
17 exposure-response curves are based on ambient concentrations at a central monitoring
18 site, not on individual data. This approach can lead to exposure misclassification, an
19 error that can bias estimates of the effect, and also diminish the ability to observe a
20 threshold if one exists (Brauer et al, 2002). Cakmak et al (1999) conclude that
21 uncertainty in threshold estimates increase with measurement error. Brauer et al (2002)
22 simulated population risks for a series of ambient concentrations to show that the use of
23 surrogate measures that are not highly correlated with personal exposures can obscure a

1 threshold. This may occur for PM_{2.5}, because indoor sources and activities impact
2 personal exposure to PM_{2.5}. Along with the question of causality, the uncertainty about a
3 possible threshold is the focus of ongoing research (Cakmak et al, 1999; Brunekreef and
4 Holgate, 2002).

5 **Q. How is the concept of threshold used in setting standards?**

6 A. For environmental agents that pose a hazard under some circumstances, the usual
7 approach is to set an exposure limit, or standard at a level below the known or estimated
8 threshold. The standard then reflects the level that, based on the research data, is unlikely
9 to lead to effects in most people. To reduce the likelihood of health effects, standards
10 incorporate a margin of safety. Below this level, risk to human health may be negligible
11 or nonexistent (Goldstein and Henefin, 2000). However, for many substances, exposure
12 thresholds are unknown or uncertain.

13 **Q. On the basis of these data, do you believe the ERGS will result in increased
14 mortality and cause health problems?**

15 A. No. The proposed ERGS meets the NAAQS for PM, SO₂, and NO₂. The increase in
16 PM₁₀ from operating the proposed plant is so small an increment as to be insignificant:
17 the modeled estimate at population centers is only 0.26 µg/m³, less than 1% increase over
18 existing levels. No epidemiology data addresses such minute differences. It will
19 contribute only 0.5% of the NAAQS limit. Exposures in the regions will still be less than
20 55% of the NAAQS standard. The modeled estimate for SO₂ is 1.2 µg/m³, a 15%
21 increase over existing levels. It contributes 15% of the NAAQS limit. The modeled
22 estimate for NO_x is 2.3 µg/m³, a 7.5% increase over existing background. It contributes
23 2.4% of the limit (see Table 2).

1 **Studies of Respiratory Health Effects and Coal-Fueled Power Plants**

2 **(CFPP)**

3 **Q. You previously described cohort studies that analyzed mortality in relation to air**
4 **quality and explained that none provide a basis to draw specific conclusions about**
5 **CFPP. Are there other studies that have been done regarding CFPP?**

6 A. Yes, a number of epidemiologic studies have focused specifically on respiratory
7 conditions among people who lived near coal-fueled power plants. The conditions have
8 included diagnosed diseases (e.g., asthma, chronic obstructive pulmonary disease),
9 respiratory symptoms (e.g., cough, phlegm, wheeze, shortness of breath), and lung
10 function. Most studies focused on exposures to sulfur dioxide, however, other pollutants
11 were considered.

12 **Q. Have the results of these studies been summarized anywhere?**

13 A. Not to our knowledge. Therefore, we reviewed the studies that specifically related to
14 coal-fueled power plants.

15 **Q. What were your findings?**

16 A. In studies of highly exposed populations living near older power plants with inadequate
17 or absent pollution control devices, respiratory health effects were generally found.
18 These plants usually burned low grade, high sulfur coal and were often located in valleys
19 – therefore susceptible to temperature inversions that trapped pollutants close to the
20 ground.

21 Studies of populations with lower exposures who lived near more modern power
22 plants have not shown respiratory health effects. These results are consistent across a
23 number of studies. When exposures were below about 100 $\mu\text{g}/\text{m}^3$ of SO_2 , respiratory

1 health effects were not seen.

2 **Q. On the basis of these studies, do you expect the ERGS to have a significant impact**
3 **on the respiratory health of people who live near the plant?**

4 A. No. The projected levels for SO₂ (that include the ERGS plus the other sources) indicate
5 that ambient levels will be well below 100 µg/m³. In addition, they will be well below
6 the NAAQS.

7 **Q. Did any of the studies look at the effects of nitrogen oxides on respiratory health of**
8 **populations around coal-fueled power plants?**

9 A. Although the focus was usually on SO₂, several of the studies examined nitrogen oxides.
10 This was usually nitrogen dioxide, but one study looked at “suspended nitrates.” None of
11 these studies found associations between levels of nitrogen compounds (nitrogen oxides
12 or suspended nitrates) and respiratory problems. In most cases levels of nitrogen oxides
13 were well below ambient standards.

14 **Asthma and Air Pollution**

15 **Q. Is there any scientific basis to assume that emissions from the ERGS would cause**
16 **asthma?**

17 A. No, but concern about asthma is understandable. Physicians in Wisconsin and elsewhere
18 in the U.S. are seeing increasing numbers of people with asthma and media articles
19 frequently mention air pollution as a potential risk factor for asthma.

20 **Q. Are these increases in asthma rates caused by air pollution?**

21 A. It is unlikely. Over the same time period that asthma rates have been increasing, air
22 pollution levels have been decreasing.

23 **Q. Is asthma related to air pollution?**

1 A. There is a substantial body of literature examining the relationships between various air
2 pollutants and respiratory health in general and asthma in particular. The specific
3 pollutants varied by study but usually included one or more subsets of particulate matter,
4 ozone, nitrogen oxides and sulfur dioxide. Many of these studies found relationships
5 between one or more air pollutants and some indicator of respiratory health. This could
6 be a respiratory symptom (e.g., cough) or worsening of asthma (e.g., determined by
7 increases in visits to hospital emergency departments for asthma). Most of these studies
8 were conducted in large cities or metropolitan areas. The specific sources of the
9 pollutants were not identified. The differences in studies of respiratory health related to
10 general urban air pollution and studies near coal-fueled power plants may arise from
11 differences in, for example, the types of particles produced by power plants and those
12 found in ambient urban air.

13 It should be noted that the studies that have found associations between general
14 air pollution and asthma have documented changes in asthma status among people who
15 already have asthma, which is reflected by increased rates of symptoms. Air pollution
16 has not been shown to increase the risk of developing asthma.

17 **Cancer and Coal-Fueled Power Plants**

18 **Q. Do we know anything about cancer and emissions from coal-fueled power plants?**

19 A. The EPA conducted a risk assessment of coal-fueled power plants (French et al, 1997).
20 The assessment considered the emissions of EPA-defined Hazardous Air Pollutants from
21 all of the coal-fueled power plants in the United States (424 plants). They assumed that
22 people living within 50 kilometers of the power plants were exposed to the maximum,
23 modeled, ground-level concentrations of pollutants from the plants, even if no one lived

1 in the area where the maximum concentrations were found. They made several other
2 assumptions that tended to increase the projected risks. Even with these assumptions,
3 they estimated that there would only be about one and a half additional cancer cases in all
4 of the U. S. per year. When they examined the effects of their assumptions, they
5 concluded that a more realistic number would be one additional cancer case every three
6 to six years in all of the U.S.

7 No epidemiologic studies have examined the rate of cancer in people living near
8 coal-fueled power plants, but workers in power plants (who might be expected to have
9 higher exposures to emissions) are not reported to be at greater risk of cancer or other
10 causes of death.

11 **Summary**

12 **Q. What is your overall conclusion regarding PM, SO₂, and NO₂ emissions from the**
13 **proposed ERGS?**

14 A. Based upon the modeling we have reviewed, the ERGS will not cause the NAAQS limits
15 to be exceeded. The NAAQS are the primary means of preventing health impacts of air
16 pollutants in susceptible populations. Our review of studies of modern coal-fueled power
17 plants and epidemiology studies of mortality associated with ambient levels of these
18 substances, does not suggest that the small incremental contribution of the ERGS to
19 ambient levels of these substances would pose detectable health risk.

20 **Q. Does this conclude your pre-filed direct testimony?**

21 A. Yes, it does.

1 **References**

- 2 Abbey, DE; Nishino, N; McDonnell, WF; et al. 1999. Long-term inhalable particles and other
3 air pollutants related to mortality in nonsmokers. *Am. J. Respir. Crit. Care Med.* 159:373-382.
- 4 Brauer, M; Brumm, J; Vedal, S; Petkau, AJ. 2002. Exposure misclassification and threshold
5 concentrations in time series analyses of air pollution health effects. *Risk Anal.* 22:1183-1193.
- 6 Brunekreef, B and Holgate, ST. 2002. Air pollution and health. *The Lancet.* 360:1233-1242.
- 7 Burnett, R; Ma, R; Jerrett, M; et al. 2001. The spatial association between community air
8 pollution and mortality: a new method of analyzing correlated geographic data. *Environ. Health*
9 *Perspect.* 109 Suppl 3:375-380.
- 10 Burns and McDonnell. 2002. An Updated Prevention of Significant Deterioration and Non-
11 Attainment New Source Review, and Maximum Achievable Control Technology Permit
12 Application for Construction and Operation of Elm Road Generating Station: Oak Creek,
13 Wisconsin. Prepared for Wisconsin Electric, October 2002.
- 14 Cakmak, S; Burnett, RT; Krewski, D. 1999. Methods for detecting and estimating population
15 threshold concentrations for air pollution-related mortality with exposure measurement error.
16 *Risk Analysis.* 19:487-496.
- 17 Dockery, DW; Pope, AC; Xu, X; et al. 1993. An association between air pollution and mortality
18 in six U.S. cities. *N. Engl. J. Med.* 329:1753-1759.
- 19 Dominici, F; McDermott, A; Zeger, SL; Samet, JM. 2002. On the use of generalized additive
20 models in time series studies of air pollution and health. *Am. J. Epidemiol.* 156:193-203.
- 21 French, C; Peters, W; Maxwell, B; et al. 1997. Assessment of health risks due to hazardous air
22 pollutant emissions from electric utilities. *Drug Chem. Toxicol.* 20:375-386
- 23 Goldstein, BD and Henifin, M. 2000. Reference Guide on Toxicology. pp. 401-437. In:
24 Reference Manual on Scientific Evidence, Second Edition, Federal Judicial Center 2000.
- 25 International Agency for Research on Cancer (IARC). 2002. IARC Monographs on the
26 evaluation of carcinogenic risks to humans. Volume 80: Static and Extremely Low-Frequency
27 (ELF) Electric and Magnetic Fields. IARC Press, Lyon, France.
- 28 Klemm, RJ; Mason, RM; Heilig, CM; Neas, LM; Dockery, DW. 2000. Is daily mortality
29 associated specifically with fine particles? Data reconstruction and replication of analysis. *J. Air*
30 *Waste Manage. Assoc.* 50:1215-1222.
- 31 Krewski, D. 2000. Reanalysis of the Harvard Six Cities study and the American Cancer Society
32 study of particulate air pollution and mortality: executive summaries and commentary. Health
33 Effects Institute (HEI). July 2000.

1 Kunzli, N; Medina, S; Kaiser, R; et al. 2001. Assessment of deaths attribute to air pollution:
2 should we use risk estimates based in time series or cohort studies? *Am. J. Epidemiol.*
3 153:1050-1055.

4 Leksell, I and Rabl, A. 2001. Air pollution and mortality: quantification and valuation of years
5 of life lost. *Risk Analysis.* 21:843-857.

6 Lipfert, FW and Wyzga, RE. 1995. Uncertainties in identifying responsible pollutants in
7 observational epidemiology studies. *Inhal. Toxicol.* 7:671-689.

8 Lipfert, FW; Perry, HM; Miller, JP; et al. 2000. The Washington University-EPRI veterans'
9 cohort mortality study: preliminary results. *Inhal. Toxicol.* 12:41-73.

10 McDonnell, WF; Nishino-Ishikawa, N; Petersen, FF; Chen, LH, Abbey, DE. 2000.
11 Relationships of mortality with the fine and coarse fractions of long-term ambient PM₁₀
12 concentrations on nonsmokers. *J. Expo. Anal. Environ. Epidemiol.* 10:427-436.

13 McMichael, AJ; Anderson, HR; Brunekreet, B; Cohen, AJ. 1998. Inappropriate use of daily
14 mortality analyses to estimate longer-term mortality effects of air pollution. *Inter. J. Epidemiol.*
15 27:450-453.

16 National Research Council (NRC). 1983. Risk assessment in the federal government: managing
17 the process. National Academy Press, Washington DC.

18 Peters, A and Pope, CA. 2002. Cardiopulmonary mortality and air pollution. *The Lancet.*
19 360:1184-1185.

20 Pope, CA; Thun, MJ; Namboodiri, NM; et al. 1995. Particulate air pollution as a predictor of
21 mortality in a prospective study of U.S. adults. *Am. J. Respir. Crit. Care Med.* 151:669-674.

22 Pope, CA; Burnett, RT; Thun, MJ; et al. 2002. Lung cancer, cardiopulmonary mortality, and
23 long-term exposure to fine particulate air pollution. *J. Am. Med. Assoc.* 287:1132-1141.

24 Rabl, A. 2003. Interpretation of air pollution mortality: number of deaths or years of life lost?
25 *J. Air Waste Manage. Assoc.* 53:41-50.

26 Samet, JM; Dominici, F; Curriero, F; Coursac, I; Zeger, SL. 2000a. Fine particulate air
27 pollution and mortality in 20 US. cities. *N. Engl. J. Med.* 343:1742-1749.

28 Samet, JM; Zeger, SL; Dominici, F; et al. 2000b. National morbidity, mortality, and air
29 pollution. part I: morbidity, mortality, and air pollution in the United States. *Res Rep Health Eff.*
30 *Inst.* 94 Pt 1:5-14.

31 Stieb, DM; Judek, S; Burnett, RT. 2003. *J. Air Waste Manage. Assoc.* 53:258-261.

32 U.S. Environmental Protection Agency (USEPA). 2003. National Emissions Trend Database.
33 Website: <http://www.epa.gov/air/data/netdb.html>.

- 1 U.S. Environmental Protection Agency (USEPA). 1997. Federal register part II: national
2 ambient air quality standards for particulate matter; final rule. 40 CFR, Part 50. Prepared by
3 USEPA, July 18, 1997.
- 4 U.S. Department of Health, Education, and Welfare (USDHEW). 1964. Smoking and health:
5 report of the advisory committee to the surgeon general of the public health service. D. Van
6 Nostrand Company, Inc.: Princeton, New Jersey.
- 7 Wisconsin Department of Natural Resources (WDNR). 2002. Website:
8 <http://www.dnr.state.wi.us/>.
- 9 Zanobetti, A; Schwartz, J; Samoli, E; et al. 2002. The temporal pattern of mortality responses to
10 air pollution: a multicity assessment of mortality displacement. *Epidemiology*. 13:87-93.