# Public Health Impacts of Old Coal-Fired Power Plants in Michigan

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## Acknowledgements

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## Who We Are

Since 1980, the Michigan Environmental Council has been at the forefront of efforts to protect our Great Lakes, promote sustainable cities, safeguard public health and establish clean energy policies for a more vibrant economy. Representing over 60 member organizations throughout the state, MEC provides agenda-setting leadership at the State Capitol and with our Congressional delegation in Washington.

## About the Author

Environmental Health & Engineering, Inc. (EH&E) is a professional services firm established in 1987 to provide businesses and institutions with a reliable resource for environmental consulting and engineering services. The company is composed of 75 highly trained individuals based in Needham, Massachusetts, who share a passion and enthusiasm for excellence.

This project for the Michigan Environmental Council was led by David L. MacIntosh, Sc.D., C.I.H. In addition to his role as a Principal Scientist and Associate Director of Advanced Analytics and Building Science at EH&E, Dr. MacIntosh is an Adjunct Associate Professor of Environmental Health at the Harvard School of Public Health where he teaches a course on environmental exposure assessment to masters and doctoral degree students. Dr. MacIntosh also is a technical advisor to the World Health Organization and is the first-draft author of several publications for that organization including a Toolkit for Human Health Risk Assessment which was published in 2011.

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Prepared For:

**Michigan Environmental Council**
**Lansing, Michigan 48912**

June 24, 2011

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## LIST OF ABBREVIATIONS AND ACRONYMS

| | |
|---|---|
| ACS | American Cancer Society |
| AHSMOG | Adventist Health and Smog |
| BenMAP | Environmental Benefits Mapping and Analysis Program |
| C-R | concentration-response |
| COI | cost-of-illness |
| COMEAP | Committee on the Medical Effects of Air Pollutants |
| CRDM | Climatological Regional Dispersion Model |
| EGU | electricity generating unit |
| EH&E | Environmental Health & Engineering, Inc. |
| EPA | U.S. Environmental Protection Agency |
| ER | emergency room |
| GIS | Geographic Information System |
| HIA | health impact assessment |
| ICD-9 | International Statistical Classification of Diseases |
| MRAD | minor restricted activity days |
| MW | megawatt |
| NAAQS | National Ambient Air Quality Standards |
| NAS | National Academy of Sciences |
| NCHS | National Center for Health Statistics |
| NEI | National Emissions Inventory |
| NHDS | National Hospital Discharge Survey |
| $NO_X$ | nitrogen oxides or oxides of nitrogen |
| S-R | Source Receptor |
| $PM_{2.5}$ | particulate matter that is 2.5 micrometers or smaller in size |
| $PM_{10}$ | particulate matter that is 10 micrometers or smaller in size |
| $SO_2$ | sulfur dioxide |
| TSP | total suspended particulates |
| VA | Veterans Administration |
| VSL | value of statistical life |
| U.S. | United States |
| WTP | willingness-to-pay |
| $\mu g/m^3$ | micrograms per cubic meter |

# 1.0 EXECUTIVE SUMMARY

Environmental Health and Engineering Inc. (EH&E) estimated the public health impacts expected due to airborne particulate matter less than 2.5 microns in aerodynamic diameter ($PM_{2.5}$) attributable to emissions from nine coal-fired electricity generating units (EGUs) which began operation in the State of Michigan between 1949 and 1968. A recent study released by the National Academy of Sciences found that the dirtiest (strongly correlated with the oldest) 10% of the plants, accounted for 25% of the generation and 43% of the public health damages borne by people living in the United States (U.S.) (NRC, 2010).

We estimate the Michigan-specific health-related damages associated with $PM_{2.5}$ emissions from the nine coal-fired facilities to be $1.5 billion annually and the national impacts from those same facilities to $5.4 billion annually. The nine Michigan EGUs included in this analysis are listed in Table 1.1.

**Table 1.1** Electricity Generating Units Included in Assessment of Public Health Impacts Associated with Air Pollutant Emissions from Older Coal-fired Power Plants in Michigan

| Electricity Generating Unit | Parent Company | County | Initial Startup Year | Nameplate Capacity of Modeled Units (MW) | Approximate Coal Consumption (tons/year)* |
|---|---|---|---|---|---|
| BC Cobb** | CMS Energy | Muskegon | 1956 | 313 | 900,000 |
| Dan E Kar**n | CMS Energy | Bay | 1956 | 544 | 1,500,000 |
| JC Weadock** | CMS Energy | Bay | 1955 | 313 | 800,000 |
| Harbor Beach | DTE Energy | Huron | 1968 | 121 | 100,000 |
| JH Campbell** | CMS Energy | Ottawa | 1962 | 669 | 1,600,000 |
| JR Whiting | CMS Energy | Monroe | 1952 | 345 | 1,000,000 |
| River Rouge | DTE Energy | Wayne | 1957 | 651 | 1,300,000 |
| St. Clair | DTE Energy | St. Clair | 1953 | 1,547 | 3,000,000 |
| Trenton Channel | DTE Energy | Wayne | 1949 | 776 | 1,700,000 |

MW megawatts

* Based on conversion of million BTUs to tons of bituminous coal published by the U.S. Department of Energy. http://www.energystar.gov/ia/business/tools_resources/target_finder/help/Energy_Units_Conversion_Table.htm

** Values do not include boilers that began operation after 1969.

Our estimates were derived by applying a widely accepted methodology for conducting health impact assessments of air pollutants which is used by the U.S. Environmental Protection Agency (EPA) to evaluate policy options. Our analysis is grounded in the scientific conclusions reached by EPA and its independent scientific advisors that inhalation of $PM_{2.5}$ over both short and long periods of time is a cause of cardiovascular effects, including heart attack and the associated mortality (EPA, 2009; CASAC, 2010).

Particulate matter, also known as particle pollution or PM, is a complex mixture of extremely small particles and liquid droplets. Particle pollution is made up of a number of substances, including acids (such as nitrates and sulfates), organic chemicals, metals, and soil or dust particles. Fine particulate matter or $PM_{2.5}$ is well-known by health professionals because of its connection to a wide variety of health ailments. Combustion of fossil fuels is the primary source of $PM_{2.5}$ in the atmosphere. Air pollutants generated by burning coal to generate electricity are the largest components of $PM_{2.5}$ in the eastern U.S.

Fine particles can affect the heart and lungs and cause serious health effects. When inhaled by people, some $PM_{2.5}$ particles deposit along the respiratory tract, while others penetrate deeply into the lung where they can enter the bloodstream. These particles (i) aggravate the severity of chronic lung diseases and impair airways function, and (ii) cause inflammation of lung tissue which results in the release of chemicals that impact heart function, and leads to changes in blood chemistry that produces clots which can cause heart attacks (EPA 2010a).

Health impact assessments of the type we conducted for selected EGUs in Michigan combine information on changes in air pollutant concentrations, the relationship between air pollutant concentrations and the risk of an adverse health outcome, the baseline incidence of each health outcome, and the size of the population exposed to the air pollutants.

Our analysis considered the following health outcomes: premature mortality, hospital admissions for cardiovascular and respiratory disease, emergency room (ER) visits for asthma, asthma exacerbation, chronic bronchitis, and minor restricted activity days (MRADs). The annual number of cases of each health outcome associated with air

pollutant emissions from the Michigan coal-fired EGUs facilities was estimated for each county in the continental U.S. By conducting our analysis at the resolution of counties rather than a larger geographic area (e.g., state), we maintained spatial relationships among population sizes, baseline incidence of disease, and air quality important for ascertaining a reasonable estimate of public health impacts associated with pollutant emissions from the eleven modeled Michigan EGUs.

As shown in Table 1.2, 180 premature deaths per year in Michigan are expected to be associated with particle emissions from the nine modeled Michigan EGUs. Our estimates of the annual morbidity-related impacts (e.g., hospital visits, asthma attacks, etc.) in Michigan associated with air pollutant emissions from the coal-fired Michigan EGUs are summarized in Table 1.2 as well. Approximately 230 hospital admissions or ER visits and 68,000 asthma exacerbations in Michigan each year are estimated to be associated with these older Michigan-based coal-fired facilities.

**Table 1.2** Annual Mortality and Morbidity Impacts in Michigan Associated with Air Pollutant Emissions from Older Coal-fired Electricity Generating Units in Michigan

| Outcome | Cases |
|---|---|
| Premature mortality | 180 |
| Cardiovascular hospital admissions | 38 |
| Respiratory hospital admissions | 55 |
| Chronic bronchitis | 76 |
| Asthma emergency room visits | 140 |
| Asthma exacerbations | 68,000 |
| Minor restricted activity days | 72,000 |

Public health impacts across the entire U.S. associated with emissions from the Michigan EGUs are presented in Table 1.3. The number of adverse health outcomes annually includes 660 premature deaths, 360 hospital admissions for cardiovascular or respiratory disease, 450 ER visits for asthma, 250,000 asthma exacerbations (e.g., asthma attacks), 280 new cases of chronic bronchitis, and approximately 260,000 minor restricted activity days.

**Table 1.3** Annual Mortality and Morbidity Impacts in the Continental United States Associated with Particulate Matter-related Air Pollutant Emissions from Older Coal-fired Electricity Generating Units in Michigan

| Outcome | Cases |
|---|---|
| Premature mortality | 660 |
| Cardiovascular hospital admissions | 150 |
| Respiratory hospital admissions | 210 |
| Chronic bronchitis | 280 |
| Asthma emergency room visits | 450 |
| Asthma exacerbations | 250,000 |
| Minor restricted activity days | 260,000 |

We estimate the annual national and Michigan-specific health-related damages associated with $PM_{2.5}$ emissions from the nine coal-fired EGUs to be $5.4 billion and $1.5 billion respectively. Table 1.4 displays the EGU specific impacts.

**Table 1.4** Valuation of Annual Plant-specific Public Health Damages Associated with Contributions of Older Coal-fired Power Plants in Michigan to Fine Particle Levels in Air

| Electricity Generating Units | Economic Value ($ million) | |
|---|---|---|
| | Michigan* | Continental U.S. |
| BC Cobb | 68 | 450 |
| Dan E Karn / JC Weadock | 120 | 720 |
| Harbor Beach | 11 | 63 |
| JH Campbell | 150 | 700 |
| JR Whiting | 560 | 1,040 |
| River Rouge | 340 | 780 |
| St. Claire | 65 | 560 |
| Trenton Channel | 140 | 1,080 |
| Total | 1,500 | 5,400 |

A significant portion of the public health impacts are anticipated to be realized in the states of the upper Midwest and Northeast—Illinois, Indiana, Ohio, Pennsylvania, New York—states that are proximate or downwind to the coal-fired EGUs included in this assessment. As an illustration of this point, Figure 1.1 contains a map that provides the number of premature deaths associated with the modeled EGUs in each state. Health impacts for the morbidity endpoints are distributed similarly across the states.

The basis of the estimated public health impacts is the increment of $PM_{2.5}$ in each county that is estimated to result from emissions from the Michigan coal-fired EGUs. The

incremental annual average exposure to $PM_{2.5}$ is centered in Michigan, extending east to the Atlantic Ocean, and as far west as Colorado. An illustration of the geographic distribution of the incremental annual average $PM_{2.5}$ levels associated with emissions of $PM_{2.5}$ and particle precursors from the Michigan coal-fired EGUs is presented in Figure 1.2.

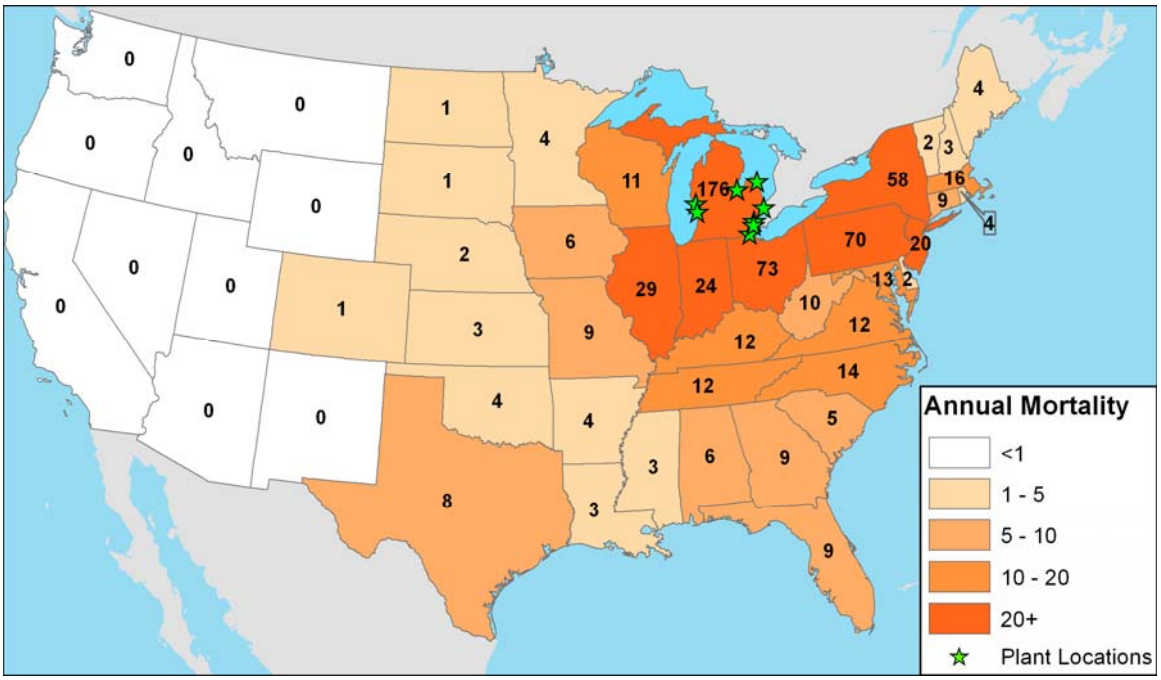


**Figure 1.1** Estimated Number of Fine Particle-related Premature Deaths for Each Year Associated with Emissions from Older Coal-fired Electricity Generating Units in Michigan

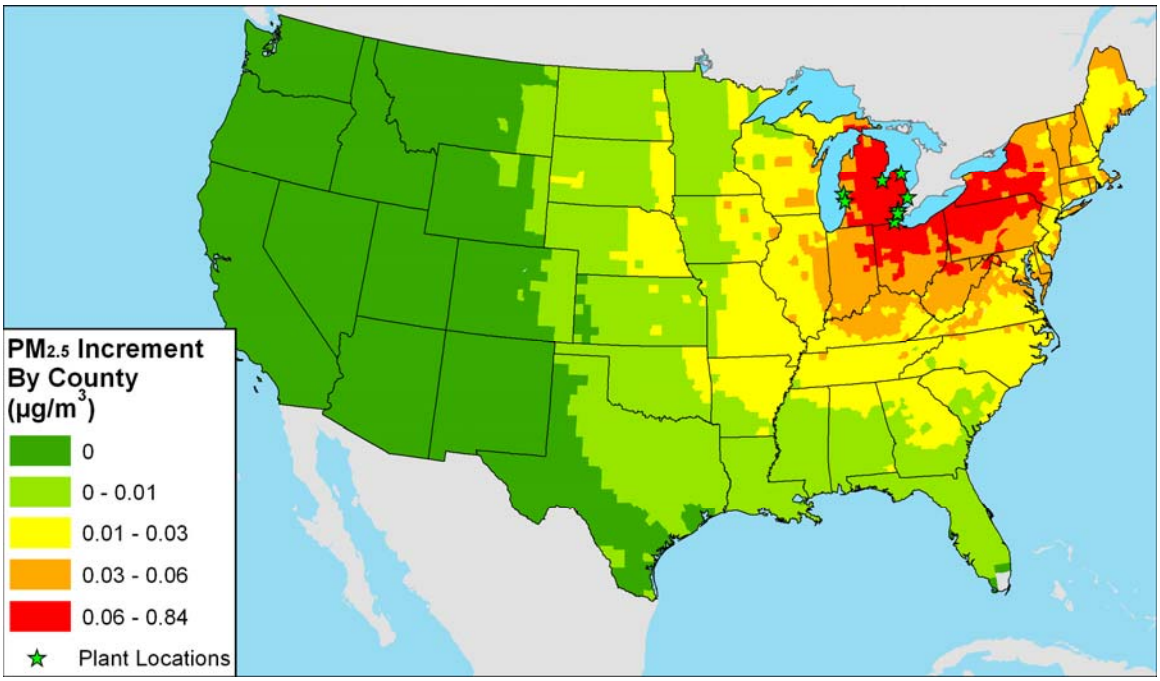


**Figure 1.2** Estimated Annual Average Increment of Fine Particle Concentrations ($\mu g/m^3$) Associated with Older Coal-fired Electricity Generating Units in Michigan

The reasonableness of our estimates is informed by the widely accepted nature of our methodology and the similarity between our outputs and those from related studies. The methodology used has been widely vetted in the scientific and regulatory community and is used by the EPA to report to the U.S. Office of Management and Budget and others on the benefits of various air pollution control programs. The method used to incorporate the more recent literature on air pollution associations with premature mortality and morbidity is widely accepted and used as a matter of routine in meta-analysis of multiple studies on various topics, not just health effects of air pollution. The method to predict $PM_{2.5}$ concentrations, the Source Receptor Matrix is an EPA model that has been used in a number of regulatory impact assessments. The body of epidemiological literature for health effects of particulate matter is large and robust, and is supported by good mechanistic understanding of how particulate matter can influence human health. Had we included the population of Southeast Canada and used forecasted future population values for 2010 rather than population information from the 2000 Census, the estimates of public health impacts would have been substantially greater.

Additionally, consideration of pollutants other than $PM_{2.5}$, such as mercury, could have further increased our public health impact estimates. Electricity generating stations

powered by coal account for 58% of mercury released to air in Michigan from point sources. Coal-fired power plants can be significant contributors to deposition of mercury on soil and water, reportedly accounting for 70% of the mercury present in rainfall in eastern Ohio (Keeler et al., 2006). Mercury that deposits to the earth's surface from air can make its way into waterways where it is converted by microorganisms into methylmercury, a highly toxic form of mercury (Grandjean 2010). As these microorganisms are eaten by larger organisms, methylmercury concentrations increase with each successive level of the food chain, in a process called bioaccumulation. The large and long-lived predators of marine and freshwater ecosystems, including many fish favored by consumers in the U.S., end up with the highest methylmercury concentrations. As a result, consumption of fish and other aquatic organisms is the predominant pathway of exposure to mercury. The amount of mercury in people correlates with typical fish intake (MacIntosh et al., 1997; Carta et al., 2003; Mozaffarian and Rimm, 2006). Methylmercury is a potent neurotoxin, and high accumulation in humans is a cause of brain damage, while lower body burdens are associated with impairment of people's ability to learn and fine motor control, and may be a factor in heart disease. Because of concern about the effect of methylmercury on the developing brain, numerous government agencies have issued recommendations on fish consumption to minimize dietary intake of mercury for women who are or may become pregnant, nursing mothers, and young children. One in six women in Michigan are reported to have body burdens of mercury that exceed values recommended by health protective agencies.

In summary, the approach and inputs to our calculations of the premature mortality and annual morbidity associated with particulate emissions from coal-fired Michigan EGUs are reasonable. Premature mortality and morbidity attributable to fossil fuel-related particulate matter persists in the U.S. and reduction in emissions from coal-fired power plants will have health benefits of the magnitude estimated within this report.

We have estimated that the nine modeled coal-fired EGUs release approximately 11,300 tons of $PM_{2.5}$ and 194,000 tons of sulfur dioxide and nitrogen oxides per year. As a consequence, millions of people across the country, but especially in the upper Midwest and Northeast are exposed to $PM_{2.5}$ and other pollutants. Each year these facilities operate, approximately 180 lives are lost prematurely in Michigan. But since the

byproducts of coal-fired power plant emissions are dispersed over great distances, over 480 additional lives are lost annually in 39 other states influenced by these sources. Furthermore, we estimate that 30 lives per year would be lost prematurely in impacted areas of Canada. Other important health impacts, such as asthma morbidity, chronic bronchitis, hospital admissions due to pollutant exposures are associated with emissions from these EGUs.

# 2.0 INTRODUCTION

The purpose of this report is to present our assessment of the health impacts associated with changes in $PM_{2.5}$ concentrations estimated to result from a reduction in air pollutant emissions from nine coal-fired electricity generating units (EGUs) operated in the state of Michigan. Attributes of the nine EGUs are reported in Table 2.1, with a combined nameplate capacity of 5,279 megawatts (MW), equivalent to approximately 20 percent of Michigan's total capacity. The general approach and specific methods that we used in this assessment are detailed in Section 3. The results of our health impact assessment are presented in Section 4. Conclusions drawn from our work are summarized in Section 5 and detailed results are presented in Appendix A and Appendix B. A description of EH&E is presented in Appendix C.

**Table 2.1** Power Plants Included in Assessment of Public Health Impacts Associated with Air Pollutant Emissions from Older Coal-fired Electricity Generating Units in Michigan

| Electricity Generating Unit | Parent Company | County | Initial Startup Year | Nameplate Capacity of Modeled Units (MW) | Approximate Coal Consumption (tons/year)* |
|---|---|---|---|---|---|
| BC Cobb** | CMS Energy | Muskegon | 1956 | 313 | 900,000 |
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| JH Campbell** | CMS Energy | Ottawa | 1962 | 669 | 1,600,000 |
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| Trenton Channel | DTE Energy | Wayne | 1949 | 776 | 1,700,000 |

MW megawatts

* Based on conversion of million BTUs to tons of bituminous coal published by the U.S. Department of Energy. http://www.energystar.gov/ia/business/tools_resources/target_finder/help/Energy_Units_Conversion_Table.htm

** Values do not include boilers that began operation after 1969.

# 3.0 METHODS

## 3.1 GENERAL APPROACH

In assessing public health benefits from a reduction of population exposure to $PM_{2.5}$, we used a health impact assessment (HIA) approach published in numerous peer-reviewed scientific publications (Levy et al. 1999; Spadaro and Rabl 2001; Levy and Spengler 2002; Levy et al. 2002; Levy et al. 2003; Hubbell et al. 2005; Chestnut et al. 2006; Ostro et al. 2006), used by the EPA in regulatory assessments (EPA 1997; EPA 1999a; EPA 1999b; EPA 2004b; EPA 2005; EPA 2010b; EPA 2010c), and endorsed by the EPA Science Advisory Board (EPA 2004a), the U.S. Office of Management and Budget (EPA 1997), and the National Academy of Sciences (NRC 2002).

In a HIA, public health benefits of reduced air pollutant emissions are calculated from information on an anticipated change in exposure to harmful pollutants, concentration-response relationships for specific types of illness, the baseline incidence of those illnesses, and the number of exposed people at risk of the pollutant-related illness.

In a recent application of the HIA approach, the EPA estimated the public health benefits of reduced air pollutant emissions anticipated to result from implementation of the Clean Air Interstate Rule that has been proposed by the Agency (EPA 2005). Similarly, the EPA used the HIA approach for a 2004 assessment of delayed mortality and reduced morbidity associated with lower emissions of air pollutants from non-road diesel vehicles such as trains (EPA 2004b). The EPA has even codified the HIA methodology in a software tool known as the Environmental Benefits Mapping and Analysis Program (BenMAP).[1] One of the first HIA for air pollutants—specifically, particulate matter, was published by the EPA in 1997 (EPA 1997).

HIAs have also been published in the peer-reviewed scientific literature, of which a number have been co-authored by EH&E scientists and collaborators. For example, the robustness and sensitivity of the HIA approach in studies was evaluated for the number of premature deaths and cases of non-fatal disease associated with sulfur dioxide ($SO_2$),

[1] See http://www.epa.gov/ttn/ecas/benmodels.html, Benefits Analysis Models/Tools, U.S. Environmental Protection Agency.

nitrogen oxides ($NO_X$), and primary $PM_{2.5}$ emissions from coal-fired power plants in Massachusetts, Illinois, the District of Columbia, Maryland, Virginia, and Georgia (Levy et al. 1999; Levy and Spengler 2002; Levy et al. 2002; Levy et al. 2003). We have used the HIA approach to estimate the public health benefits of energy savings associated with increasing the use of insulation in new housing from current practice to the latest international energy conservation standards (Nishioka et al. 2002). In addition, we have published methodologies for deriving specific inputs to HIA such as baseline concentration-response functions for particulate matter (Levy et al. 2000), estimates of population exposure to emissions from point sources such as power plants (Zhou et al. 2003), and ability of air cleaners to control residential exposures to $PM_{2.5}$ (MacIntosh et al. 2009).

## 3.2 FINE PARTICLES AND DISEASE

The first step in our application of the HIA approach to emission impacts from Michigan coal-fired EGUs involves determining the appropriate pollutants to incorporate into the analysis. Based on reviews of past air pollution benefit-cost analyses conducted by the EPA (EPA 1997; EPA 1999b; EPA 2004b; EPA 2005; EPA 2010b), we concluded that $PM_{2.5}$ would contribute to a significant fraction of the total health impacts associated with emissions from the nine coal-fired EGUs. Therefore, we focused our estimates on pollutants that would influence ambient concentrations of $PM_{2.5}$. For $PM_{2.5}$, there is strong and consistent evidence supporting health effects at current levels of exposure in the U.S.

For example, the most recent Integrated Science Assessment for Particulate Matter contains over 2,000 pages of documentation regarding exposures to and health risks from $PM_{2.5}$ (EPA 2009). This includes not only epidemiological evidence, which forms the centerpiece of our concentration-response functions as described in Section 4.6, but also extensive toxicological evidence. While it is implausible to summarize the entirety of the information reported within this document, it is important to recognize that the cardiovascular and respiratory effects observed in human populations have been observed in laboratory studies of humans and animals as well. Our opinions about the health effects of particle matter are consistent with the key insights summarized in the Integrative Health and Welfare Effects Overview (Chapter 2) of the EPA Integrated

Science Assessment for Particulate Matter and with the opinions of the EPA Clean Air Science Advisory Committee, a group of renowned air pollution and health scientists from outside of the federal government.

To summarize, while other air pollutants from power plants have anticipated health consequences, previous assessments (EPA 1997; EPA 1999a; EPA 1999b; EPA 2004b; EPA 2005) have demonstrated that most of the public health burden (especially once placed in economic terms) can be attributed to $PM_{2.5}$. Thus, given the strength of the literature and the likelihood that a significant portion of the health risks from power plant emissions are associated with $PM_{2.5}$, we focus our efforts on understanding exposure to and health risks from $PM_{2.5}$. The precise list of health outcomes considered is provided in Section 4.6, but includes both premature mortality and morbidity endpoints of varying severity (ranging from hospital admissions to minor symptoms).

To assess the health impacts of $PM_{2.5}$ exposure due to power plant pollutant emissions, two facts must be true. First, these pollutants must have effects over the range of exposures that have occurred during the epidemiologic studies and are anticipated to occur in the future (i.e., concentration thresholds are not relevant). Second, for $PM_{2.5}$, it must be the case that the $PM_{2.5}$ coming from the power plants under study has similar toxicity to the $PM_{2.5}$ being evaluated in published epidemiological studies.

Addressing the first question, it is first important to realize that the National Ambient Air Quality Standards (NAAQS) are not necessarily population risk thresholds. Although the NAAQS are meant to be protective of public health with an adequate margin of safety, they are not static, and are continually being revised as new health evidence becomes available. More generally, it is important to understand that the NAAQS is not meant to be a zero-risk level (a concentration below which no one in the population would exhibit adverse effects). Quoting from the EPA, “The Act does not require the Administrator to establish a primary NAAQS at a zero-risk level, but rather at a level that reduces risk sufficiently so as to protect public health with an adequate margin of safety” (EPA 1997).

We can look directly at the health evidence to determine whether health effects are anticipated at current levels of exposure. The American Cancer Society cohort study (Pope et al. 2002) did not find a threshold for $PM_{2.5}$ health effects. Instead, that study

found effects for concentrations below 10 micrograms per cubic meter ($\mu g/m^3$) on an annual average basis. More broadly, some time-series studies have examined the possibility of non-linearity in the relationship between particulate matter exposures and mortality (Daniels et al. 2000; Schwartz et al. 2002; Dominici et al. 2003). All concluded that a linear no-threshold model was the best fit to the observed data, explainable by the substantial variability in individual susceptibilities. For example, an analysis of data from the Six Cities Study found an essentially linear relationship between daily mortality and $PM_{2.5}$ concentrations down to 2 $\mu g/m^3$ (Schwartz et al. 2002).

Thus, as summarized in the EPA Integrated Science Assessment (EPA 2009) studies that examined the relationship between mortality and short- and long-term exposures to PM have "consistently found no evidence for deviations from linearity or a safe threshold" (Daniels et al. 2004; Schwartz 2004; Samoli et al. 2005; Schwartz et al. 2008).

The issue of a threshold for PM-mediated health effects has been addressed by the National Academy of Sciences (NAS) as well. In 2000, Congress directed the Agency to ask the NAS to review the methodologies used by the EPA to perform benefit-cost analyses for air quality and to recommend a common methodology to be used in all future analyses by the Agency (U.S. Senate 2000). In its ensuing report (NRC 2002), the National Research Council (the research branch of the NAS) stated "For pollutants such as $PM_{10}$ and $PM_{2.5}$, there is no evidence for any departure of linearity in the observed range of exposure, nor any indication of a threshold."

Regarding the second case, the issue is whether PM from power plants has different (either larger or smaller) health impacts from particulate matter from all sources. A quotation from the EPA Integrated Science Assessment (EPA 2009) sheds some light on this question, synthesizing the available evidence:

"Overall, the results indicate that many constituents of PM can be linked with differing health effects and the evidence is not yet sufficient to allow differentiation of those constituents or sources that are more closely related to specific health outcomes. These findings are consistent with the conclusions of the 2004 Air Quality Criteria Document review for PM (EPA 2004a) (i.e., that a number of source types, including motor vehicle

emissions, coal combustion, oil burning, and vegetative burning, are associated with health effects)."

Nonetheless, the EPA considered the potential for differential toxicity of $PM_{2.5}$ as part of a regulatory impact analysis for off-road vehicles. In that report, the EPA stated that although the chemical composition of fine particles from various types of sources differs, "…no clear scientific grounds exist for supporting differential effects estimates by particle type" (EPA 2004b). Similar reasoning was stated by EPA in the recent regulatory impact assessment for controls on emissions of hazardous air pollutants from coal-fired power plants.

Further supporting the findings cited in the EPA documents is a follow-up study (Laden et al. 2006) of a paper authored by Dockery (1993) on mortality in the Harvard Six Cities Study. Laden and colleagues examined mortality rates in the six cities for 1990 – 1998 after $PM_{2.5}$ levels decreased in comparison to the original period of study (1974 – 1989) for these cities. The $PM_{2.5}$ in these cities is composed of a mixture of primary and secondary aerosol from local, regional, and distant sources and annual average concentrations ranged from approximately 10 to 40 $\mu g/m^3$. Furthermore, the relative contribution of source categories (i.e., power plants, industry, mobile sources) was different across these communities. The analysis demonstrated that decreases in annual average $PM_{2.5}$ concentrations in these cities over 25 years were associated with declines in the corresponding mortality rates. The results of this study offer evidence for public health benefits from a general reduction of particle air pollution at concentrations below the current NAAQS, including in cities where the air pollution is heavily influenced by power plant contributions (e.g., Kingston-Harriman, TN, and Steubenville, OH).

Further evidence for a coal-related $PM_{2.5}$ effect on mortality is provided by an intervention study in Dublin, Ireland (Clancy et al. 2002). Particulate air pollution and mortality were evaluated before and after a ban on coal sales in Dublin. Black smoke concentrations (a surrogate for $PM_{2.5}$) declined 70% after the ban on coal sales. There was a coincident drop in non-trauma deaths, respiratory deaths, and cardiovascular deaths of 5.7%, 15.5%, and 10.3%, respectively. The findings of this study suggest that control of coal-related particulate air pollution could reduce daily mortality.

Thus, there is no specific evidence that would lead us to quantitatively deviate from a baseline assumption of equal toxicity, and there is substantial evidence that $PM_{2.5}$ from coal combustion influences public health at current levels of exposure.

### 3.3 SPECIFIC APPROACH

EPA's county-resolution Source Receptor (S-R) Matrix was used to forecast the difference or incremental change in $PM_{2.5}$ concentrations due to the removal of emissions from the modeled EGUs for each county within the continental U.S. The county-level reductions (or incremental change) in $PM_{2.5}$ exposure constitute the air quality impacts of emissions from the nine coal-fired EGUs contemplated by our HIA.

We used relative risks reported in the best available epidemiological studies of air pollution and illness to estimate the incremental change in the incidence (i.e., rate) of selected health endpoints for a given change in air pollutant exposure. The concentration-response functions used in our analysis are described in Section 4.6. In brief, the health endpoints considered in the analysis were premature mortality (for both adults and infants), respiratory and cardiovascular hospital admissions, asthma related ER visits, asthma exacerbation, chronic bronchitis, and MRADs. The endpoints and concentration-response (C-R) functions used in this HIA are consistent with the assessments conducted by the EPA and the scientific community for estimating public health impacts of changes in air pollution levels in the U.S.

The baseline incidence of the respective health endpoints was obtained from the best available sources. For example, mortality rates were obtained from a public access database maintained by the Centers for Disease Control and Prevention. Baseline rates of hospital admissions and ER visits for specific disease codes were obtained from public databases produced by the National Center for Health Statistics. The baseline incidence of the other outcomes was obtained from the information reported in epidemiological studies.

The number of people in each county exposed to $PM_{2.5}$ was based upon population data reported for the 2000 U.S. Census.

## 3.4 EMISSIONS INVENTORY

Annual emissions of $PM_{2.5}$, $SO_2$, and $NO_x$ for each modeled EGU are used as inputs into the S-R Matrix. As part of the Clean Air Interstate Rule, large EGUs are required to report $SO_2$ and $NO_x$ emissions on a quarterly basis. Data from 2000 to 2009 was obtained from the EPA Clean Air Markets Emissions database to calculate an annual average emission of $SO_2$ and $NO_x$ as input data into the S-R Matrix. EGUs are not required to report $PM_{2.5}$ emissions to EPA in the same way as $SO_2$ and $NO_x$ emissions. $PM_{2.5}$ emissions data is available for 2005 and 2007 from the EPA National Emissions Inventory (NEI) for each EGU. In order to generate an annual average emission over the last 10 years, as we did for $SO_2$ and $NO_x$, we used available emissions data for 2005 and 2007 we compared the emissions for those years to annual heat input data for years 2000 to 2009 (available from the Clean Air Markets Database) to estimate $PM_{2.5}$ emissions for the years in which $PM_{2.5}$ emissions were not available. We then calculated an estimated annual $PM_{2.5}$ emissions for each EGU based on the estimated annual emissions for 2000 to 2009. Estimated emissions for each modeled EGU are presented in Table 3.1.

**Table 3.1** Estimated Annual Emissions of Primary $PM_{2.5}$ and Precursors of $PM_{2.5}$ for Older Coal-fired Electricity Generating Units in Michigan

| Plant | $PM_{2.5}$ (tons/year) | $SO_2$ (tons/year) | $NO_x$ (tons/year) |
|---|---|---|---|
| BC Cobb* | 984 | 12,065 | 3,096 |
| Dan E Karn*/JC Weadock* | 513 | 24,332 | 8,109 |
| Harbor Beach | 409 | 1,544 | 895 |
| JH Campbell* | 1,148 | 17,690 | 6,045 |
| JR Whiting | 1,981 | 10,736 | 3,318 |
| River Rouge | 685 | 14,099 | 4,851 |
| St. Clair | 1,474 | 15,942 | 5,462 |
| Trenton Channel | 2,241 | 28,086 | 5,973 |

$PM_{2.5}$ particulate matter that is 2.5 micrometers or smaller in size
$SO_2$ sulfur dioxide
NOx nitrogen oxides or oxides of nitrogen

* Values do not include boilers that began operation after 1969.

## 3.5 EXPOSURE ASSESSMENT

As noted in Section 3.3, we calculated changes in population exposure to $PM_{2.5}$ for each county in the United States using EPA's county-resolution S-R Matrix. The S-R Matrix is a reduced form model that provides relationships between emissions of $PM_{2.5}$ and particle precursors and county level $PM_{2.5}$ concentrations. S-R Matrix is based on the Climatological Regional Dispersion Model (CRDM), a sector-averaged Gaussian dispersion model that includes wet and dry deposition and first-order chemical conversion of $SO_2$ and $NO_x$ to sulfate and nitrate particles. While not as sophisticated as other EPA models used to estimate $PM_{2.5}$, S-R Matrix has been used in a number of regulatory impact assessments and has been shown to be able to estimate the impacts of individual power plants. The incremental county-level $PM_{2.5}$ concentrations associated with emissions from each modeled EGU were linked to human populations specified for the corresponding counties by the U.S. Census Bureau using Geographic Information System (GIS) computer software.

## 3.6 BASELINE INCIDENCE OF HEALTH ENDPOINTS

For mortality, county-specific incidence rates for each state in the modeling domain were obtained from the Centers for Disease Control and Prevention WONDER website. Specifically, annual mortality data by age groups for all non-accidental causes (International Classification of Diseases, 10th Revision, group codes A00 – R99) was obtained for the years 1999 – 2006. The average of the latest five years of mortality data was used to obtain stable estimates of mortality incidence. For use in estimating mortality related to $PM_{2.5}$ exposures, population weighted mortality incidence rates were calculated for the population 30 years and older and the population under one year of age to be consistent with the epidemiological evidence and previously cited methodology.

For the population 30 years or older, the county-specific non-accidental mortality rates ranged from 5.6 to 30.2 deaths per 1,000 people. The National Center for Health Statistics (NCHS) cites a mortality rate for all ages for the entire country of 8.41 per 1,000 people for the year 2000.

To estimate the baseline incidence of respiratory-related hospital admissions, regional (Northeast, Midwest, South, West) hospitalization counts were obtained from the Year 2000 NCHS National Hospital Discharge Survey (NHDS). Hospitalization records for all respiratory causes (International Classification of Diseases, 9th Revision, 460 – 519) were used. Pneumonia, asthma, chronic bronchitis, and acute bronchitis were the most common causes for respiratory-related hospital admissions. The weights of the records with the specified ICD-9 code were then summed to produce an estimate of the number of hospital discharges by region for the specific health outcomes. Per capita hospitalization rates were calculated by dividing the estimate of the number of hospital discharges in the region by the estimated regional population derived from the Year 2000 Census data obtained from the U.S. Census Bureau. The regional respiratory hospitalization rates were then applied to each of the counties in the respective regions. The regional respiratory-related hospital admissions rates for the Northeast, Midwest, South, and the West are 14.28, 12.79, 12.83, and 9.00 per 1,000 people, respectively.

To estimate the baseline incidence of cardiovascular-related hospital admissions, regional (Northeast, Midwest, South, West) hospitalization counts were obtained from the Year 2000 NCHS NHDS. Hospitalization records for all cardiovascular-related admissions (International Classification of Diseases, 9th Revision, 390 – 429) for patients 65 years and older were identified. Heart failure, chronic ischemic heart disease, cardiac dysrhythmias, and acute myocardial infarctions were the most common causes of cardiovascular-related hospital admissions. The weights of the records with the specified ICD-9 code were then summed to produce an estimate of the number of hospital discharges by region for the specific health outcomes. Per capita hospitalization rates were calculated by dividing the estimate of the number of hospital discharges in the region by the estimated regional population derived from the Year 2000 Census data obtained from the U.S. Census Bureau for individuals 65 years and older. The regional cardiovascular hospitalization rates were then applied to each of the counties in the respective regions. For the population 65 years and older, the regional cardiovascular-related hospital admissions rates for the Northeast, Midwest, South, and West are 104.05, 91.95, 84.75, and 60.35 per 1,000 people, respectively.

To estimate the baseline incidence of asthma-related emergency room visits, regional (Northeast, Midwest, South, West) hospitalization counts were obtained from the Year

2000 National Hospital Ambulatory Medical Care Survey. Records of emergency room visits for asthma (International Classification of Diseases, 9th Revision, 493) were identified and the weights of the records with the specified ICD-9 code were then summed to produce an estimate of the number of asthma-related emergency room visits by region. Per capita emergency room visit rates were calculated by dividing the estimate of the number of visits in the region for all patients and for patients under the age of 18 by the estimated regional population derived from the Year 2000 Census data obtained from the U.S. Census Bureau for all individuals or for individuals under the age of 18. The regional ER visit rates were then applied to each of the counties in the respective regions. For all ages, the regional ER visit rates for asthma for the Northeast, Midwest, South, and the West are 7.22, 9.63, 6.02, and 3.05 per 1,000 people, respectively.

Chronic bronchitis is an inflammation of the airways in the lungs that is long-lasting (as opposed to acute bronchitis, a shorter-term episode usually caused by an infection). It is generally diagnosed based on cough and excessive mucus production for three months or more for at least two years. There are no national estimates of the incidence of new cases of chronic bronchitis. Similar to the EPA (EPA 2004b; EPA 2010c), we used an incidence rate of 3.78 cases per 1,000 people based on a study of non-smokers in California (Abbey et al. 1993). This incidence rate was applied to the entire modeling domain. While the use of non-smokers to estimate incidence may underestimate the true incidence in our modeling domain, this represents the best available evidence and is in agreement with the approach used by the EPA.

A baseline rate of 20.08 attacks per year per asthmatic, based on the 1999 National Health Interview Survey, was also used in our analysis. In the survey, asthmatics 18 years and older were asked about the number of wheezing attacks they experienced per year. This rate was used as a surrogate for asthma attacks for all age groups and applied to the entire modeling domain. The American Lung Association reports current prevalence of asthmatics in the U.S. for six age groups (<5 years old, 5-17, 18-44, 45-64, and 65 and older) (American Lung Association 2010). Based on this information, we estimated the number of individuals at risk in the modeling domain by multiplying the age specific populations of each county by their respective prevalence. This same methodology was employed by the EPA in recent HIA (EPA 2010c). While there are

clearly variations in asthma prevalence and attack rates by geographic location, socioeconomic status, race, and other factors (with much greater prevalence among low-income urban populations), the data were not available to provide more geographically refined estimates across the entire domain.

An MRAD is defined as a day that does not result in missing work, but involves minor symptoms and generally reduced activity. A study published in 1989 estimated the impact of ozone and $PM_{2.5}$ on MRADs in a national sample of the adult working population, ages 18 to 65 (Ostro and Rothschild 1989). This study provides an estimated annual incidence rate of 7.8 MRADs per person. This rate was applied to our entire modeling domain as was done in analyses conducted by the EPA (EPA 2004b; EPA 2010c).

## 3.7 CONCENTRATION-RESPONSE FUNCTIONS

We developed concentration-response functions for $PM_{2.5}$, considering a number of health outcomes. Of note, we did not develop concentration-response functions for all health outcomes associated with these pollutants. For example, influences on lung function, heart rate variability, or other pre-clinical states were not considered, as they cannot be formally incorporated into economic analyses, and may overlap with clinical endpoints. In addition, if we judged the epidemiological literature to be insufficiently robust to specify a concentration-response function, we did not include the health outcome. The outcomes included in our assessment are similar to those incorporated into the EPA's regulatory impact analyses (EPA 1997; EPA 1999b; EPA 2004b; EPA 2005; EPA 2010b). Below, we explain the methods used to develop our best estimates for the concentration-response functions.

In general, we rely on statistical meta-analyses to pool the evidence from multiple studies of the same health endpoint. A meta-analysis is a systematic and quantitative synthesis of the epidemiological evidence from a number of published studies, often addressing both the central estimate resulting from the pooled evidence base and factors that contribute to any variability across studies. Meta-analyses are common in HIA and in many other applications, as they provide enhanced statistical power and means to determine a concentration-response function or relative risk estimate based on

an objective synthesis of the totality of the literature. The approach for conducting meta-analyses has been well established and is comprehensively described in textbooks related to medical decision-making (Petitti 2000). Meta-analyses are used extensively both in air pollution epidemiology and in numerous other applications. In two recent examples meta-analysis techniques were used to evaluate adherence to antiretroviral therapy in Africa and North America (Mills et al. 2006) and to determine the effects of early treatment with stations on short-term clinical outcomes in acute coronary syndromes (Briel et al. 2006), among many other applications.

Often, investigators use inverse-variance weighting to combine the evidence across studies. Inverse-variance weighting is a standard technique used in meta-analyses that weights the results of studies in proportion to the precision of the estimated risk coefficient. In this way, studies with greater statistical precision are given added weight.

A key step in any meta-analysis involves deciding which studies to include or exclude. Studies must be comparable to one another in methods and approach to be reasonably pooled together, so individual studies may be excluded because they used a different measure of exposure or outcome, a very different statistical technique, and so forth. In this context, we have a preference for studies conducted in the U.S., as there may be important differences in disease prevalence, exposure patterns, and health care utilization patterns in other countries. That being said, in situations where there are relatively few studies in the U.S., we utilize studies conducted elsewhere, and evaluate whether the concentration-response function would be significantly different if the meta-analysis were restricted to U.S. studies only. Similarly, while we have a preference for studies of the general U.S. population, studies of subpopulations are applicable when the evidence base is more limited. This is similar to the approach used by the EPA, which relies on the global literature to derive concentration-response functions for regulatory impact analyses in the U.S. In Sections 4.6.1 and 4.6.2, we describe our rationale for study selection and include sensitivity calculations where applicable to ensure that our concentration-response functions are not strongly dependent on our selection criteria.

There was not a recent publication synthesizing the long-term exposure $PM_{2.5}$ concentration-response functions for the purpose of health impact assessment. While a

formal evaluation of the literature was conducted in a 1999 publication (Levy et al. 1999), there has been a tremendous growth in $PM_{2.5}$ publications in the ensuing years. Therefore, it was important to reconstruct these functions. Although this literature has been frequently evaluated by the EPA as part of their regulatory impact analyses, we chose to develop the concentration-response functions independently.

### 3.7.1 Premature Mortality

There is strong evidence of effects of long-term exposure to $PM_{2.5}$ on the risk of premature mortality. The following table (Figure 4.1), taken from a recent literature synthesis by Pope and Dockery (Pope and Dockery 2006), lists the publications that considered the mortality risks of long-term exposure to $PM_{2.5}$.

**Table 2.** Comparison of percentage increase (and 95% CI) in relative risk of mortality associated with long-term particulate exposure.

| | | | Percent Increases in Relative Risk of Mortality (95% CI) | | |
|---|---|---|---|---|---|
| **Study** | **Primary Sources** | **Exposure Increment** | **All Cause** | **Cardiopulmonary** | **Lung Cancer** |
| Harvard Six Cities, original | Dockery et al. 1993[26] | 10 μg/m³ $PM_{2.5}$ | 13 (4.2, 23) | 18 (6.0, 32) | 18 (−11, 57) |
| Harvard Six Cities, HEI reanalysis | Krewski et al. 2000[177] | 10 μg/m³ $PM_{2.5}$ | 14 (5.4, 23) | 19 (6.5, 33) | 21 (−8.4, 60) |
| Harvard Six Cities, extended analysis | Laden et al. 2006[184] | 10 μg/m³ $PM_{2.5}$ | 16 (7, 26) | 28 (13, 44)[a] | 27 (−4, 69) |
| ACS, original | Pope et al. 1995[27] | 10 μg/m³ $PM_{2.5}$ | 6.6 (3.5, 9.8) | 12 (6.7,17) | 1.2 (−8.7, 12) |
| ACS, HEI reanalysis | Krewski et al. 2000[177] | 10 μg/m³ $PM_{2.5}$ | 7.0 (3.9, 10) | 12 (7.4, 17) | 0.8 (−8.7, 11) |
| ACS, extended analysis | Pope et al. 2002[179] | 10 μg/m³ $PM_{2.5}$ | 6.2 (1.6, 11) | 9.3 (3.3, 16) | 13.5 (4.4, 23) |
| | Pope et al. 2004[180] | | | 12 (8, 15)[a] | |
| ACS adjusted using various education weighting schemes | Dockery et al. 1993[26] | 10 μg/m³ $PM_{2.5}$ | 8–11 | 12–14 | 3–24 |
| | Pope et al. 2002[179] | | | | |
| | Krewski et al. 2000[177] | | | | |
| ACS intrametro Los Angeles | Jerrett et al. 2005[181] | 10 μg/m³ $PM_{2.5}$ | 17 (5, 30) | 12 (−3, 30) | 44 (−2, 211) |
| Postneonatal infant mortality, U.S. | Woodruff et al. 1997[185] | 20 μg/m³ $PM_{10}$ | 8.0 (4, 14) | – | – |
| Postneonatal Infant mortality, CA | Woodruff et al. 2006[186] | 10 μg/m³ $PM_{2.5}$ | 7.0 (−7, 24) | 113 (12, 305)[c] | – |
| AHSMOG[b] | Abbey et al. 1999[187] | 20 μg/m³ $PM_{10}$ | 2.1 (−4.5, 9.2) | 0.6 (−7.8, 10) | 81 (14, 186) |
| AHSMOG, males only | McDonnell et al. 2000[188] | 10 μg/m³ $PM_{2.5}$ | 8.5 (−2.3, 21) | 23 (−3, 55) | 39 (−21, 150) |
| AHSMOG, females only | Chen et al. 2005[189] | 10 μg/m³ $PM_{2.5}$ | – | 42 (6, 90)[a] | – |
| Women's Health Initiative | Miller et al. 2004[190] | 10 μg/m³ $PM_{2.5}$ | – | 32 (1, 73)[a] | |
| VA, preliminary | Lipfert et al. 2000, 2003[190,192] | 10 μg/m³ $PM_{2.5}$ | 0.3 (NS)[d] | – | – |
| VA, extended | Lipfert et al. 2006[193] | 10 μg/m³ $PM_{2.5}$ | 15 (5, 26)[e] | – | – |
| 11 CA counties, elderly | Enstrom 2005[194] | 10 μg/m³ $PM_{2.5}$ | 1 (−0.6, 2.6) | – | – |
| Netherlands | Hoek et al. 2002[195] | 10 μg/m³ BS | 17 (−24, 78) | 34 (−32, 164) | – |
| Netherlands | Hoek et al. 2002[195] | Near major road | 41 (−6, 112) | 95 (9, 251) | – |
| Hamilton, Ontario, Canada | Finkelstein et al. 2004[197] | Near major road | 18 (2, 38) | – | – |
| French PAARC | Filleul et al. 2005[198] | 10 μg/m³ BS | 7 (3, 10)[f] | 5 (−2,12)[f] | 3 (−8,15)[f] |
| Cystic fibrosis | Goss et al. 2004[200] | 10 μg/m³ $PM_{2.5}$ | 32 (−9, 93) | – | – |

[a]Cardiovascular only; [b]Pooled estimates for males and females; pollution associations were observed primarily in males and not females; [c]Respiratory only; [d]Reported to be nonsignificant by author; overall, effect estimates to various measure of particulate air pollution were highly unstable and not robust to selection of model and time windows; [e]Estimates from the single pollutant model and for 1989–1996 follow-up; effect estimates are much smaller and statistically insignificant in an analysis restricted to counties with nitrogen dioxide data and for the 1997–2001 follow-up; furthermore, county-level traffic density is a strong predictor of survival and stronger than $PM_{2.5}$ when included with $PM_{2.5}$ in joint regressions; [f]Estimates when six monitors that were heavily influenced by local traffic sources were excluded; when data from all 24 monitors in all areas were used, no statistically significant associations between mortality and pollution were observed.

**Figure 3.1** Summary of Publications that Consider Mortality Risks of Long-term Exposure to $PM_{2.5}$ (reproduced from Pope and Dockery, 2006)

This table summarizes numerous studies that examined the relationship between ambient particle pollution and mortality. However, some of the publications may not be suitable for developing a concentration-response function applicable to the study population. For example, the Adventist Health and Smog (AHSMOG) study focused only on non-smoking Seventh Day Adventists living in California, so there are concerns regarding the generalizability of the population. Similarly, the Veterans Administration (VA) studies focus on middle-aged, hypertensive, male patients from VA clinics, most of whom were current or former smokers. Thus, while studies such as these can provide some useful insight about disease etiology or high-risk populations, studies of the general population are preferable, when available, to develop concentration-response functions applicable to the general population.

Of the studies listed above, the only ones that had an exposure measure relevant for power plants (i.e., not proximity to a major road) and a generalizable population were the Harvard Six Cities Study and the American Cancer Society (ACS) study. Both of these studies were large, long-term cohort studies, in which a population was enrolled and followed for a number of years to determine the association between mortality risks and air pollution. The Harvard Six Cities Study was based in six cities in the eastern U.S. (Watertown, MA; Kingston and Harriman, TN; St. Louis, MO; Steubenville, OH; Portage and Pardeeville, WI; and Topeka, KS), while the ACS study was based in all 50 states, the District of Columbia, and Puerto Rico.

As these studies had individual-level data, they were able to control for potentially important factors like smoking, alcohol consumption, and socioeconomic status, which could otherwise confound the association between air pollution and health outcomes. Both of these studies have yielded multiple estimates over the years, with the original publications followed by extensive re-analyses by independent investigators and more recent publications with more years of follow up.

As can be seen in Figure 4.3 above, the Harvard Six Cities Study yields estimates on the order of a 1.3% to 1.6% increase in premature mortality per $\mu g/m^3$ increase of long-term $PM_{2.5}$. The ACS study generally yields somewhat lower estimates, on the order of 0.6% or 0.7%, which has been attributed to multiple factors including the fact that the population was generally of higher socioeconomic status and education than in the

Harvard Six Cities Study (and than in the U.S. population at large) and the fact that air pollution data were gathered retrospectively from central site monitors rather than prospectively in community-specific monitors. This latter fact would tend to increase exposure misclassification and result in downwardly biased estimates. This was illustrated in a recent publication (Jerrett et al. 2005), in which more refined exposure estimates were derived for the ACS cohort using geographic information systems, resulting in an estimate of 1.7%, nearly triple the original estimate. The Jerrett et al. 2005 study, while informative, focused exclusively on air pollution and mortality in the Los Angeles basin and therefore may not be as applicable as the results based on the entire ACS cohort and the Six Cities Study for a health impact assessment centered in the Midwestern U.S.

Thus, the estimates from the relevant literature range from 0.6% to 1.7% increases in mortality per $\mu g/m^3$ of long-term $PM_{2.5}$, with the lower values likely biased downward due to exposure misclassification in the ACS study and due to a higher socioeconomic status population than the U.S. average. We would therefore consider a value of a 1% increase in mortality per $\mu g/m^3$ of long-term $PM_{2.5}$ to reasonably represent the current knowledge base and use this value as our best estimate.

Long-term exposure to particulate matter affects the probability of survival. As clearly shown in the Harvard Six Cities Study, the higher the $PM_{2.5}$ concentration is in a city, the higher the mortality rates after adjusting for smoking and other risk factors. It cannot be demonstrated absolutely whether the risk is from cumulative long-term exposure or the effects of higher short-term exposure, which will also be lowered if permanent air pollution controls are put in place. However, the coal ban study in Dublin (Clancy et al. 2002) suggests that benefits are immediate, and the recent Six Cities Study follow-up (Laden et al. 2006) demonstrates public health benefits relatively close to the time when air pollution reductions occur. Nevertheless, in the Regulatory Impact Analysis for the Clean Air Interstate Rule, the EPA has recommended that the benefits from reduced air pollution mortality be distributed over 20 years as follows (with corresponding discounting of the economic value of a life saved): 30% in the first year after exposure reduction; 50% evenly over years 2 through 5; and 20% evenly over years 6 to 20 after the reduction (EPA 2005). We recommend following the EPA practice established in the

Clean Air Interstate Rule Regulatory Impact Analysis for purposes of valuing our estimates of $PM_{2.5}$-related premature mortality avoided presented in this report.

### 3.7.2 Infant Mortality

The above studies of premature mortality only address adults age 30 and older, but there is also evidence that $PM_{2.5}$ exposure influences infant mortality. This outcome has been evaluated in various forms in a number of publications (Bobak and Leon 1992; Woodruff et al. 1997; Loomis et al. 1999; Lipfert et al. 2000; Dales et al. 2004; Ritz et al. 2006; Woodruff et al. 2006).

For an outcome such as infant mortality, which varies substantially across countries, it is preferable to develop the concentration-response function from studies in the U.S., or at a minimum, to omit studies in countries where the baseline infant mortality rate differs greatly from the rate in the U.S. Given a good evidence base in the U.S., as described below, we do not use evidence from the Bobak and Leon study (from the Czech Republic) or the Loomis et al. study (from Mexico) to develop our concentration-response function. We also do not formally consider the study by Dales et al., as it did not look at all-cause infant mortality (only considering sudden infant death syndrome).

The remaining studies are of two basic forms. There are two cross-sectional studies (Woodruff et al. 1997; Lipfert et al. 2000), which are less methodologically robust[2] than cohort studies but are based in the U.S. and can be used to confirm the findings from cohort studies. Woodruff et al. report a 4% increase in infant mortality per 10 $\mu g/m^3$ increase of $PM_{10}$. Assuming a $PM_{2.5}/PM_{10}$ concentration ratio of 0.6 (based on monitoring data reported in the Air Quality Criteria Document for Particulate Matter) and that the health effects observed are attributable to $PM_{2.5}$ (a standard assumption used by the EPA and others in health impact assessment), this would correspond with a 7% increase

[2] A cross-sectional epidemiological study compares rates of death or disease between cities without information on individuals. Cross-sectional studies can control for risk factors like smoking by looking at (for example) the percent of people who smoke in a given city, but they cannot look at whether the individuals who died or developed disease were also the ones who smoked. The ACS Study and Harvard Six Cities Study are two examples of cohort studies that include information on each individual.

in infant mortality per 10 $\mu g/m^3$ increase of $PM_{2.5}$. The study by Lipfert et al. reports numerous estimates but generally corroborates this magnitude of effect.

The two strongest studies from which the concentration-response function can be derived are the recent publications by (Woodruff et al. 2006) and (Ritz et al. 2006). Woodruff et al. found a central estimate of effect on all-cause infant mortality of a 7% increase per 10 $\mu g/m^3$ of $PM_{2.5,}$ with greater statistical significance seen for respiratory-related mortality. Ritz et al. reported a 4% increase in all-cause infant mortality per 10 $\mu g/m^3$ of $PM_{10}$; converting as above, this corresponds with a 7% increase per 10 $\mu g/m^3$ of $PM_{2.5}$.

Thus, the two cohort studies yield identical estimates of the effect of $PM_{2.5}$ on infant mortality when rounded to one significant figure, which agree with the values derived from the cross-sectional studies. Thus, a 0.7% increase in all-cause infant mortality per $\mu g/m^3$ of $PM_{2.5}$ represents our best estimate.

### 3.7.3 Cardiovascular Hospital Admissions

For hospital admissions for cardiovascular causes, we rely on a recent meta-analysis (COMEAP 2006) which combined 51 published studies to determine that cardiovascular hospital admissions increase by an estimated 0.9% per 10 $\mu g/m^3$ increase of $PM_{10}$. As this study included all recent publications, including multi-city estimates, and used similar statistical techniques as above (i.e., inverse-variance weighting using random effects techniques), we consider this to be the best available information.

However, as discussed above, given that differences in health care systems among countries may influence an outcome like hospital admissions, utilizing a large number of non-U.S. studies to determine a concentration-response function for this matter may not be appropriate. We therefore re-ran the meta-analysis restricted to the 33 estimates from U.S. studies, and determined a central estimate nearly identical to that above (a 0.97% increase in cardiovascular hospital admissions per 10 $\mu g/m^3$ of $PM_{10}$, slightly higher than the all-study value).

Thus, using the $PM_{2.5}/PM_{10}$ conversion described above, we would consider an appropriate best estimate to be a 0.16% increase in cardiovascular hospital admissions per $\mu g/m^3$ increase of $PM_{2.5}$. Notably, this concentration-response function is less than the value of 0.5% per $\mu g/m^3$ of $PM_{2.5}$ for hospital admissions for myocardial infarction (heart attack) noted in a recent scientific paper (Zanobetti and Schwartz 2006).

### 3.7.4 Respiratory Hospital Admissions

To derive an estimate for respiratory hospital admissions, we conducted a meta-analysis of the published literature, given a large number of studies and no recently published meta-analyses. The studies considered were taken from Levy et al. (1999), from the EPA's BenMAP program, and from the Air Quality Criteria Document for Particulate Matter (EPA 2004a). From the large number of studies available, we eliminated a subset of studies that could not be statistically pooled with other studies for a variety of reasons. This included the application of statistical methods that were not comparable with other studies, use of a pollutant measure other than $PM_{2.5}$ (i.e., only considering acid aerosols or black smoke), consideration of specific respiratory diseases rather than all-cause respiratory hospital admissions, or evaluation of effects on children only. Of note, this does not imply that these studies did not represent good scientific evidence, but simply that they were not the best studies to combine to develop concentration-response functions using statistical meta-analysis techniques.

The remaining studies were: (Thurston et al. 1994; Schwartz 1995; Schwartz 1996; Schwartz et al. 1996; Burnett et al. 1997; Wordley et al. 1997; Atkinson et al. 1999; Gwynn et al. 2000; Hagen et al. 2000; Anderson et al. 2001; Gwynn and Thurston 2001). If we pool all studies using inverse-variance weighting with statistical methods to account for the possibility of true between-site heterogeneity, as done in the Committee on the Medical Effects of Air Pollutants (COMEAP) study and other publications (Levy et al. 2000; Levy et al. 2005), the central estimate is a 0.2% increase in respiratory hospital admissions per 1 $\mu g/m^3$ increase of $PM_{2.5}$.

However, there are some differences in scope of the studies, in terms of time of year, geographic location, and subpopulation considered, so it is important to test if this estimate is robust. Our ideal approach would be to restrict only to studies conducted in

the U.S., considering all ages across the entire year. In this case, the sample size would be reduced to only two studies by Gwynn et al., which is far less statistically robust. That being said, the pooled estimate from those two studies remains 0.2%. If we broaden our scope and consider all U.S. studies (adding in four studies considering only elderly populations), the pooled estimate is still 0.2% (with the elderly-only estimate as 0.2%). This therefore appears to be a robust best estimate representative of the effects on all ages in the U.S., and we use this value for our health impact assessment.

### 3.7.5 Asthma-Related Emergency Room Visits

Another morbidity outcome of concern for $PM_{2.5}$ is ER visits among asthmatic individuals. As we did for respiratory hospital admissions, we gathered studies from Levy et al. (1999) and the Air Quality Criteria Document for Particulate Matter (EPA 2004a), and supplemented this database with an independent literature search. As this is a fairly substantial literature, we were able to restrict our focus to U.S. studies for the formal statistical meta-analysis. In total, we found five studies that were suitable for meta-analysis—(Schwartz et al. 1993; Lipsett et al. 1997; Norris et al. 1999; Tolbert et al. 2000; Peel et al. 2005).

As for respiratory hospital admissions, there were some differences in the scopes of these studies. In addition, there was one important methodological difference that had to be addressed. The study by Peel et al. in Atlanta considered a distributed lag of effects over a two-week period, in which the impact is substantially higher as compared with a three-day moving average. Ignoring the distributed lag of effects may underestimate the public health impacts, but including it creates methodological differences across the studies (as only Peel et al. evaluated the possibility of a distributed lag). Pooling all five studies yields estimates of 0.6% with Peel's three-day moving average estimate, and 1.1% with Peel's distributed lag estimate.

We consider more comparable subsets of studies to determine the range of pooled estimates that these subsets would imply. Pooling the two studies of pediatric populations (Norris et al. 1999; Tolbert et al. 2000) using inverse-variance weighting with random effects as above, we estimate a 1% increase in asthma-related ER visits per $\mu g/m^3$ increase of $PM_{2.5}$. Pooling the two all-age studies (Lipsett et al. 1997; Peel et al.

2005) yields an estimate of 0.6% using the three-day moving average estimate from Peel et al., but 1.6% if we consider the distributed lag of effects in Peel et al.

As there is broad consistency across studies in the magnitude and significance of the effect, it makes sense to consider this an all-age effect. In terms of the magnitude of the concentration-response function, we consider a best estimate to be a 0.8% increase in asthma-related ER visits per $\mu g/m^3$ increase of $PM_{2.5}$. This value represents an intermediate value between the two pooled estimates using the alternative Peel et al. values, and is identical to the pooled estimate for all studies but Peel et al.

### 3.7.6 Asthma Attacks

There are many published studies related to exacerbation of asthma of various types, including asthma attacks, symptoms, cough, wheeze, and other definitions. To ensure consistency across studies, we focus our meta-analysis on studies looking at asthma exacerbation as measured by aggregate symptoms, rather than specifically looking at cough, wheeze, or other defined outcomes. Studies were drawn from the Air Quality Criteria Document for Particulate Matter (EPA 2004a). From the studies reported in Table 8B-5 of the Criteria Document, five studies were considered applicable, based on their usage of a total symptom measure: (Delfino et al. 1998; Yu et al. 2000; Delfino et al. 2002; Desqueyroux et al. 2002; Mortimer et al. 2002). Pooling these five studies in an inverse-variance weighted meta-analysis yields an estimated 2% increase in asthma exacerbations (among asthmatics only) per $\mu g/m^3$ increase of $PM_{2.5}$. The estimate is essentially identical if non-U.S. studies are excluded, and there is consistency in the magnitude of the concentration-response function across all studies.

### 3.7.7 Chronic Bronchitis

Unlike the other morbidity outcomes described above, chronic bronchitis relates not to the effects of short-term exposures on exacerbation of disease, but rather to the effects of longer-term exposures on the development of disease. Chronic bronchitis is a distinctly different disease from acute bronchitis, involving long-term lung inflammation and a variety of associated symptoms.

Only a few studies in the literature have addressed chronic bronchitis (Abbey et al. 1993; Schwartz et al. 1993; Xu and Wang 1993; Abbey 1995). The study by Xu and Wang was based in China and was a cross-sectional study design, so we excluded it from the meta-analysis. In addition, the study by Abbey et al. in 1993 was based on the same cohort as the 1995 study, so the more recent publication supersedes the older publication.

Looking at the two remaining studies, the study by Abbey (1995) is the only prospective cohort study, which is why the EPA uses this study in their regulatory impact analyses. This publication gives an estimate corresponding to an approximate 1.3% increase in chronic bronchitis incidence per $\mu g/m^3$ increase of $PM_{2.5}$. The study by Schwartz et al. (1993) is cross-sectional, but it focuses on a more generalizable population than Abbey (all individuals living in 53 urban areas across the U.S., versus non-smoking Seventh Day Adventists living in California), and therefore merits consideration. In particular, the study by Abbey potentially underestimates the general population effect by omitting smokers, who are more susceptible to chronic bronchitis. After converting the measure of total suspended particulates (TSP) into $PM_{2.5}$ following standard concentration ratios, the estimate from Schwartz et al. corresponds to a 2% increase in chronic bronchitis rates per $\mu g/m^3$ increase of $PM_{2.5}$. Pooling these two studies, a 1.5% increase in chronic bronchitis rates per $\mu g/m^3$ increase of $PM_{2.5}$ represents our best estimate.

### 3.7.8 Minor Restricted Activity Days

The final outcome for $PM_{2.5}$ is MRADs, which is derived from Ostro and Rothschild (1989). Of note, there are additional publications related to respiratory symptoms or more severe restricted activity days from $PM_{2.5}$, which have been used by the EPA and others in addition to MRADs, but we focus on this single outcome to avoid double-counting. Using inverse-variance weighting on six individual year estimates as previously, this study implies a central estimate of a 0.7% increase per $\mu g/m^3$ increase of $PM_{2.5}$.

## 3.8 ECONOMIC VALUATION

The values that we selected for our analysis were utilized most recently in the EPA Regulatory Impact Analysis for the Proposed Federal Transport Rule and described in the EPA BenMAP Environmental Mapping and Regulatory Analysis Program (EPA 2010c; EPA 2010d). For premature mortality, EPA uses the mean from 26 value of statistical life (VSL) studies, including 5 contingent valuation and 21 labor market studies. The mean VSL from these studies was $6.3 million in year 2000 dollars (EPA 2010c; EPA 2010d). To convert to current dollars, we applied the Consumer Price Index, yielding an estimated $8 million in year 2010 dollars.

For hospital admissions and emergency room visits, since no willingness-to-pay (WTP) studies are available, the EPA (EPA 1999a; EPA 2004b) uses cost-of-illness (COI) estimates that are based on ICD-9 (International Statistical Classification of Diseases) code information (e.g., admission for chronic obstructive pulmonary disease, pneumonia, asthma, and cardiovascular disease). For cardiovascular disease, EPA reports a COI of approximately $25,000 in 2000 dollars (EPA 2010c; EPA 2010d), or approximately $27,000 in current dollars. For respiratory hospital admissions, the multitude of ICD-9 codes makes it difficult to apply a single value. The COI ranges from $8,900 (asthma) to $25,600 (all respiratory pneumonia) (EPA 2010d), however, given the relative frequency of hospital admissions for asthma compared to other respiratory outcomes, we chose to a value of $9,600 based on the asthma admissions, updated for current dollars. The use of this value may represent an underestimate of the true social cost of a hospital admission, due to the lack of inclusion of more expensive respiratory health outcomes. For asthma-related emergency room visits, EPA reports COI values of $384 (in 2006 dollars) based on two separate studies (EPA 2010c; EPA 2010d). Therefore, we used the simple average of the two values of approximately $415 in current dollars, and apply this estimate to all emergency room visits given the relative frequency of respiratory impacts in air pollution-related emergency room visits.

Several other health outcomes have also been associated with air pollution and are considered in our analysis—chronic bronchitis, asthma attacks and MRADs. For chronic bronchitis, EPA reports a WTP value of $340,000 in year 2006 dollars (EPA 2010c; EPA 2010d). Therefore, we used an estimate of $370,000 in current dollars. For asthma

attacks, EPA uses a value of $43 in year 2000 dollars (EPA 2010c; EPA 2010d), derived from reported willingness to pay to avoid a “bad asthma day”. Therefore, we used an estimate in current dollars of $46. EPA utilizes a WTP estimate for minor respiratory restricted activity days. For MRADs, we used an estimate of $67.

All estimates of impacts are rounded to two significant figures.

# 4.0 RESULTS

## 4.1 PUBLIC HEALTH IMPACTS

Our analysis of public health impacts is based on the modeled differences in population exposure to $PM_{2.5}$ if the emissions from the nine modeled EGUs in Michigan were eliminated. The results reflect estimated public health impacts of the proposed controls on a yearly basis.

Approximately 281 million people lived in the United States in 2000. In this population, 163 million are adults older than 29 years, 4 million are infants under one year, and 72 million are children under 18 years. Between 2000 and 2010 this population grew by approximately 27 million people.[3] The average age of the U.S. increased as well.[4] The increase in average age of the population means that if public health impacts were estimated for 2010, the results would be even greater than our estimates based on the 2000 Census data.

Our estimates of the annual health-related impacts in Michigan associated with reduced air pollutant emissions from the coal-fired EGUs are summarized in Table 4.1. One hundred and eighty (180) premature deaths are estimated to be avoided among the residents of Michigan for each year that the EGUs plants are operated. Morbidity impacts estimated as a result of reduced emissions from EGUs include 68,000 asthma exacerbations, and 72,000 MRADs avoided. Public health impacts attributable to $PM_{2.5}$ for each Michigan county are presented in Table A.1 in Appendix A.

[3] http://www.census.gov/prod/cen2010/briefs/c2010br-01.pdf
[4] http://www.census.gov/prod/cen2010/briefs/c2010br-03.pdf

**Table 4.1** Annual Mortality and Morbidity Impacts in Michigan Associated with Air Pollutant Emissions from Older Coal-fired Electricity Generating Units in Michigan

| Outcome | Cases |
|---|---|
| Premature mortality | 180 |
| Cardiovascular hospital admissions | 38 |
| Respiratory hospital admissions | 55 |
| Chronic bronchitis | 76 |
| Asthma emergency room visits | 140 |
| Asthma exacerbations | 68,000 |
| Minor restricted activity days | 72,000 |

Our estimates of premature mortality and morbidity impacts for the entire U.S. population for each year are presented in Table 4.2. The estimates include 660 premature deaths and approximately 800 hospital admissions or ER visits, and 250,000 exacerbations of asthma (e.g., an asthma attack) avoided for each year of controls. Over 260,000 MRADs are also estimated to be avoided annually.

**Table 4.2** Annual Mortality and Morbidity Impacts in the Continental United States Associated with Particulate Air Pollutant Emissions from Older Coal-fired Electricity Generating Units in Michigan

| Outcome | Cases |
|---|---|
| Premature mortality | 660 |
| Cardiovascular hospital admissions | 150 |
| Respiratory hospital admissions | 210 |
| Chronic bronchitis | 280 |
| Asthma emergency room visits | 450 |
| Asthma exacerbations | 250,000 |
| Minor restricted activity days | 260,000 |

Annual public health impacts for each state are listed by health endpoint in Table A.2 in Appendix A. After Michigan, the states with the greatest health impacts were Ohio, Pennsylvania, New York, and Illinois. The geographic distribution of public health impacts is a function of the patterns of incremental $PM_{2.5}$ exposure as well as the size and location of at-risk populations.

The geographic distribution of estimated public health impacts is illustrated in Figure 4.1 using premature deaths as an example. The impacts are most pronounced in the upper Midwest. Maps of state-specific estimates of total hospital impacts and asthma exacerbation impacts are provided in Figures B.1 and B.2 of Appendix B.

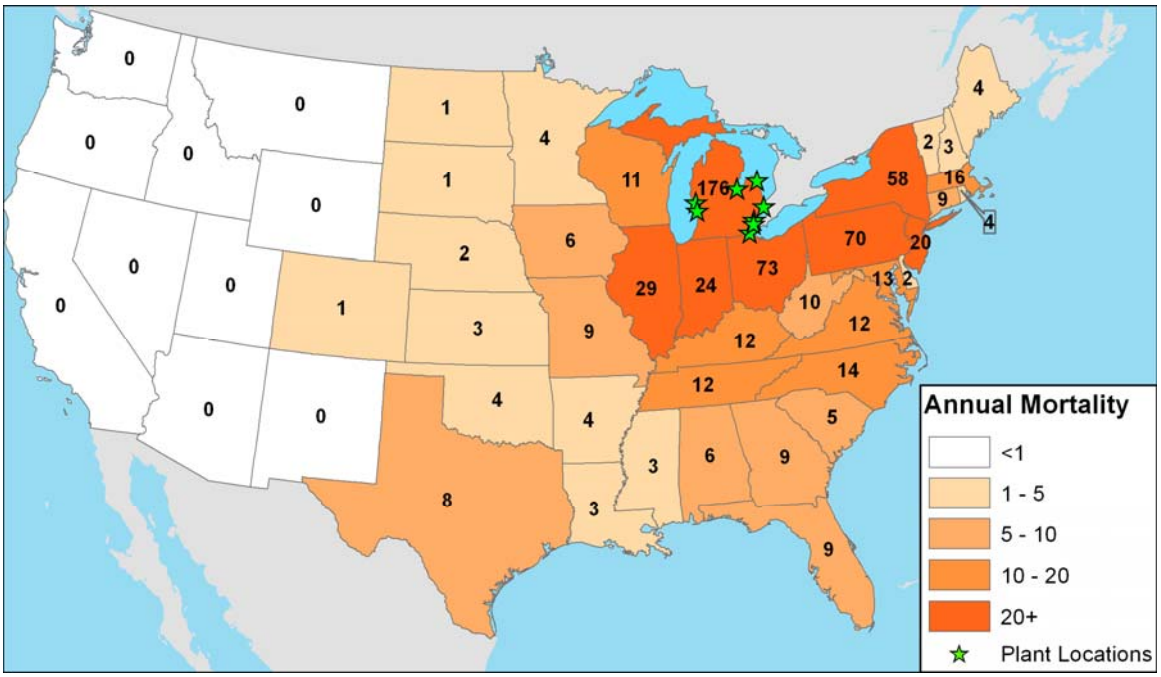


**Figure 4.1** Estimated Number of Premature Deaths for Each Year Associated with Fine Particle-related Emissions from Older Coal-fired Electricity Generating Units in Michigan

As described earlier, we determined the incremental change in population exposure to annual average $PM_{2.5}$ concentrations associated with emissions from nine coal-fired EGUs in Michigan for each county in the U.S. As shown in Figure 4.2, the Michigan counties with the greatest incremental change in annual average $PM_{2.5}$ exposure were in central and eastern Michigan, with the largest impacts in Monroe, Wayne and St. Clair counties. In Michigan, approximately 2.5 million people live in counties predicted to have a incremental reduction greater than 0.25 $\mu g/m^3$, 2.1 million with a reduction of 0.15 to 0.25 $\mu g/m^3$, 1.6 million with a reduction of 0.1 to 0.15 $\mu g/m^3$, 2.9 million with a reduction of 0.05 to 0.1 $\mu g/m^3$, and 800,000 people with a reduction of less than 0.05 $\mu g/m^3$.

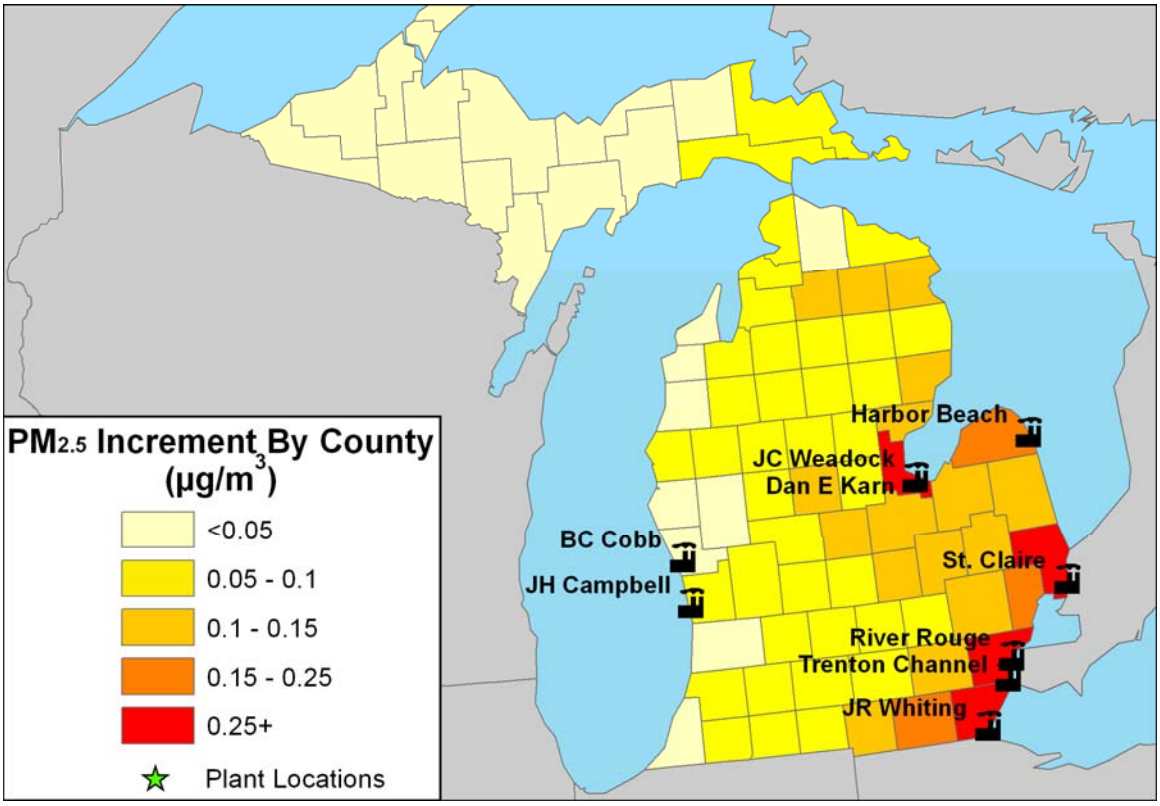


**Figure 4.2** Annual Average Fine Particle Concentrations (µg/m$^3$) in Michigan Associated with Emissions from Older Coal-fired Electricity Generating Units in Michigan

In the continental U.S., approximately 26 million people live in counties predicted to have a incremental reduction greater than 0.06 µg/m$^3$, 48 million with a reduction of 0.03 to 0.06 µg/m$^3$, 99 million with a reduction of 0.01 to 0.03 µg/m$^3$, and 103 million with a reduction of less than 0.01 µg/m$^3$ (see Figure 4.3).

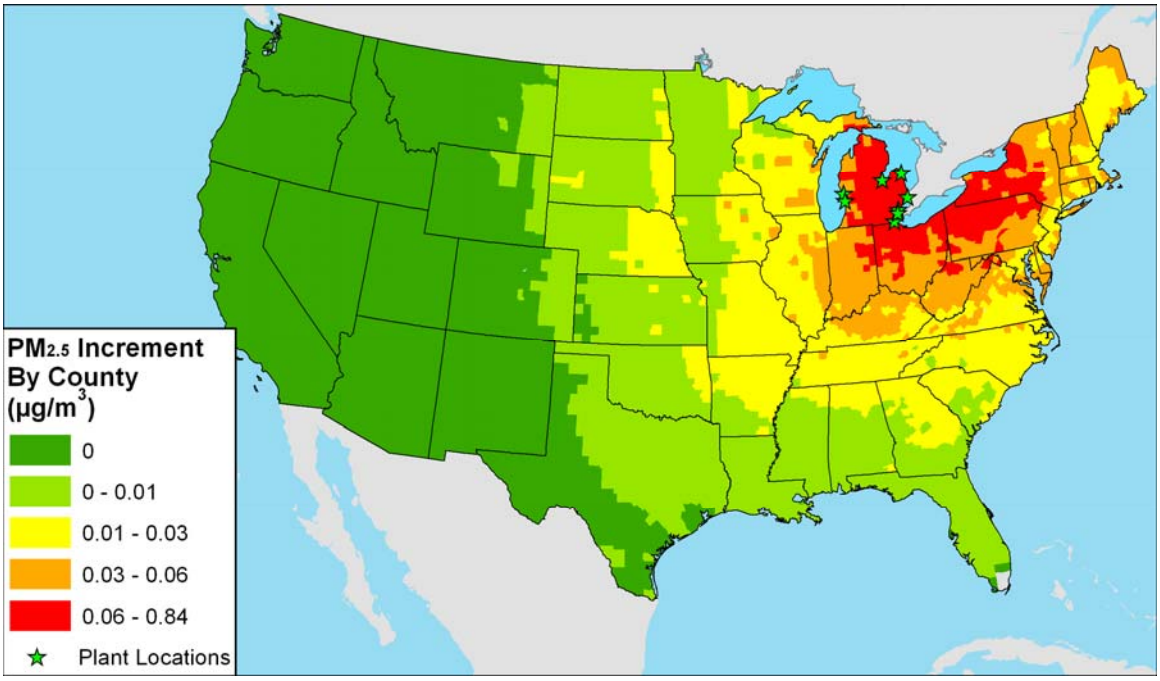


**Figure 4.3** Annual Average Increment of Fine Particle Concentrations (µg/m$^3$) in the Continental U.S. Associated with Emissions from Older Coal-fired Electricity Generating Units in Michigan

## 4.2 POTENTIAL IMPACTS FOR CANADA

One assumption that clearly represents an underestimate of the public health impacts associated with the modeled EGUs is limiting the modeling domain to the continental U.S. The S-R Matrix does not include Canada and therefore it is not possible to quantitatively determine the impacts to the Canadian population. However, the areas in Southeastern Ontario east of Michigan have the largest population and highest population densities in Canada, and would undoubtedly be impacted by emissions from the modeled EGUs. Statistics Canada estimates that approximately 9 million people live in the area highlighted in Figure 4.4 that would be most impacted by emissions from the modeled EGUs. As shown in Figure 4.4, we would expect the incremental $PM_{2.5}$ concentration for the majority of southeast Ontario to be at least 0.06 µg/m$^3$. Using that assumption, and a published value for baseline mortality incident rates in Canada (Public Health Agency of Canada 2010), we estimate that the premature mortality impacts due to emissions of the modeled EGUs in Canada to be at least 30 additional cases of premature mortality per year.

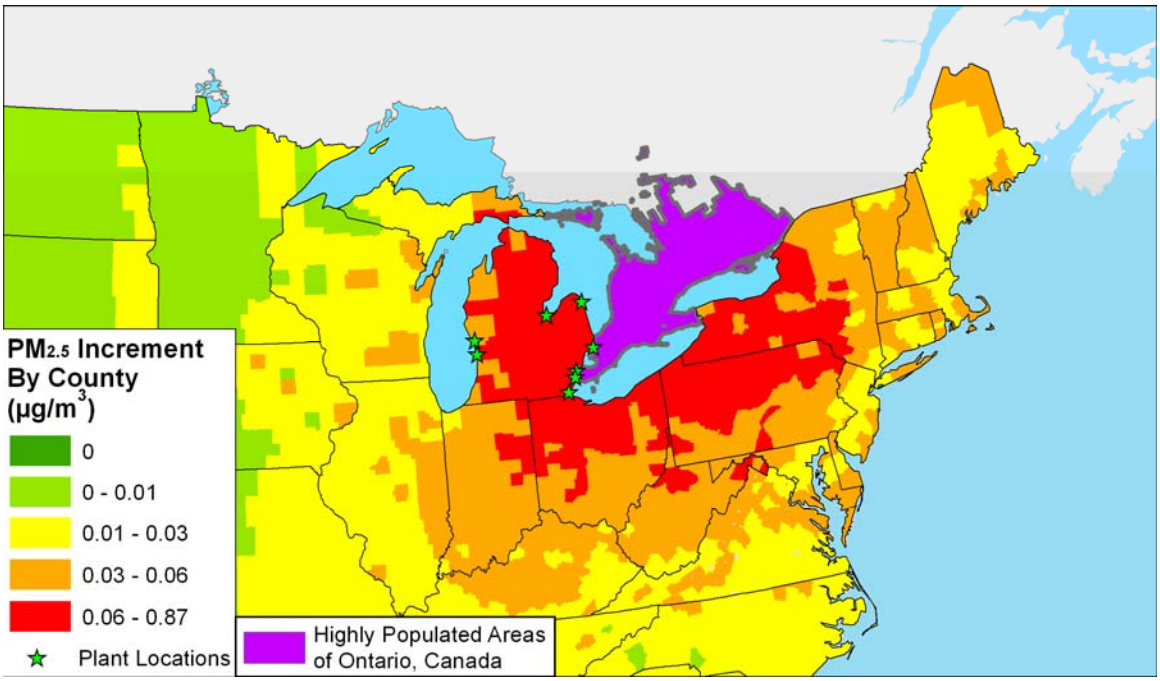


**Figure 4.4** Annual Average Increment of Fine Particle Concentrations (µg/m$^3$) in the Upper Midwest and Northeast Associated with Michigan Coal-fired Electricity Generating Unit Emissions

## 4.3 ECONOMIC VALUATION OF PUBLIC HEALTH IMPACTS

We estimate the annual national and Michigan-specific health-related damages associated with $PM_{2.5}$ emissions from the nine coal-fired EGUs to be $5.4 billion and $1.5 billion respectively. Table 4.3 displays the EGU specific impacts. The underlying incremental health impacts are presented in Table 4.4, which depicts the annual mortality and morbidity impacts for Michigan and the continental U.S. It should be noted that all of the estimates above are rounded to whole numbers.

**Table 4.3** Valuation of Annual Plant-specific Public Health Damages Associated with Contributions of Older Coal-fired Power Plants in Michigan to Fine Particle Levels in Air

| Electricity Generating Units | Economic Impact ($ million) | |
|---|---|---|
| | Michigan* | Continental U.S.* |
| BC Cobb | 68 | 450 |
| Dan E Karn / JC Weadock | 120 | 720 |
| Harbor Beach | 11 | 63 |
| JH Campbell | 150 | 700 |
| JR Whiting | 560 | 1,040 |
| River Rouge | 340 | 780 |
| St. Claire | 65 | 560 |
| Trenton Channel | 140 | 1,080 |
| Total | 1,500 | 5,400 |

**Table 4.4** Valuation of Annual Outcome-specific Public Health Damages Associated with Contributions of Older Coal-fired Power Plants in Michigan to Fine Particle Levels in Air

| Plant | Economic Impact ($ million) | |
|---|---|---|
| | Michigan | National |
| Premature mortality | 1,440 | 5,300 |
| Cardiovascular hospital admissions | 1 | 4 |
| Respiratory hospital admissions | 1 | 3 |
| Asthma emergency room visit | 0.05 | 0.2 |
| Asthma exacerbation | 3 | 13 |
| Chronic bronchitis | 33 | 120 |

## 4.4 REASONABLENESS OF HEALTH IMPACT ESTIMATES

In this section, we discuss some of the assumptions within our analysis and their implications to demonstrate that our approach to calculating the annual mortality and morbidity impacts from the removal of emissions from the nine coal-fired EGUs is reasonable.

One assumption that slightly underestimates the public health impacts is the utilization of 2000 population information from the 2010 U.S. Census. The population of Michigan over that time period is expected to be stable, however, the population in the U.S. is expected to increase by 39 million people (Woods and Poole Economics 2006).

For the concentration-response functions derived above, alternative values could have been determined that may have either increased or decreased the public health impacts estimates. For example, for premature mortality from $PM_{2.5}$, the 12 expert opinions from the an expert elicitation (Industrial Economics 2006) gave median concentration-response functions ranging from 0.4% to 2.0% (versus our estimate of 1.0%). Therefore, applying either of the extreme values could either double or halve our public health impact estimates. However, neither would represent the best interpretation of the current scientific literature in our view. Had we used the median value among the expert opinions, our estimate of premature mortality impacts would have differed (increased) by only 5% from our present estimates.

Our estimates of public health impacts are also influenced by the baseline incidence of mortality and morbidity used in the HIA calculations. Our incidence estimates reflect the best information currently available. However, rates may change as the population ages, as medical science advances, and as health care practices evolve. Specifying the direction and magnitude of the influence of these factors (and others) on baseline incidence of mortality and morbidity related to $PM_{2.5}$ are beyond the scope of this analysis. However, the net effect of these factors would have to be on the order of 10% or more to be significant compared to the effect of the forecasted population growth.

In general, the reasonableness of our estimates is informed by the widely accepted nature of our methodology and the similarity between our outputs and those from related studies. As discussed above, the methodology used has been widely vetted in the scientific and regulatory community and is used by the EPA to report to the U.S. Office of Management and Budget and others on the benefits of various air pollution control programs. The body of epidemiological literature for health effects of particulate matter is large and robust, and is supported by good mechanistic understanding of how these pollutants can influence human health. Premature mortality and morbidity attributable to fossil fuel-related particulate matter persist in the U.S. and reduction in emissions from coal-fired power plants will have health benefits of the magnitude estimated within this report.

# 5.0 CONCLUSIONS

Retiring the nine coal-fired EGUs featured in this study would reduce emissions of $PM_{2.5}$ by 11,300 tons per year and sulfur dioxide and nitrogen oxides by 194,000 tons per year. As a consequence, $PM_{2.5}$ exposure will be reduced for millions of people in those states. For each year the emissions from the modeled EGUs are not released, approximately 180 lives will be extended in Michigan. But since the byproducts of coal-fired power plant emissions are dispersed over great distances, over 480 additional lives are lost annually in 39 other states influenced by these sources. Furthermore, we estimate that 30 lives would be lost annually in impacted areas of Canada. Other important health impacts, such as asthma morbidity, chronic bronchitis, hospital admissions due to pollutant exposures are associated with emissions from these EGUs. We estimate that the annual economic impacts associated with these health impacts to be approximately $1.5 billion in the State of Michigan and $5.4 nationwide.

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# APPENDIX A

# DETAILED RESULTS

# DETAILED RESULTS

**Table A.1** Annual Reduction in County-Specific Mortality and Morbidity Associated with Reduced Air Pollutant Emissions from Michigan Coal-fired Electric Generating Units

| County* | Premature Deaths | Respiratory Hospital Admissions | Cardiovascular Hospital Admissions | Emergency Room Visits for Asthma | Asthma Exacerbations | Chronic Bronchitis | Minor Restricted Activity Days |
|---|---|---|---|---|---|---|---|
| Wayne | 100 | 30 | 21 | 76 | 37,000 | 40 | 38,000 |
| Oakland | 12 | 4.7 | 3 | 12 | 5,700 | 6.7 | 6,300 |
| Macomb | 11 | 3.3 | 2.6 | 8.4 | 4,000 | 4.7 | 4,300 |
| Monroe | 8.9 | 3.1 | 2 | 8 | 3,900 | 4.3 | 4,100 |
| Genesee | 4.2 | 1.3 | 0.9 | 3.4 | 1,600 | 1.8 | 1,700 |
| Kent | 3.6 | 1.5 | 0.9 | 3.8 | 1,800 | 1.9 | 1,900 |
| St. Clair | 3.4 | 1.1 | 0.8 | 2.8 | 1,300 | 1.5 | 1,400 |
| Bay | 3 | 0.8 | 0.7 | 2.1 | 1,000 | 1.2 | 1,100 |
| Saginaw | 2.8 | 0.8 | 0.6 | 2 | 980 | 1.1 | 1,000 |
| Washtenaw | 1.7 | 0.9 | 0.4 | 2.3 | 1,100 | 1.2 | 1,300 |
| Ingham | 1.6 | 0.7 | 0.4 | 1.7 | 790 | 0.8 | 930 |
| Lenawee | 1.3 | 0.4 | 0.3 | 1.1 | 510 | 0.6 | 540 |
| Calhoun | 1.2 | 0.3 | 0.3 | 0.8 | 400 | 0.5 | 420 |
| Jackson | 1.1 | 0.3 | 0.2 | 0.8 | 400 | 0.5 | 430 |
| Ottawa | 1.1 | 0.5 | 0.3 | 1.3 | 640 | 0.7 | 680 |
| Huron | 1 | 0.2 | 0.3 | 0.6 | 280 | 0.3 | 270 |
| Kalamazoo | 1 | 0.3 | 0.2 | 0.9 | 420 | 0.5 | 470 |
| Shiawassee | 0.8 | 0.3 | 0.2 | 0.7 | 330 | 0.4 | 350 |
| Muskegon | 0.7 | 0.2 | 0.2 | 0.6 | 270 | 0.3 | 270 |
| Berrien | 0.7 | 0.2 | 0.2 | 0.5 | 250 | 0.3 | 250 |
| Livingston | 0.7 | 0.4 | 0.2 | 0.9 | 440 | 0.5 | 470 |
| Lapeer | 0.6 | 0.3 | 0.1 | 0.7 | 320 | 0.4 | 350 |
| Tuscola | 0.6 | 0.2 | 0.1 | 0.5 | 230 | 0.3 | 240 |
| Eaton | 0.6 | 0.2 | 0.1 | 0.5 | 250 | 0.3 | 270 |
| Midland | 0.5 | 0.2 | 0.1 | 0.5 | 240 | 0.3 | 250 |
| Montcalm | 0.5 | 0.2 | 0.1 | 0.4 | 200 | 0.2 | 210 |
| Gratiot | 0.5 | 0.1 | 0.1 | 0.3 | 160 | 0.2 | 170 |
| Sanilac | 0.5 | 0.1 | 0.1 | 0.3 | 150 | 0.2 | 150 |
| Hillsdale | 0.4 | 0.1 | 0.1 | 0.3 | 160 | 0.2 | 170 |
| Alpena | 0.4 | 0.1 | 0.1 | 0.3 | 130 | 0.2 | 130 |
| Van Buren | 0.4 | 0.1 | 0.1 | 0.4 | 170 | 0.2 | 170 |
| Isabella | 0.4 | 0.2 | 0.1 | 0.5 | 220 | 0.2 | 270 |
| Iosco | 0.4 | 0.1 | 0.1 | 0.2 | 97 | 0.1 | 94 |
| Ionia | 0.4 | 0.2 | 0.1 | 0.4 | 180 | 0.2 | 200 |
| St. Joseph | 0.4 | 0.1 | 0.1 | 0.3 | 140 | 0.2 | 140 |
| Barry | 0.4 | 0.1 | 0.1 | 0.3 | 160 | 0.2 | 160 |
| Clinton | 0.3 | 0.1 | 0.1 | 0.4 | 170 | 0.2 | 180 |
| Grand Traverse | 0.3 | 0.1 | 0.1 | 0.3 | 140 | 0.2 | 150 |
| Roscommon | 0.3 | 0.1 | 0.1 | 0.2 | 74 | 0.1 | 73 |
| Allegan | 0.3 | 0.1 | 0.1 | 0.3 | 140 | 0.2 | 140 |
| Branch | 0.3 | 0.1 | 0.1 | 0.2 | 110 | 0.1 | 120 |
| Cass | 0.3 | 0.1 | 0.1 | 0.2 | 110 | 0.1 | 120 |
| Clare | 0.3 | 0.1 | 0.1 | 0.2 | 79 | 0.1 | 80 |
| Ogemaw | 0.3 | 0.1 | 0.1 | 0.1 | 69 | 0.1 | 69 |
| Gladwin | 0.2 | 0.1 | 0.1 | 0.1 | 69 | 0.1 | 70 |
| Mecosta | 0.2 | 0.1 | 0.1 | 0.2 | 92 | 0.1 | 100 |

**Table A.1** Continued

| County* | Premature Deaths | Respiratory Hospital Admissions | Cardiovascular Hospital Admissions | Emergency Room Visits for Asthma | Asthma Exacerbations | Chronic Bronchitis | Minor Restricted Activity Days |
|---|---|---|---|---|---|---|---|
| Charlevoix | 0.2 | 0.1 | 0.1 | 0.2 | 79 | 0.1 | 80 |
| Otsego | 0.2 | 0.1 | 0.1 | 0.2 | 83 | 0.1 | 85 |
| Arenac | 0.2 | 0 | 0 | 0.1 | 57 | 0.1 | 60 |
| Mason | 0.2 | 0 | 0 | 0.1 | 58 | 0.1 | 60 |
| Newaygo | 0.2 | 0.1 | 0 | 0.2 | 78 | 0.1 | 78 |
| Emmet | 0.2 | 0.1 | 0 | 0.1 | 68 | 0.1 | 71 |
| Presque Isle | 0.2 | 0 | 0 | 0.1 | 46 | 0.1 | 46 |
| Alcona | 0.2 | 0 | 0 | 0.1 | 38 | 0.1 | 37 |
| Chippewa | 0.2 | 0.1 | 0 | 0.2 | 70 | 0.1 | 82 |
| Marquette | 0.2 | 0 | 0 | 0.1 | 58 | 0.1 | 66 |
| Montmorency | 0.2 | 0 | 0 | 0.1 | 37 | 0 | 36 |
| Wexford | 0.2 | 0 | 0 | 0.1 | 59 | 0.1 | 60 |
| Antrim | 0.2 | 0 | 0 | 0.1 | 51 | 0.1 | 51 |
| Osceola | 0.2 | 0 | 0 | 0.1 | 55 | 0.1 | 55 |
| Cheboygan | 0.1 | 0 | 0 | 0.1 | 41 | 0.1 | 41 |
| Crawford | 0.1 | 0 | 0 | 0.1 | 42 | 0.1 | 43 |
| Manistee | 0.1 | 0 | 0 | 0.1 | 35 | 0 | 36 |
| Kalkaska | 0.1 | 0 | 0 | 0.1 | 37 | 0 | 39 |
| Delta | 0.1 | 0 | 0 | 0.1 | 30 | 0 | 31 |
| Oscoda | 0.1 | 0 | 0 | 0.1 | 28 | 0 | 27 |
| Lake | 0.1 | 0 | 0 | 0 | 23 | 0 | 24 |
| Mackinac | 0.1 | 0 | 0 | 0.1 | 26 | 0 | 27 |
| Oceana | 0.1 | 0 | 0 | 0.1 | 36 | 0 | 36 |
| Missaukee | 0.1 | 0 | 0 | 0.1 | 33 | 0 | 33 |
| Menominee | 0.1 | 0 | 0 | 0 | 22 | 0 | 23 |
| Leelanau | 0.1 | 0 | 0 | 0.1 | 31 | 0 | 31 |
| Dickinson | 0.1 | 0 | 0 | 0 | 21 | 0 | 20 |
| Benzie | 0.1 | 0 | 0 | 0 | 22 | 0 | 23 |
| Houghton | 0.1 | 0 | 0 | 0 | 18 | 0 | 20 |
| Luce | 0.1 | 0 | 0 | 0 | 11 | 0 | 13 |
| Iron | 0 | 0 | 0 | 0 | 8.6 | 0 | 8.1 |
| Gogebic | 0 | 0 | 0 | 0 | 7.8 | 0 | 7.8 |
| Schoolcraft | 0 | 0 | 0 | 0 | 9 | 0 | 9.2 |
| Alger | 0 | 0 | 0 | 0 | 8.8 | 0 | 9.7 |
| Ontonagon | 0 | 0 | 0 | 0 | 4.9 | 0 | 5 |
| Baraga | 0 | 0 | 0 | 0 | 5 | 0 | 5.3 |
| Keweenaw | 0 | 0 | 0 | 0 | 1.3 | 0 | 1.2 |

* Counties are listed in descending order by cases of premature mortality.

**Table A.2** Annual Reduction in State-Specific Mortality and Morbidity Associated with Reduced Air Pollutant Emissions from Michigan Coal-fired Electric Generating Units

| State* | Premature Deaths | Respiratory Hospital Admissions | Cardiovascular Hospital Admissions | Emergency Room Visits for Asthma | Asthma Exacerbations | Chronic Bronchitis | Minor Restricted Activity Days |
|---|---|---|---|---|---|---|---|
| Michigan | 180 | 55 | 38 | 140 | 68,000 | 76 | 72,000 |
| Ohio | 73 | 21 | 16 | 54 | 26,000 | 29 | 27,000 |
| Pennsylvania | 70 | 20 | 19 | 37 | 22,000 | 26 | 23,000 |
| New York | 58 | 21 | 16 | 38 | 23,000 | 26 | 24,000 |
| Illinois | 29 | 9.6 | 6.7 | 24 | 12,000 | 13 | 13,000 |
| Indiana | 24 | 7.6 | 5.5 | 19 | 9,400 | 10 | 10,000 |
| New Jersey | 20 | 7 | 5.5 | 13 | 7,700 | 9.1 | 8,300 |
| Massachusetts | 16 | 5.6 | 4.4 | 10 | 6,100 | 7.2 | 6,600 |
| North Carolina | 14 | 4.6 | 2.9 | 8 | 5,600 | 6.4 | 6,200 |
| Maryland | 13 | 4.5 | 2.7 | 7.9 | 5,600 | 6.4 | 6,100 |
| Kentucky | 12 | 3.7 | 2.4 | 6.4 | 4,500 | 5.1 | 4,900 |
| Tennessee | 12 | 3.6 | 2.4 | 6.3 | 4,400 | 5.1 | 4,800 |
| Virginia | 12 | 4.4 | 2.6 | 7.6 | 5,400 | 6.2 | 6,000 |
| Wisconsin | 11 | 3.8 | 2.8 | 9.9 | 4,700 | 5.4 | 5,100 |
| West Virginia | 9.9 | 2.4 | 1.9 | 4.2 | 2,900 | 3.5 | 3,200 |
| Missouri | 9.3 | 2.7 | 2.1 | 6.8 | 3,300 | 3.7 | 3,500 |
| Florida | 9.1 | 2.5 | 2.3 | 4.3 | 3,000 | 3.6 | 3,100 |
| Connecticut | 8.9 | 3.1 | 2.5 | 5.8 | 3,500 | 4.1 | 3,700 |
| Georgia | 8.7 | 3.3 | 1.7 | 5.8 | 4,100 | 4.5 | 4,500 |
| Texas | 7.8 | 3.1 | 1.7 | 5.4 | 3,800 | 4 | 4,100 |
| Iowa | 5.7 | 1.7 | 1.4 | 4.5 | 2,100 | 2.4 | 2,300 |
| Alabama | 5.5 | 1.5 | 1.1 | 2.7 | 1,900 | 2.1 | 2,000 |
| South Carolina | 5.2 | 1.7 | 1.1 | 2.9 | 2,000 | 2.3 | 2,200 |
| Arkansas | 4.3 | 1.2 | 0.9 | 2.1 | 1,500 | 1.6 | 1,500 |
| Minnesota | 4.3 | 1.6 | 1.1 | 4 | 1,900 | 2.2 | 2,100 |
| Oklahoma | 3.8 | 1.1 | 0.8 | 1.8 | 1,300 | 1.4 | 1,400 |
| Rhode Island | 3.7 | 1.2 | 1 | 2.2 | 1,300 | 1.5 | 1,400 |
| Maine | 3.6 | 1.1 | 1 | 2.1 | 1,200 | 1.5 | 1,300 |
| Louisiana | 3.3 | 1 | 0.6 | 1.7 | 1,200 | 1.3 | 1,300 |
| New Hampshire | 3 | 1.2 | 0.9 | 2.2 | 1,300 | 1.5 | 1,400 |
| Mississippi | 2.9 | 0.8 | 0.5 | 1.5 | 1,000 | 1.1 | 1,100 |
| Kansas | 2.7 | 0.9 | 0.6 | 2.2 | 1,100 | 1.2 | 1,100 |
| Nebraska | 2.5 | 0.8 | 0.6 | 2 | 950 | 1.1 | 1,000 |
| Delaware | 2.4 | 0.8 | 0.5 | 1.3 | 930 | 1.1 | 1,000 |
| Vermont | 2.1 | 0.8 | 0.6 | 1.4 | 850 | 1 | 920 |
| District of Columbia | 1.5 | 0.4 | 0.3 | 0.7 | 510 | 0.6 | 610 |
| South Dakota | 1 | 0.3 | 0.2 | 0.8 | 370 | 0.4 | 390 |
| North Dakota | 0.7 | 0.2 | 0.2 | 0.5 | 250 | 0.3 | 270 |
| Colorado | 0.6 | 0.2 | 0.1 | 0.3 | 340 | 0.4 | 380 |
| Arizona | 0.5 | 0.1 | 0.1 | 0.2 | 230 | 0.3 | 240 |
| New Mexico | 0.2 | 0.1 | 0 | 0.1 | 100 | 0.1 | 100 |
| Montana | 0.2 | 0 | 0 | 0.1 | 74 | 0.1 | 78 |
| Nevada | 0.2 | 0.1 | 0 | 0.1 | 93 | 0.1 | 100 |
| Utah | 0.1 | 0.1 | 0 | 0.1 | 90 | 0.1 | 90 |
| Idaho | 0.1 | 0 | 0 | 0 | 64 | 0.1 | 65 |
| Wyoming | 0.1 | 0 | 0 | 0 | 49 | 0.1 | 52 |
| California | 0.1 | 0 | 0 | 0 | 47 | 0 | 47 |
| Washington | 0.1 | 0 | 0 | 0 | 33 | 0 | 35 |
| Oregon | 0 | 0 | 0 | 0 | 7.8 | 0 | 8.2 |

* States are listed in descending order by cases of premature mortality.

# APPENDIX B

# ADDITIONAL FIGURES

# ADDITIONAL FIGURES

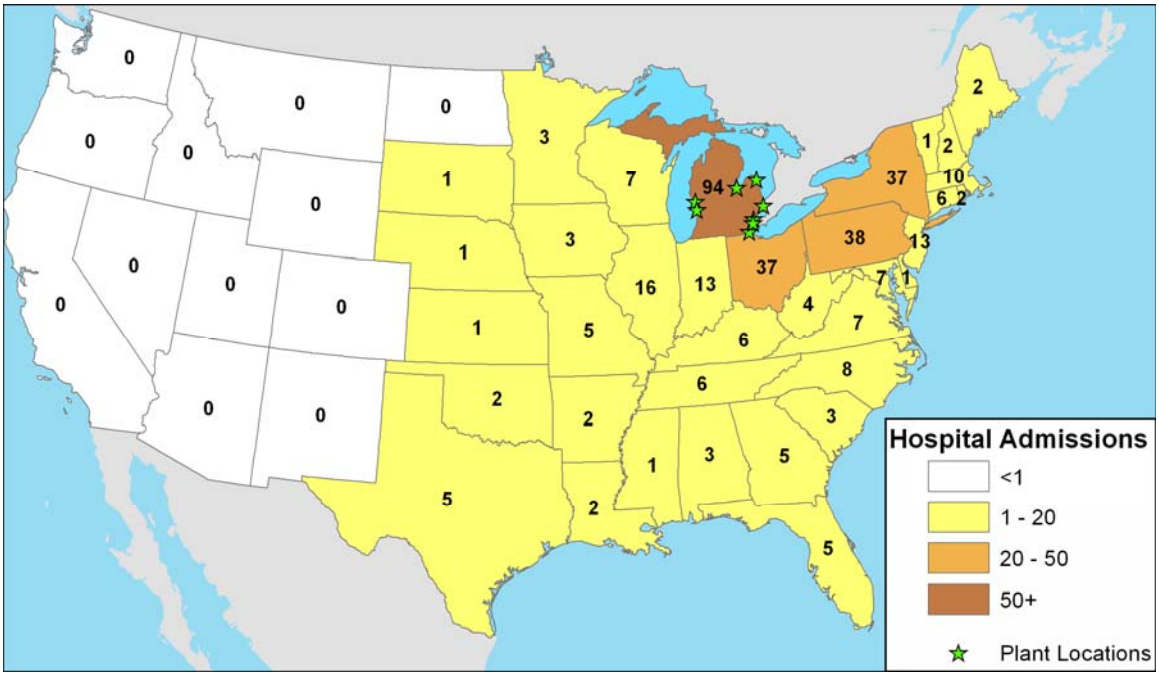


**Figure B.1** Estimated Annual Number of Hospital Admissions Associated with Emissions for Older Coal-fired Electricity Generating Units in Michigan

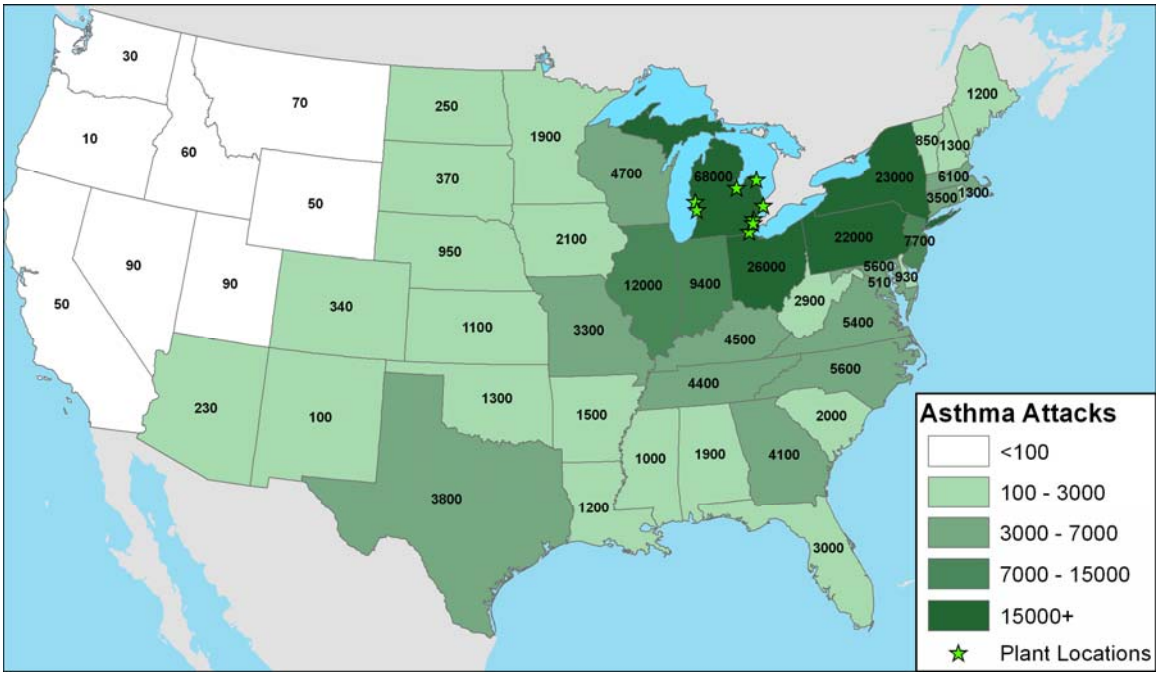


**Figure B.2** Estimated Annual Number of Asthma Attacks for Each Year Associated with Emissions for Older Coal-fired Electricity Generating Units in Michigan

# APPENDIX C

# ABOUT EH&E

## ABOUT EH&E

Environmental Health & Engineering, Inc. (EH&E) is a professional services firm established in 1987 to provide businesses and institutions with a reliable resource for environmental consulting and engineering services. The company is composed of 75 highly-trained individuals based in Needham, Massachusetts, who share a passion and enthusiasm for excellence.

EH&E has a combination of depth of knowledge and credibility that is unsurpassed in the environmental services industry. The knowledge base at EH&E includes doctoral level scientists with degrees from, and academic appointments at, some of the most prestigious universities in the world including Harvard University, Brandeis University, Boston University and the University of Massachusetts. These scientists are supported by a wide range of Masters and Bachelor level support staff with degrees in fields such as chemistry, biology, engineering and environmental science. Many EH&E employees have nationally or internationally recognized certifications, e.g., Certified Industrial Hygienist, Certified Safety Professional, and Professional Engineer. The EH&E team is also active in professional organizations such as the International Society for Exposure Science, International Society for Indoor Air Quality, and American Industrial Hygiene Association. The academic and real world experience of our team members are part of what makes EH&E different. Furthermore, cross-training in sciences and engineering at EH&E ensures that our staff have field and project management experiences that are necessary to meet the demands of high pressure projects.

EH&E scientists work with a wide variety of clients to evaluate, solve, and communicate environmental health risks in occupational, educational, and community settings. EH&E staff synthesizes information on exposure, toxicology, and epidemiology to characterize health status using multiple methods, including quantitative risk analyses, health impact assessment techniques, epidemiological analyses, and medical screening performed by environmental and occupational physicians. EH&E scientists possess deep understanding and knowledge of state-of-the-art measurement and modeling methods used to evaluate human exposure to

environmental stressors. Through these innovative practices, EH&E has produced some of the most up-to-date research and results in the field.

Among its numerous areas of expertise, EH&E's experts have a deep understanding of the current state of knowledge as well as the on-going studies in air pollution and health science underway at research centers across the U.S. and elsewhere. In addition, these individuals are active in air pollution health research and translation of that research to policy, and as a result have strong relationships with scientists and policymakers at EPA and several state-level regulatory organizations.

This project for the Michigan Environmental Council was led by David L. MacIntosh, Sc.D., C.I.H. In addition to his role as a Principal Scientist and Associate Director of Advanced Analytics and Building Science at EH&E, Dr. MacIntosh is an Adjunct Associate Professor of Environmental Health at the Harvard School of Public Health where he teaches a course on environmental exposure assessment to masters and doctoral degree students. Dr. MacIntosh also is a technical advisor to the World Health Organization and is the first-draft author of several publications for that organization including a *Toolkit for Human Health Risk Assessment* which was published in 2011. Other key contributors to this report were Theodore A. Myatt, Sc.D., and Matthew A. Kaufman of EH&E, and Jonathan I. Levy, Sc.D., of Boston University.