A Case Study: The Public Health Consequences of Air Emissions from Coal-Fired Power Plants in the St. Louis Area

Prepared for
American Coalition for Clean Coal Electricity (ACCCE)

Prepared by
Christopher M. Long, Sc.D., DABT
Peter A. Valberg, Ph.D.

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GRADIENT

www.gradientcorp.com
20 University Road
Cambridge, MA 02138
617-395-5000

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About Gradient

Gradient is an environmental and risk science consulting firm, nationally renowned for its specialties in toxicology, epidemiology, human health and ecological risk assessment, environmental/forensic chemistry, contaminant fate and transport modeling, risk-based remedial alternatives assessment, and the application of database management and Geographic Information Systems (GIS) tools for addressing environmental contamination. Since 1985, Gradient's staff scientists have served a combination of private and public sector clients, bringing sound science and creative solutions to a variety of complex and challenging environmental issues. Certified as a Women's Business Enterprise by the Center for Women and Enterprise, a regional partner of the Women's Business Enterprise National Council (WBENC), Gradient has its principal office in Cambridge, Massachusetts, and a second main office in Seattle, Washington.

Dr. Christopher Long is a principal scientist at Gradient and an expert in air pollution exposure assessment and inhalation risk assessment. Since joining Gradient 15 years ago, he has worked on a wide variety of indoor and outdoor air quality projects, assessing both potential exposures and health risks to numerous airborne substances that include airborne particulate matter (PM), diesel exhaust, engineered nanomaterials, asbestos, gaseous criteria pollutants, and a variety of air toxics. Prior to joining Gradient, he received a doctorate in Environmental Health from the Harvard School of Public Health, where he conducted a research study to characterize particulate matter mass concentrations, size distributions, and chemical composition inside and outside residential homes. He has prepared approximately 25 peer-reviewed journal articles or book chapters in the general areas of indoor and outdoor air pollution and exposure/risk assessment. He is certified as a Diplomate of the American Board of Toxicology (DABT) and is an Associate Editor for the Journal of Exposure Science and Environmental Epidemiology (JESEE).

Dr. Peter Valberg is a Principal at Gradient and an expert in human health risk assessment (HHRA), inhalation toxicology, and modeling of human exposure to environmental chemicals. He has 30 years of experience on the faculty of the Harvard School of Public Health and with health-risk assessment projects at Gradient. He has provided air quality and health risk expertise to the Department of Justice, the United States Environmental Protection Agency (US EPA), and the National Academy of Sciences. He is the author of more than 100 peer-reviewed scientific publications on biological effects of environmental exposures on humans and animals. His health risk assessment expertise covers air pollutants, chemical exposures, and radionuclides, with recent project work and publications addressing the health impacts of airborne particulate matter (PM), diesel exhaust, metals, and organics. He is often called upon to prepare and interpret health risk findings for a variety of audiences, and he regularly interprets and applies scientific research results to the regulatory, litigation, and public policy arenas. He holds advanced degrees from Harvard University in physics and human physiology, and he is a Fellow of the Academy of Toxicological Sciences (FATS).

Executive Summary

Gradient was asked to conduct a case study assessment of air quality and public health claims related to stack emissions from certain coal-fired power plants (CFPPs). Four Ameren Missouri CFPPs in the St. Louis area were selected for the case study assessment, including the 2,407-MW Labadie Plant in Franklin County, MO; the 839-MW Meramec Plant in St. Louis County, MO; the 1,204-MW Rush Island Plant in Jefferson County, MO; and the 986-MW Sioux Plant in St. Charles County, MO. Consistent with recent allegations of public health impacts from coal-fired power plant emissions (*e.g.*, CATF, 2014; EIP, 2012), our analysis is primarily focused on the incremental impact of the Ameren CFPP emissions on levels of airborne particulate matter (PM), and specifically fine particulate matter ($PM_{2.5}$).[1] In addition, we analyzed air quality data for several additional criteria air pollutants – ozone (O_3), nitrogen dioxide (NO_2),[2] and sulfur dioxide (SO_2) – that are either directly emitted by coal-fired power plants (NO_2, SO_2) or that can be formed from coal-fired power plant emissions (O_3), and that have been linked with air pollution health effects.

Our evaluation can be summarized by the following six main findings, which are expanded upon in the corresponding sections of this report:

1. Air quality in the St. Louis Core Based Statistical Area (CBSA) is similar to, and often better than, that of other regions of the U.S. (Section 1)
2. Air quality in the St. Louis Area has shown significant improvement in recent years, with air pollutant levels at almost all monitoring locations now in compliance with the health-protective NAAQS. (Section 2)
3. Expected incremental ambient $PM_{2.5}$ air quality impacts due to air emissions from the four Ameren CFPPs are small compared to everyday ambient $PM_{2.5}$ exposure levels in the St. Louis Area. (Section 3).
4. Total personal exposures to air pollutants will generally be dominated by contributions from other common indoor and outdoor sources rather than by contributions from local CFPPs. (Section 4)
5. Hypothetical health impacts calculated for Ameren CFPP air emissions are unreliable because of many hidden assumptions, uncertainties, and limitations. (Section 5)
6. Asthma is a complex, multi-factorial disease, with a multitude of known triggers and risk factors. Scientific studies provide evidence that asthma prevalence (that is, the percentage of people with doctor-diagnosed asthma) and morbidity are more closely linked to allergic status, lifestyle factors, and indoor air pollution than to indicators of outdoor air pollution exposure. In fact,

[1] $PM_{2.5}$, also referred to as "fine" PM, is a commonly used indicator of respirable particles that is defined as including particles with aerodynamic diameters of 2.5 μm and smaller (a μm is one-millionth of a meter). Based on the current recognition that $PM_{2.5}$ better represents the particle sizes that can penetrate deep into the lungs and elicit adverse health effects, the current U.S. EPA NAAQS for PM are focused on $PM_{2.5}$. Currently, the PM NAAQS include an annual average $PM_{2.5}$ standard of 12 $\mu g/m^3$ and a 24-hour $PM_{2.5}$ standard of 35 $\mu g/m^3$. In addition, there remains a 24-hour standard (150 $\mu g/m^3$) for PM_{10} from the prior time period (1987 to 1997) when the PM NAAQS were focused on PM_{10} (*i.e.*, particles with aerodynamic diameters of 10 μm and smaller).

[2] NO_2 has been adopted by U.S. EPA as the indicator for the criteria air pollutant oxides of nitrogen (NO_x) that consist of all oxidized nitrogen compounds, including gases such as NO_2 and nitric oxide (NO). NO_x also reacts with other species forming particulate matter. The NAAQS for NO_x is thus specified in terms of NO_2, and NO_2 is more routinely monitored than other NO_x species.

ambient air pollutant emissions and concentrations have decreased significantly over the past several decades while the prevalence of asthma has increased, providing support for the conclusion that factors other than exposure to outdoor air pollutants are more important risk factors underlying the trends in increased asthma prevalence. (Section 6)

As a centerpiece to our analysis, we relied upon actual air-quality measurement data for the St. Louis area, making comparisons with data for other urban areas and with the health-based U.S. EPA National Ambient Air Quality Standards (NAAQS) to discern if there are potential public health impacts of the Ameren CFPP stack emissions. In data comparisons that are provided in Sections 1 and 2, we evaluated both historical and recent monitoring data against today's health-protective primary NAAQS, which are required to be set at levels that protect even the most sensitive groups from air pollution health effects. Ambient air monitoring data are a useful screening tool for assessing the public health impacts of air pollution sources such as coal-fired power plants, but importantly, measured concentrations reflect not only the air quality impacts of specific local sources such as the Ameren CFPPs, but also the combined air quality contributions of a variety of local and distant natural and anthropogenic air pollutant sources. As a result, in Section 3, we estimated the incremental $PM_{2.5}$ air quality impacts of the four Ameren St. Louis-area CFPPs and compared this estimate to the total, all-source measured $PM_{2.5}$ levels at St. Louis-area air quality monitors; our findings indicate that the CFPPs contribute a very small portion to overall $PM_{2.5}$ levels in St. Louis.

We examined carefully the reliability of health impact analyses that purport to quantify excess morbidity and mortality associated with CFPP stack emissions, focusing specifically on the Clean Air Task Force (CATF) and the Environmental Integrity Project (EIP) model calculations, which included the Ameren CFPPs as examples. As discussed in Section 5, rather than relying on actual data, these model calculations have projected health claims that are hypothetical and overstated. We highlight why these projections are not reliable because of the underlying assumptions, methods, and data inputs. We discuss several recent epidemiologic studies that call into question the causal connection between small increments in ambient $PM_{2.5}$ and premature mortality, including the Cox and Popken (2015) study that cautioned, "[Our] findings suggest that predicted substantial human longevity benefits resulting from reducing $PM_{2.5}$ and ozone may not occur, or may be smaller than previously estimated." Overall, we concluded that, when compliance with the $PM_{2.5}$ NAAQS and the uncertainties in the modeling approach and its estimated impacts are properly considered, it becomes clear that the health effects calculated by environmental organizations for Ameren power plant emissions are theoretical and may well be zero.

Other sections in the report provide background information on typical everyday air pollution exposures that derive from common indoor sources, other outdoor sources, and personal sources (Section 4); and on what is known regarding agents that cause or trigger asthma (Section 6).

Each subsequent section of this report summarizes the key scientific data underlying our six main findings. For each finding, we provide comments, graphs, and/or tables that explain the basis for the finding. We also include key references for the data sources that underlie our analyses. As the following materials show, our findings are supported by a body of scientific evidence, including actual measurement data and scientific data from peer-reviewed research literature, governmental reports, and technical analyses. This work was sponsored by the American Coalition for Clean Coal Electricity (ACCCE).

1 Air quality in the St. Louis CBSA is similar to, and often better than, that of other regions of the U.S.

For the case study analysis of the air quality impacts of Ameren's four St. Louis-area coal-fired power plants (CFPPs), we compared air monitoring data from central-site ambient monitors in the St. Louis Core Based Statistical Area (CBSA)[3] to the corresponding data for other urban CBSAs across the country. Ambient monitors within the entire St. Louis CBSA were included in our assessment given the location of the four Ameren CFPPs in four counties surrounding the City of St. Louis (see Figure 1.1 showing the locations of Labadie Plant in Franklin County, MO; the Meramec Plant in St. Louis County, MO; the Rush Island Plant in Jefferson County, MO; and the Sioux Plant in St. Charles County, MO). The St. Louis CBSA consists of the city of St. Louis, seven nearby counties in Missouri (Franklin, Jefferson, Lincoln, St. Charles, St. Louis, Warren, and Washington), and eight nearby counties in Illinois (Bond, Calhoun, Clinton, Jersey, Macoupin, Madison, Monroe, and St. Clair).

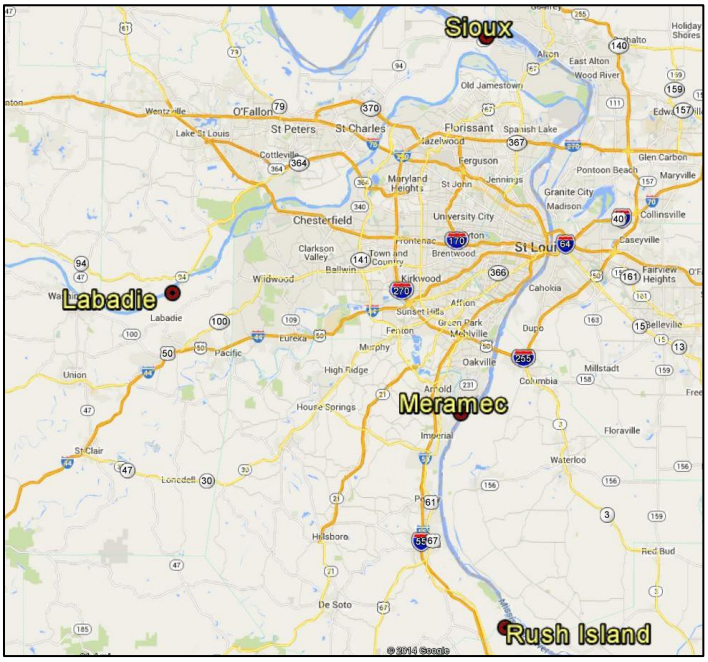


Figure 1.1 Locations of the Four St. Louis-area Ameren Coal-fired Power Plants (CFPPs)

Focusing on four U.S. EPA criteria air pollutants that are related to CFPP stack air emissions (fine particulate matter-$PM_{2.5}$, ozone-O_3, sulfur dioxide-SO_2, and nitrogen dioxide-NO_2), Figures 1.2 through 1.7 compare ambient air quality levels in the St. Louis CBSA to levels in the Chicago, Cleveland, Cincinnati, Houston, Los Angeles, Phoenix, and Pittsburgh CBSAs. As provided in 2013 design value reports obtained from the U.S. EPA Air Trends website,[4] data shown in the figures reflect the highest

[3] A CBSA is defined by the Office of Management and Budget (OMB) as a region with at least one core urbanized area having a population at or above 10,000 and a surrounding area with a high degree of social and economic integration based on community ties (OMB, 2010).
[4] http://www.epa.gov/airtrends/values.html.

reported valid three-year (2011-2013) design values at any of the monitoring sites located within the respective CBSAs.[5] In other words, the data represent upper-bounds of air quality levels in each CBSA for data statistics matching the corresponding form of the U.S. EPA primary (health-based) NAAQS for each pollutant: for $PM_{2.5}$, annual average (mean) concentrations and 98th percentiles of 24-hour concentrations; for O_3, 4th highest daily maximums of 8-hour concentrations; for SO_2, 99th percentiles of 1-hour concentrations; and for NO_2, 98th percentiles of 1-hour concentrations and annual average (mean) concentrations.

As shown in Figures 1.2 through 1.7 below, air quality in the St. Louis CBSA is either similar to or better than that of many other major CBSAs across the United States. In addition, Figures 1.2 and 1.3 indicate that 24-hour and annual average $PM_{2.5}$ levels in the St. Louis CBSA are below the NAAQS, while Figures 1.6 and 1.7 indicate that 1-hour and annual average NO_2 levels in the St. Louis CBSA are well below the respective NAAQS. Regarding annual $PM_{2.5}$, the U.S. EPA lowered the annual $PM_{2.5}$ NAAQS from 15 $\mu g/m^3$ to 12 $\mu g/m^3$ (micrograms per cubic meter) in December 2012, and just recently announced in December 2014 its final area designations for the 2012 annual $PM_{2.5}$ NAAQS.[6] While the St. Louis area was not among the 14 areas in six states that were designated by U.S. EPA as "nonattainment," it was designated as "unclassifiable" due to insufficient quality-assured data to determine compliance with the NAAQS for the Illinois portion of the area that has typically provided the highest design values in the area. Although a number of counties in the St. Louis CBSA were formerly designated as "nonattainment" for the 1997 annual $PM_{2.5}$ NAAQS of 15 $\mu g/m^3$, Figure 1.3 shows that annual $PM_{2.5}$ concentrations in the St. Louis CBSA are now well below 15 $\mu g/m^3$, and are in fact also below 12 $\mu g/m^3$. In support of this, the Missouri Department of Natural Resources (MoDNR) has concluded that all monitors in Missouri are also now in attainment with the 2012 annual $PM_{2.5}$ NAAQS, with design values still trending downward (Moore, 2014).

For the one case where the 2011-2013 design values in the figures exceed the corresponding NAAQS (Figures 1.4 showing 8-Hour O_3 concentrations), data for all other cities are also above the NAAQS. This is further illustrated by Figure 1.8 that shows that nonattainment of the current 8-hour O_3 NAAQS is a regional problem for many parts of the country, including in the Northwest, Midwest, Texas, and California, and is not unique to the St. Louis area.

For 1-hour SO_2 levels, Figure 1.5 contains two bars for the St. Louis CBSA, both of which are below the 1-hour SO_2 NAAQS. The higher of the two values is based on SO_2 data for the Herculaneum (MO) area that is the former home to the last operating primary lead smelter in the U.S., the Doe Run smelter. Importantly, the Doe Run smelter, which was not only a major SO_2 emission source but also a major source of air lead emissions, closed at the end of 2013. An estimated 2013-2015 design value is shown for the Herculaneum monitor given that the 2011-2013 design value of 192 ppb was driven by SO_2 emissions from the former smelter; moreover, the 2011-2013 design value for the Herculaneum monitor was classified by US EPA as invalid due to the occurrence of monitoring quarters not meeting minimum data completeness criteria. Since the closure of the Doe Run smelter, SO_2 levels at the Herculaneum monitoring site have dropped significantly below the SO_2 NAAQS. For example, in 2014, the 99th percentile of 1-hour SO_2 concentrations at the Herculaneum monitoring site was 18 ppb and thus well below the 2011-2013 invalid design value of 192 ppb. For unofficial data through April 13, 2015 available on the U.S. AirData website[7], the maximum 1-hour SO_2 concentration is just 19 ppb; if SO_2 concentrations are assumed to remain similar for the remainder of 2015 at the Herculaneum monitoring

[5] There were a couple of cases where no valid 2011-2013 design values were available for a CBSA and only an invalid design value was provided in the US EPA design value report (1-hour NO_2 concentrations for the Chicago CBSA and 1-hour SO_2 concentrations for the Herculaneum area within the St. Louis CBSA). It is our understanding that these design values were classified as invalid due to the occurrence of monitoring quarters not meeting minimum data completeness criteria.
[6] http://www.epa.gov/airquality/particlepollution/designations/2012standards/regs.htm.
[7] http://www.epa.gov/airdata/

site, its 2013-2015 1-hour SO_2 design value would be approximately 60 ppb. Given these trends, it is thus expected that SO_2 concentrations in this area will continue to meet the SO_2 NAAQS given that the Doe Run smelter was the primary contributor to elevated SO_2 concentrations measured at the Herculaneum air monitors (MoDNR, undated). As illustrated by the second bar which is based on the highest 1-hour SO_2 design value for all other monitors in the St. Louis CBSA, SO_2 levels in Herculaneum are not representative of SO_2 levels in other parts of the St. Louis CBSA. In fact, outside of Herculaneum, 1-hour SO_2 levels are well below the NAAQS and lower than the levels in a number of the comparison CBSAs.

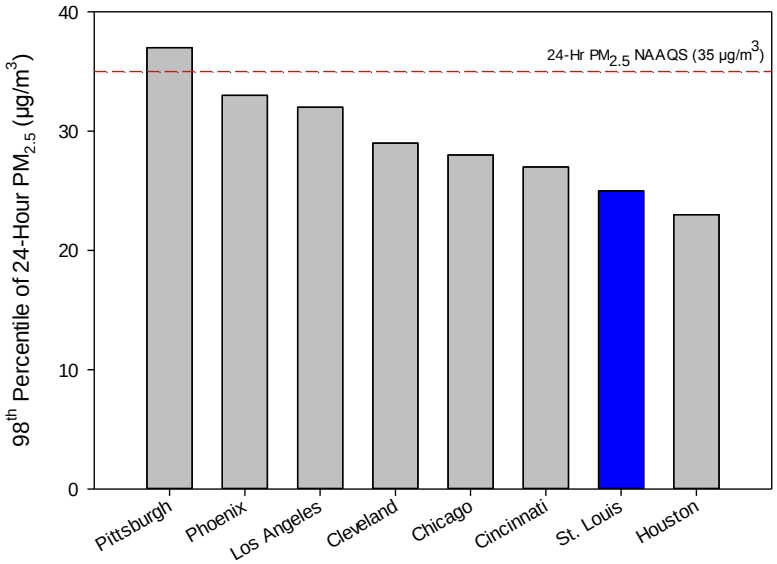


Figure 1.2 2011-2013 Design Values for 24-Hour $PM_{2.5}$ Concentrations for the St. Louis CBSA *vs*. Other Major U.S. CBSAs

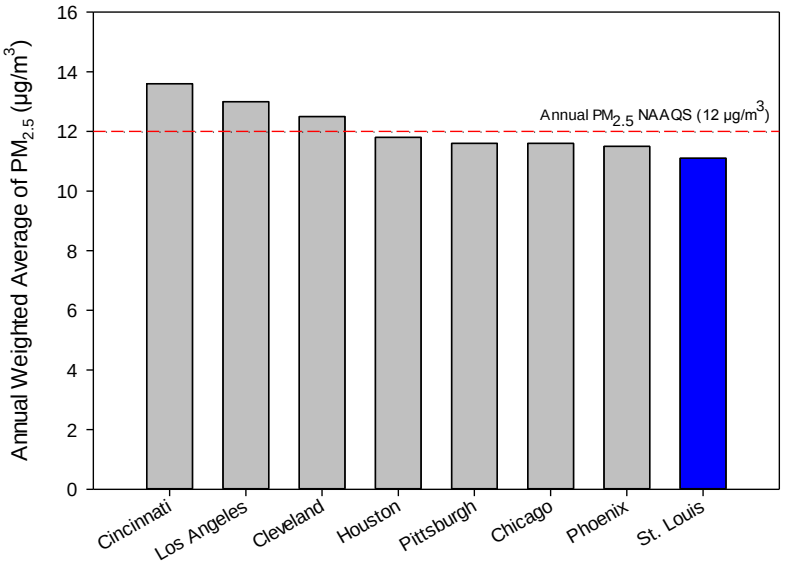


Figure 1.3 2011-2013 Design Values for Annual Average $PM_{2.5}$ Concentrations for the St. Louis CBSA *vs*. Other Major U.S. CBSAs

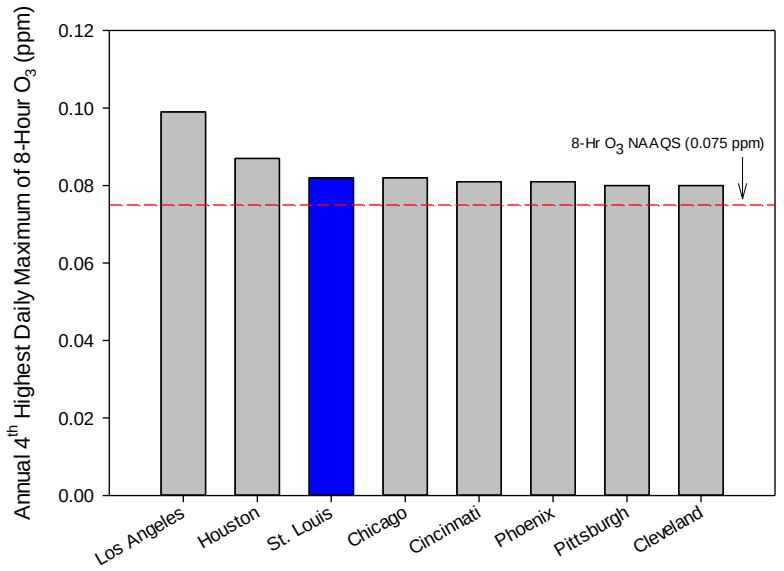


Figure 1.4 2011-2013 Design Values for Daily Maximum 8-Hour O_3 Concentrations for the St. Louis CBSA *vs*. Other Major U.S. CBSAs

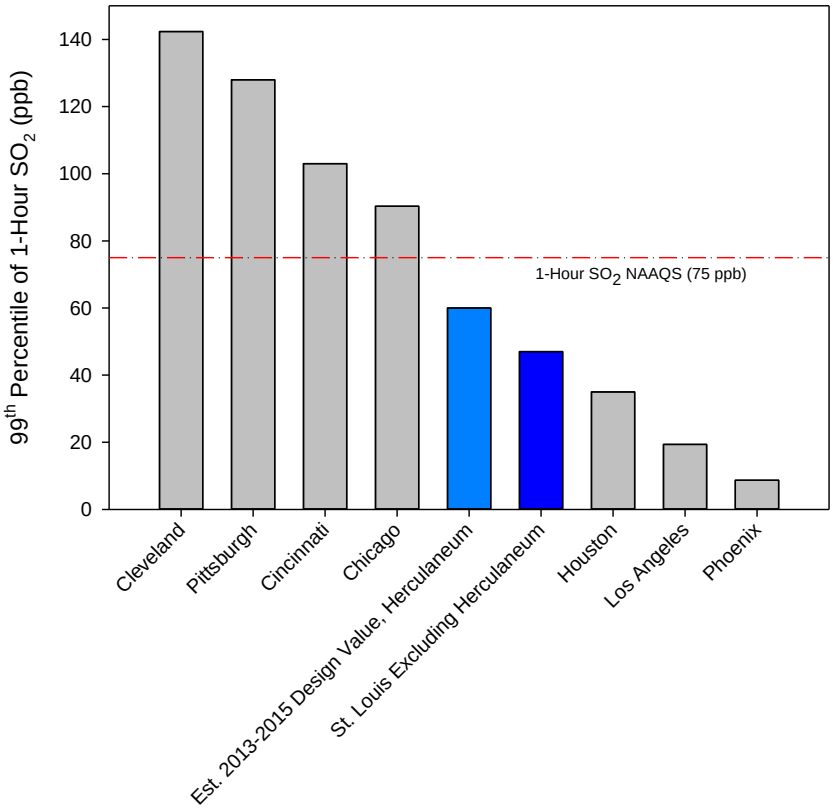


Figure 1.5 2011-2013 Design Values for 1-Hour SO_2 Concentrations for the St. Louis CBSA *vs*. Other Major U.S. CBSAs. Two bars are shown for the St. Louis CBSA, including one (Est. 2013-2015 Design Value, Herculaneum) that is based solely on data for the SO_2 monitor in Herculaneum (MO) and the other (St. Louis Excluding Herculaneum) that reflects all other SO_2 monitors in the St. Louis CBSA. An estimated 2013-2015 design value is shown for the Herculaneum monitor given that the 2011-2013 design value of 192 ppb was driven by SO_2 emissions from the Doe Run smelter that closed at the end of 2013; moreover, the 2011-2013 design value for the Herculaneum monitor was classified by US EPA as invalid due to the occurrence of monitoring quarters not meeting minimum data completeness criteria. The 2013-2015 estimated design value reflects the lower SO_2 concentrations that are now present in this area. As discussed in the text, a 2013-2015 design value of approximately 60 ppb is estimated using 2013-2014 official measurements and assuming that first quarter 2015 unofficial measurements will be representative of the entirety of 2015.

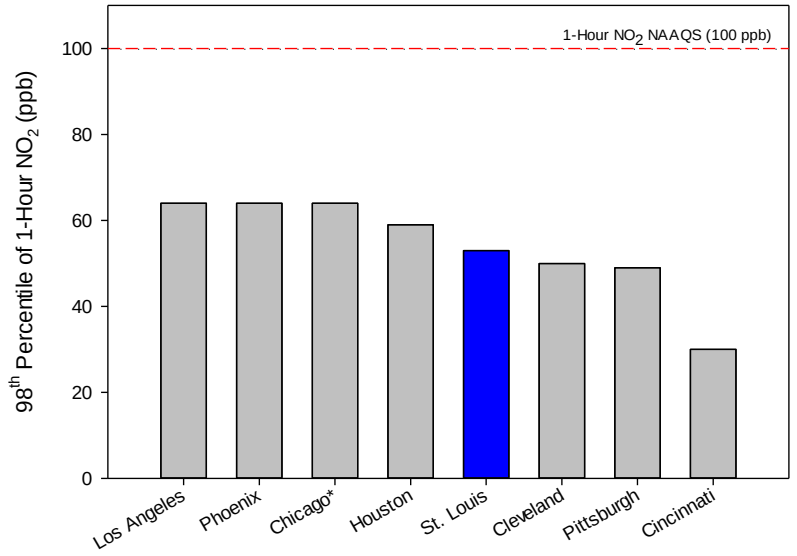


Figure 1.6 2011-2013 Design Values for 1-Hour NO_2 Concentrations for the St. Louis CBSA *vs*. Other Major U.S. CBSAs. The asterisk indicates that no valid design values were available for the Chicago CBSA and the highest invalid design value was used in the figure. Invalid design values do not meet U.S. EPA's criteria for data completeness (sometimes for just a single quarter where there are incomplete data), but they are still included in U.S. EPA design value reports.

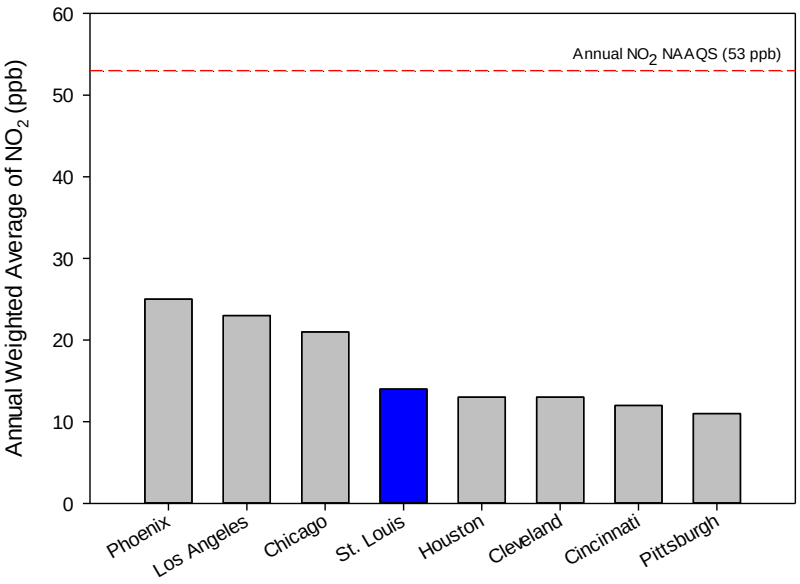


Figure 1.7 2011-2013 Design Values for Annual Average NO_2 Concentrations for the St. Louis CBSA *vs*. Other Major U.S. CBSAs

Figure 1.8 8-Hour Ozone NAAQS Nonattainment Areas (2008 NAAQS). Map from U.S. EPA (2015).

2 Air quality in the St. Louis Area has shown significant improvement in recent years, with air pollutant levels at almost all monitoring locations now in compliance with the health-protective NAAQS

As required by the Clean Air Act to help protect the public health from ambient air pollution, U.S. EPA has developed health-based ambient air quality standards known as the primary NAAQS.[8] According to the U.S. EPA NAAQS website,[9] primary NAAQS are limits that are set "to protect public health, including the health of 'sensitive' populations such as asthmatics, children, and the elderly." U.S. EPA currently has NAAQS for six air pollutants (also known as criteria air pollutants) due to the geographic scope of their occurrence and their public health significance – particulate matter (PM), sulfur dioxide (SO_2), nitrogen oxides (NO_x), ozone (O_3), lead (Pb), and carbon monoxide (CO). The primary NAAQS are intended to be protective of the public health of exposed populations, including sensitive subpopulations (*e.g.*, asthmatics, children, and the elderly), with an adequate margin of safety.

The Clean Air Act requires that U.S. EPA periodically review and, if appropriate, revise existing criteria and NAAQS every five years to ensure that they are current and based on the latest scientific knowledge. Most primary NAAQS have been revised in recent years, with movement towards increasingly lower and more stringent standards. For example, on December 14, 2012, U.S. EPA completed its last review of the PM NAAQS, issuing a final rule that lowered the $PM_{2.5}$ annual NAAQS from 15 $\mu g/m^3$ to a level of 12 $\mu g/m^3$. At this time, U.S. EPA retained the 24-hour $PM_{2.5}$ standard of 35 $\mu g/m^3$ without any change. U.S. EPA last made major changes to the NO_2 and SO_2 NAAQS in June 2010, promulgating new primary 1-hour NAAQS of 100 ppb and 75 ppb, respectively. Most recently, on November 25, 2014, U.S. EPA announced its proposed changes to the O_3 NAAQS, which include revising the current (primary and secondary) 8-hour O_3 standard of 75 ppb down to the range of 65 to 70 ppb. U.S. EPA is scheduled to issue a final decision on its O_3 standard by October 1, 2015.

Figures 2.1 through 2.5 show trends in $PM_{2.5}$, O_3, SO_2, and NO_2 levels measured since 2000 for a subset of the central-site ambient monitors in the St. Louis area. Data were obtained from the U.S. EPA AirData website.[10] Specifically, these figures provide data for two different sets of ambient monitors: (1) five monitors in or near the downtown St. Louis City area that are more centrally located in heavily-populated, urban neighborhoods, and (2) three additional monitors that are generally located in more suburban or rural areas. In the figures, the "urban" monitors are denoted with a solid line and circular icon, while the "suburban/rural" monitors are denoted with a dashed line and triangular icon. Whether considered to be more urban or suburban/rural, each of the monitors in these figures was selected specifically because they provide a long, useful record of data for the criteria air pollutants of interest (see Table 2.1).

[8] Note that U.S. EPA has also developed secondary NAAQS for each of the criteria air pollutants that are intended to protect public welfare, addressing public welfare effects that include decreased visibility and damage to animals, crops, vegetation, and buildings.
[9] http://www.epa.gov/ttn/naaqs/.
[10] http://www.epa.gov/airdata/.

Figures 2.1 through 2.5 provide evidence of significant improvements to air quality in the St. Louis area. Downward trends in ambient concentrations are most apparent for $PM_{2.5}$ and SO_2, although modest declines are also present for O_3 and NO_2. For these monitoring locations, levels of the four criteria air pollutants have decreased over time to the point where the most recent complete year of data (2013) shows levels that are all below the current NAAQS. As indicated by the upper-bound monitoring data for the St. Louis CBSA provided in Section 1, there remain monitoring locations in the St. Louis area that have three-year (2011-2013) design values above the NAAQS for O_3 (8-hour concentrations. However, at many monitoring locations including those in population centers in the urban St. Louis area, levels of the other criteria air pollutants have significantly decreased in recent years to concentrations in compliance with the health-protective NAAQS.

As indicated earlier, the "urban" monitors in the figures are denoted with a solid line and circular icon, while the "suburban/rural" monitors are denoted with a dashed line and triangular icon. Although the "suburban/rural" monitors generally have lower pollutant levels than the "urban" monitors, Figures 2.1 through 2.5 show similar time trends for the two types of monitors, especially for the cases of $PM_{2.5}$ and O_3. These similar time trends suggest that the sources of these pollutants may be regional as opposed to local. While many of the "urban" monitors frequently have higher pollutant levels than the "suburban/rural" monitors (*e.g.*, NO_2 in Figure 2.5), this is likely due in large part to greater traffic-related air quality impacts as well as the greater density of other local air pollution sources.

Table 2.1 Selected $PM_{2.5}$, O_3, SO_2, and NO_2 Monitors in the St. Louis Area

Pollutant	AQS Site ID	State Name	County Name	City Name	Local Site Name	Available Years
$PM_{2.5}$	29-510-0085	Missouri	Saint Louis	Saint Louis	Blair Street	1999-Present
	17-163-0010; 17-163-9010	Illinois	Saint Clair	NA	IEPA - RAPS Trailer aka St. Louis - Midwest Supersite	1999-Present
	29-189-3001	Missouri	Saint Louis	Ladue	Ladue	2009-Present
	29-510-0086	Missouri	Saint Louis	Saint Louis	Margaretta Category B Core Slam PM2.5	1999-2007
	29-510-0007	Missouri	Saint Louis	Saint Louis	South Broadway	2000-Present
	29-186-0006	Missouri	Sainte Genevieve	Sainte Genevieve	Ste Genevieve - PM2.5 Core Slams	1999-2009

Pollutant	AQS Site ID	State Name	County Name	City Name	Local Site Name	Available Years
O_3	29-510-0085	Missouri	Saint Louis	Saint Louis	Blair Street	2005-Present
	29-186-0005	Missouri	Sainte Genevieve	NA	Bonne Terre	1996-2013
	17-163-0010; 17-163-9010	Illinois	Saint Clair	NA	IEPA- RAPS Trailer aka St. Louis - Midwest Supersite	1990-Present
	29-189-3001	Missouri	Saint Louis	Ladue	Ladue	1990-2004
	29-510-0086	Missouri	Saint Louis	Saint Louis	Margaretta Category B Core Slam PM2.5	2000-2008
	29-189-0014	Missouri	Saint Louis	Maryland Heights	Maryland Heights	2005-2013
	29-510-0007	Missouri	Saint Louis	Saint Louis	South Broadway	1990-2003
SO_2	29-510-0085	Missouri	Saint Louis	Saint Louis	Blair Street	2010-Present
	17-163-0010; 17-163-9010	Illinois	Saint Clair	NA	IEPA- RAPS Trailer aka St. Louis - Midwest Supersite	1990-Present
	29-189-3001	Missouri	Saint Louis	Ladue	Ladue	1990-2010
	29-510-0086	Missouri	Saint Louis	Saint Louis	Margaretta Category B Core Slam PM2.5	2000-Present
	29-189-0014	Missouri	Saint Louis	Maryland Heights	Maryland Heights	2005-2010
	29-510-0007	Missouri	Saint Louis	Saint Louis	South Broadway	1990-2010
NO_2	29-510-0085	Missouri	Saint Louis	Saint Louis	Blair Street	2013-Present
	29-186-0005	Missouri	Sainte Genevieve	NA	Bonne Terre	1996-2010
	17-163-0010; 17-163-9010	Illinois	Saint Clair	NA	IEPA- RAPS Trailer aka St. Louis - Midwest Supersite	1990-Present
	29-189-3001	Missouri	Saint Louis	Ladue	Ladue	1990-2010
	29-510-0086	Missouri	Saint Louis	Saint Louis	Margaretta Category B Core Slam PM2.5	2000-Present
	29-189-0014	Missouri	Saint Louis	Maryland Heights	Maryland Heights	2005-2010

Note:
Source: U.S. EPA AirData website.

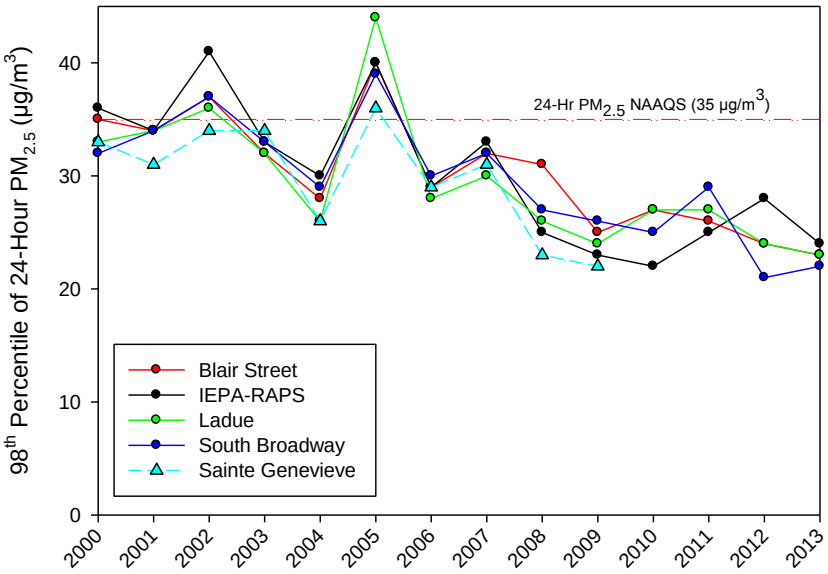


Figure 2.1 2000-2013 Time Series of 98th Percentile 24-Hour $PM_{2.5}$ Concentrations for One "Suburban/Rural" and Four "Urban" Monitors Representative of the St. Louis CBSA

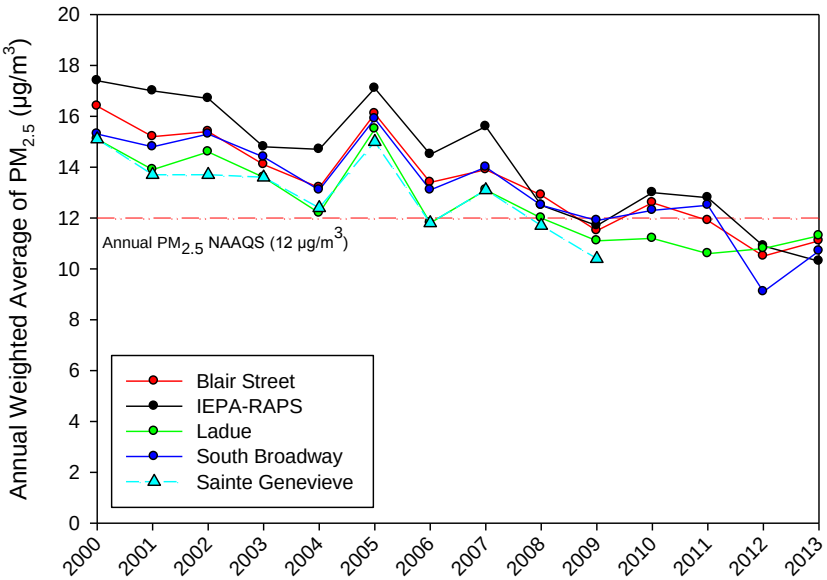


Figure 2.2 2000-2013 Time Series of Annual Average $PM_{2.5}$ Concentrations for One "Suburban/Rural" and Four "Urban" Monitors Representative of the St. Louis CBSA

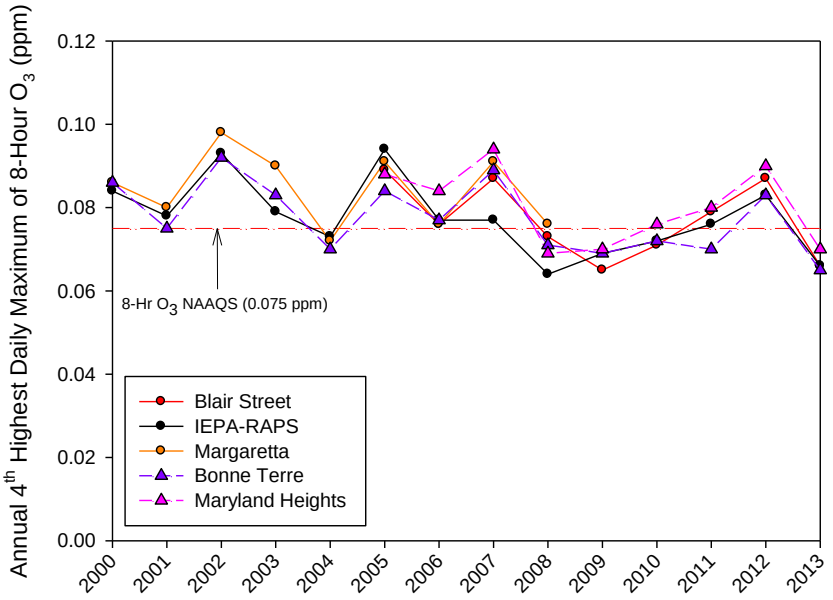


Figure 2.3 2000-2013 Time Series of Annual 4th Highest Daily Maximum O_3 Concentrations for Two "Suburban/Rural" and Three "Urban" Monitors Representative of the St. Louis CBSA

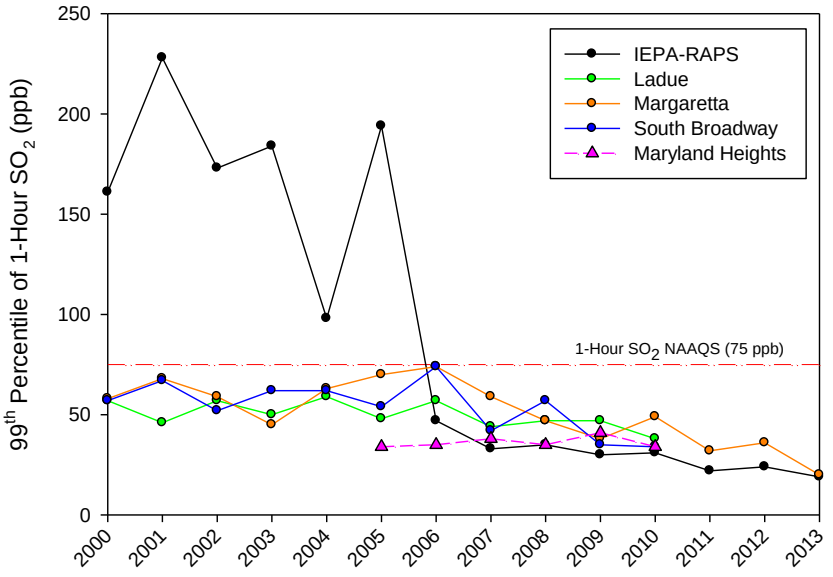


Figure 2.4 2000-2013 Time Series of 98th Percentile 1-Hour SO_2 Concentrations for One "Suburban/Rural" and Four "Urban" Monitors Representative of the St. Louis CBSA

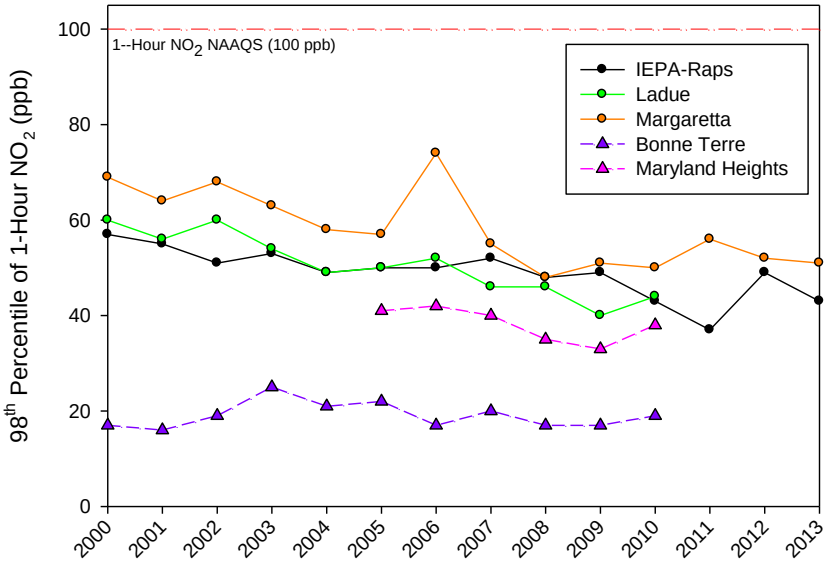


Figure 2.5 2000-2013 Time Series of 98th Percentile 1-Hour NO_2 Concentrations for Two "Suburban/Rural" and Three "Urban" Monitors Representative of the St. Louis CBSA

3 Expected incremental ambient $PM_{2.5}$ air quality impacts due to air emissions from the four Ameren CFPPs are small compared to everyday ambient $PM_{2.5}$ exposure levels in the St. Louis Area

Fine particulate matter, also known as $PM_{2.5}$, is ubiquitous in ambient (outdoor) air due to a large number of common natural and anthropogenic sources that include windblown dust, volcanoes, forest fires, bioaerosols, vehicle exhaust, tire wear particles, road debris, and power plant and other industrial emissions. Because coal-fired power plants such as the Ameren CFPPs are but one of many common sources of ambient $PM_{2.5}$ in urban areas such as St. Louis, it is useful to understand how the incremental ambient $PM_{2.5}$ impacts due to air emissions from the four Ameren CFPPs compare to everyday ambient $PM_{2.5}$ exposure levels in the St. Louis area. Air modeling is a commonly used tool for quantifying the incremental ambient $PM_{2.5}$ impacts from specific sources, such as coal-fired power plants. While $PM_{2.5}$ air modeling studies are typically available for newer power plants due to their preparation as part of today's air permitting process, it is our understanding that $PM_{2.5}$ air modeling studies have not been conducted for the four St. Louis-area CFPPs because they were not necessary for demonstrating attainment with the $PM_{2.5}$ NAAQS.

Given the lack of $PM_{2.5}$ air modeling data to predict incremental ambient $PM_{2.5}$ impacts of the four Ameren CFPPs, we explored whether the numerous published $PM_{2.5}$ source apportionment studies that have been conducted in the St. Louis area (*e.g.*, Amato and Hopke, 2012; Wang *et al*., 2009; Lee and Hopke, 2006; U.S. EPA, 2003) could provide information specific to the $PM_{2.5}$ impacts of the Ameren CFPPs. However, while some of these studies reported findings indicating $PM_{2.5}$ source contributions due to coal combustion (*e.g.*, Wang *et al*., 2009; Lee and Hopke, 2006; U.S. EPA, 2003), they could not distinguish $PM_{2.5}$ mass contributions of specific CFPPs.

We also sought to identify air modeling studies conducted for other U.S. coal-fired power plants that could be used to estimate the approximate incremental $PM_{2.5}$ impacts of the four Ameren CFPPs. Although relatively few air modeling studies of $PM_{2.5}$ impacts have been published for U.S. CFPPs, we identified the Levy *et al*. (2002a) air modeling study of the $PM_{2.5}$ impacts of nine older Chicago-area power plants as providing modeling results of relevance to the $PM_{2.5}$ impacts of the four Ameren St. Louis-area CFPPs. Levy *et al*. (2002a) used the U.S. EPA regulatory air dispersion model CALPUFF to predict the $PM_{2.5}$ impacts of both primary $PM_{2.5}$ emissions and atmospheric secondary formation of sulfate and nitrate particles from SO_2 and NO_x emissions, respectively. There is certainly some uncertainty in using air modeling results for Chicago-area CFPPs to represent the $PM_{2.5}$ impacts of the four Ameren St. Louis-area CFPPs, but as shown in Table 3.1, it is reasonable to assume that the Levy *et al*. (2002a) modeling results likely reflect overestimates of the $PM_{2.5}$ impacts of the four Ameren St. Louis-area CFPPs given that the nine Chicago-area CFPPs included in the Levy *et al*. modeling study total higher amounts of primary $PM_{2.5}$, SO_2, and NO_x emissions. In particular, the combined as-modeled emissions for the nine Chicago-area CFPPs in the Levy *et al*. study were more than 1.5, 2, and 4 times higher than the combined 2013 emissions of primary $PM_{2.5}$, SO_2, and NO_x, respectively, for the four Ameren CFPPs. Moreover, similarities in other factors that influence ground-level air pollutant concentrations from sources such as tall power plant stacks, including meteorological conditions and

terrain, provide support for the use of the Chicago-area CFPPs to represent the local $PM_{2.5}$ impacts of the four Ameren St. Louis-area CFPPs. While the Chicago-area CFPPs are somewhat more geographically dispersed than the St. Louis-area CFPPs, other factors including the taller stack heights and lesser emissions of the St. Louis-area CFPPs are expected to result in the Levy *et al*. (2002a) $PM_{2.5}$ impacts being overestimates of the local $PM_{2.5}$ impacts of the four Ameren St. Louis-area CFPPs.

As discussed in Levy *et al*. (2002a), they predicted maximum annual average impacts from all nine plants of 0.3, 0.2, and 0.2 $\mu g/m^3$ for primary $PM_{2.5}$, secondary sulfates, and secondary nitrates, respectively, in their modeling domain that encompassed much of the Midwest. If to be conservative, these maximum impacts are totaled (which they should not be because they all occurred at different locations, although all in the Chicago-Peoria region where many of the CFPPs were clustered), a maximum annual average $PM_{2.5}$ air quality impact of approximately 0.7 $\mu g/m^3$ is obtained. Table 3.2 shows that the Levy *et al*. (2002a) maximum model-predicted $PM_{2.5}$ concentrations for the nine Chicago-area CFPPs are similar to maximum model-predicted $PM_{2.5}$ concentrations (reflecting primary $PM_{2.5}$ concentrations as well as concentrations of secondary sulfate and nitrate $PM_{2.5}$) that these same investigators reported for a group of seven Atlanta-area CFPPs with a significantly greater combined capacity (>13,000 MW *versus* >7,500 MW). Albeit a county-average concentration rather than a maximum concentration, Perkins *et al*. (2009) reported a smaller model-predicted $PM_{2.5}$ concentration of 0.16 $\mu g/m^3$ for both primary $PM_{2.5}$ emissions and secondarily formed $PM_{2.5}$ from stack emissions of 3 coal-fired power plants and 18 gas-fired power plants in the San Antonio (Texas) metropolitan area.

If a $PM_{2.5}$ concentration of 0.7 $\mu g/m^3$ is conservatively assumed to represent the incremental $PM_{2.5}$ impacts of the four Ameren St. Louis-area CFPPs, it can be compared to total, all-source $PM_{2.5}$ concentrations measured at St. Louis-area monitors to provide perspectives on the incremental $PM_{2.5}$ impacts of the CFPPs relative to background ambient $PM_{2.5}$ from other local and regional sources. In 2013, annual average $PM_{2.5}$ concentrations for air quality monitors within the St. Louis CBSA ranged from 9.6 $\mu g/m^3$ (for the Forest Park monitor at 96 Forest Park in St. Louis) to 11.3 $\mu g/m^3$ (for both the Ladue monitor at 73 Hunter Avenue in Ladue, MO; and for the Branch St. monitor at 100 Branch Street in St. Louis).[11] As shown in Figure 3.1 below, 0.7 $\mu g/m^3$ thus corresponds to 6.2% to 7.2% of the $PM_{2.5}$ levels measured at St. Louis-area $PM_{2.5}$ monitors in 2013, suggesting that *greater than 90% of the airborne ambient $PM_{2.5}$ in the St. Louis-area is from other sources* (*e.g.*, vehicle exhaust, local industrial and commercial sources including smelters and steel mills, windblown dust, and regionally transported $PM_{2.5}$, such as from Ohio River Valley power plants and Midwestern agricultural farming). In summary, while the Levy *et al*. (2002a) maximum model-predicted $PM_{2.5}$ concentrations for the nine Chicago-area CFPPs are only highly approximate estimates and very likely overestimates of the incremental $PM_{2.5}$ impacts of the four Ameren St. Louis-area CFPPs, it would appear reasonable to conclude that *air emissions from the Ameren CFPPs are only relatively minor contributors to airborne ambient $PM_{2.5}$ levels in the St. Louis area*. In other words, it is expected that ambient $PM_{2.5}$ levels at St. Louis-area air quality monitors would not be significantly different if the four Ameren CFPPs were not in existence.

[11] 2013 $PM_{2.5}$ data from Monitor Values Report obtained from the U.S. EPA AirData website (http://www.epa.gov/airdata/ad_rep_mon.html) for the St. Louis CBSA.

Table 3.1 Comparison of Plant Specifications and Emissions for the Four Ameren St. Louis-area CFPPs *versus* the Nine Chicago-area CFPPs Included in the Levy *et al*. (2002a) Modeling Study

Power Plants		Total Capacity (MW)	Heat Input (Million BTU)	Primary $PM_{2.5}$ Emissions (tons)		SO_2 Emissions (tons)	NOx Emissions (tons)
				Filterable	Condensable		
Ameren St. Louis-area Power Plants[1,2]	Labadie	2407	159,859,424	1,838	879	38,384	7,474
	Meramec	839	27,881,128	105	78	5,962	2,088
	Rush Island	1204	74,341,112	818	256	19,587	3,067
	Sioux	986	49,307,431	64	160	2,799	6,004
	Total	5436	311,389,094	2,825	1,374	66,733	18,633
Levy *et al.* (2002a) Modeled Chicago-area Power Plants[3]	Crawford	597.6	26,569,896	160	132	8,468	4,454
	E.D. Edwards	780.3	38,313,345	66	2,862	51,601	9,082
	Fisk	374.1	18,901,367	108	94	5,269	3,490
	Hennepin	306.3	19,210,906	201	97	6,820	3,833
	Joliet 29	1320	57,434,963	361	291	17,379	9,092
	Joliet 9	360.4	15,430,328	149	76	4,739	5,543
	Powerton	1785.6	70,942,171	434	354	19,423	26,898
	Waukegan	802.7	48,501,637	402	291	18,327	5,984
	Will County	1268.9	56,498,731	222	222	19,180	11,243
	Total	7596	351,803,344	2,102	4,419	151,205	79,620

Notes:

(1) With the exception of the primary $PM_{2.5}$ emissions data, 2013 data for the four Ameren St. Louis-area power plants were obtained from the U.S. EPA Clean Air Markets Database (CAMD).

(2) Primary $PM_{2.5}$ emissions data were obtained from Ameren and are mainly for the 2010-2011 timeframe. They are considered to be generally representative of primary $PM_{2.5}$ emissions from the four power plants.

(3) All data for the nine Chicago-area power plants were obtained from Levy *et al.* (2002a), with the emissions data representing annual average emissions that were used in the air modeling analysis. As described in Levy *et al.* (2002a), the plant capacity and heat input data are for 1998, while the emissions data are estimates of expected annual emissions for 2000.

Table 3.2 Published Model-predicted Ground-level $PM_{2.5}$ Concentrations for Groups of U.S. CFPPs

Source	Number of Modeled CFPPs	Plant Location(s)	Plant Capacity	Model-predicted Ground-level Annual Average $PM_{2.5}$ Air Conc. ($\mu g/m^3$)[1]
Levy *et al.* (2002a)	9	Illinois, in close proximity to or upwind of the Chicago area	>7,500 MW total nameplate capacity	0.7, including both primary and secondary $PM_{2.5}$
Levy *et al.* (2003)	7	Georgia, in the Atlanta area	>13,000 MW total nameplate capacity	0.6-0.9 depending on the air modeling approach, including both primary and secondary $PM_{2.5}$
Perkins *et al.* (2009)	3 coal plants/units (and 18 gas plants/units)	Bexar County, Texas, in the San Antonio metropolitan area	1,425 MW capacity for 3 coal plants/units (and ~2,300 MW capacity for 18 gas plants/units)	0.16 for year 2002 emissions, including both primary and secondary $PM_{2.5}$

Notes:
MW = megawatt; NA = not available, $PM_{2.5}$ = fine particulate matter (up to 2.5 µm in diameter).
(1) The Levy *et al.* (2002a, 2003) $PM_{2.5}$ results are for maximum impacted model receptor locations, while the Perkins *et al.* (2009) $PM_{2.5}$ results are county-average impacts.

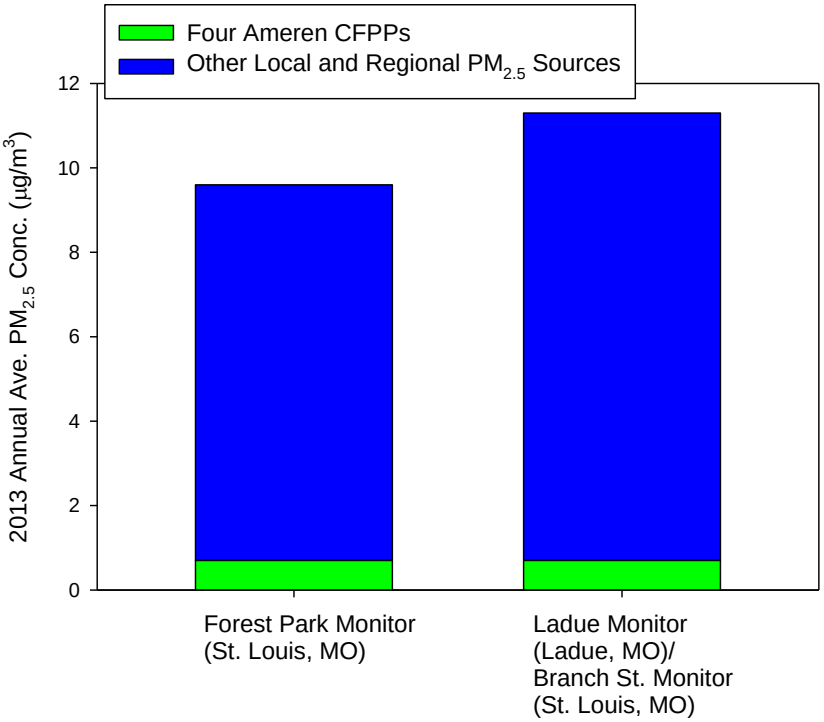


Figure 3.1 Comparison of the Estimated Incremental Annual Average $PM_{2.5}$ Impact of the Four Ameren St. Louis-area CFPPs *vs*. Total All-Source Measured $PM_{2.5}$ Levels at St. Louis-area Air Quality Monitors

4 Total personal exposures to air pollutants, including $PM_{2.5}$, NO_x, and ozone, will generally be dominated by contributions from other common indoor and outdoor sources rather than by contributions from CFPPs

The various air pollutants associated with CFPP stack emissions, including those that are directly emitted by CFPPs (*e.g.*, primary $PM_{2.5}$, NO_x, SO_2, CO) as well as those for which CFPP stack emissions contribute to secondary formation in the atmosphere (*e.g.*, O_3, sulfate and nitrate $PM_{2.5}$), are all generated (either directly or indirectly) by other outdoor combustion sources that include on-road and non-road mobile sources, industrial processes, fires, volcanoes, geothermal hot springs, lightning, and natural gas- and oil-fired boilers. In addition, breathing indoor air is well recognized to be a major source of our everyday exposures to a variety of air pollutants, including criteria air pollutants such as $PM_{2.5}$, NO_x, and O_3 (Spengler *et al*., 2001). This is both because we spend the majority of our time indoors,[12] and because there are a number of common indoor sources of air pollutants. As discussed below, both $PM_{2.5}$ and NO_x are produced indoors by common combustion appliances and sources (gas ranges, wood stoves, kerosene heaters, fireplaces, candles and tobacco smoke). There are fewer indoor O_3 sources, although O_3 is emitted as an intentional or unintentional by-product by some consumer products (*e.g.*, ionizing air cleaners, electrostatic precipitators, and office printing / copy equipment). O_3 can also be formed indoors *via* chemical reactions. Overall, it is well-documented that indoor concentrations of numerous air pollutants are often greater than outdoor concentrations, in part because pollutants from indoor sources can build up and remain confined within indoor spaces over extended periods of time (Long *et al*., 2000).

With a focus on $PM_{2.5}$, NO_x, and O_3, we provide below an overview of common exposure sources in order to illustrate that total personal exposures to these criteria air pollutants will generally be dominated by contributions from common indoor and outdoor sources other than by contributions from local CFPPs. Moreover, as discussed below, indoor levels of pollutants of ambient origin (*i.e.*, $PM_{2.5}$ and NO_x emitted by power plants, O_3 formed in power plant plumes) are often significantly reduced compared to outdoor levels due to infiltration and deposition losses during transport indoors, where most human exposure occurs. As a result, CFPP-related emissions are dispersed and concentrations significantly reduced by the time they reach ground-level and indoor environments where human exposure can occur; in contrast, other ground-level ambient sources (*e.g.*, mobile sources) and indoor sources can have a significantly higher exposure potential given that their emissions typically occur closer or in direct proximity to people breathing the air.

[12] U.S. EPA (2009) states that people spend roughly 90% of their time indoors.

4.1 $PM_{2.5}$

$PM_{2.5}$ is always present in both outdoor and indoor air due to a multitude of common sources. As discussed earlier, outdoor $PM_{2.5}$ has a diverse number of natural and human sources, including windblown dust, volcanoes, forest fires, bioaerosols, vehicle exhaust, tire wear particles, road debris, power plants, and commercial and industrial emissions. Indoors, we have routine exposures to $PM_{2.5}$ from a variety of ordinary personal and indoor sources. Each of us generates airborne $PM_{2.5}$ everyday through typical daily activities, such as cooking (baking, frying, grilling, barbecuing, toasting, *etc.*), dusting, vacuuming, folding clothes, making beds, mowing the lawn, driving a car, heating a home, smoking, burning candles, *etc.* Indoor $PM_{2.5}$ sources can be a large contributor to total personal $PM_{2.5}$ exposures, with U.S. EPA studies showing that cooking events can increase average $PM_{2.5}$ concentrations throughout the home by about 50 $\mu g/m^3$ (Wallace *et al*., 2004). Table 4.1 illustrates how common indoor $PM_{2.5}$ sources, such as cooking and cleaning activities, are capable of elevating short-term indoor $PM_{2.5}$ levels by tens to hundreds of $\mu g/m^3$.

Figures 4.1 and 4.2, which are adapted from the Levy *et al*. (2002b) and Long *et al*. (2000) studies, respectively, illustrate how we are all routinely exposed to elevated $PM_{2.5}$ levels in a number of everyday indoor and outdoor microenvironments, such as buses, subway cars and station platforms, food courts, restaurants, and our homes. For comparison, these figures also include the Section 3 estimates of the incremental annual average $PM_{2.5}$ impacts of the four Ameren CFPPs in the St. Louis-area. As indicated in these figures, $PM_{2.5}$ exposures from common indoor locales are expected to far exceed any $PM_{2.5}$ exposures associated with the four Ameren CFPPs.[13]

Table 4.1 Average Airborne Particle Levels During Typical Indoor Activities Compared to the Estimated Incremental Annual Average $PM_{2.5}$ Concentration from St. Louis-area Power Plants

Activity	Indoor Activity $PM_{2.5}$ Concentration ($\mu g/m^3$)	Section 3 Estimated Incremental Annual Average $PM_{2.5}$ Concentration from St. Louis-area Power Plants ($\mu g/m^3$)
Baking (gas)	101	**0.7**
Sautéing	66	**0.7**
Toasting	54	**0.7**
Frying	41	**0.7**
Stir-frying	37	**0.7**
Broiling	29	**0.7**
Burning candles	28	**0.7**
Dusting	23	**0.7**
Baking (electric)	15	**0.7**
Walking vigorously over carpet indoors	12	**0.7**
Cleaning with Pine Sol	11	**0.7**
Vacuuming	7	**0.7**

Note:
Indoor activity $PM_{2.5}$ data from Long *et al*. (2000).

[13] Although for different averaging periods, the short-term air exposure data for common indoor and outdoor microenvironments shown in Figures 4.1 and 4.2 can be compared to the estimated incremental annual average $PM_{2.5}$ impacts for the four Ameren St. Louis-area CFPPs given that these types of short-term exposures occur on a frequent, if not daily, basis.

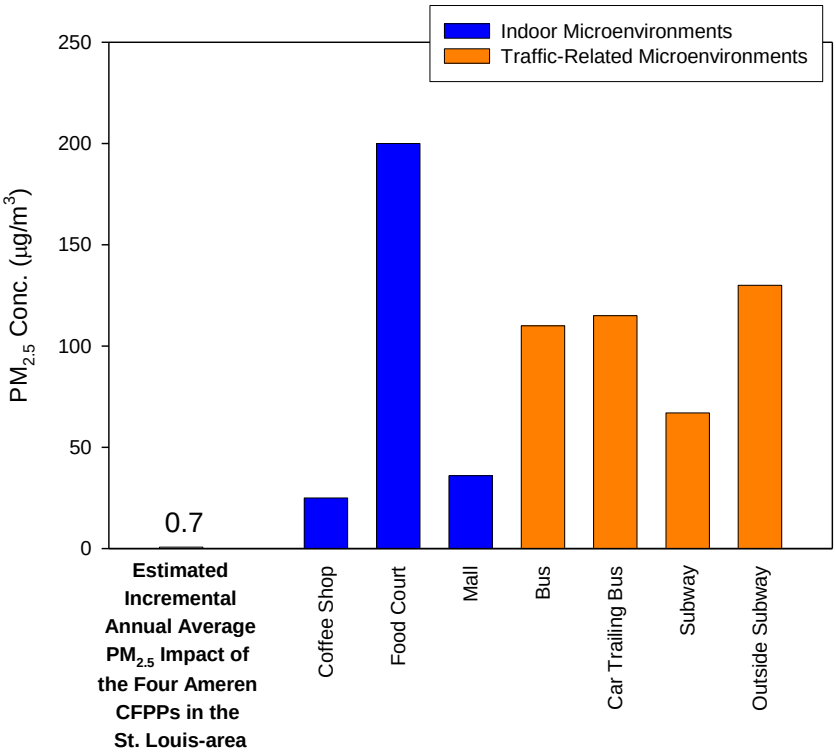


Figure 4.1 Mean Measurements of $PM_{2.5}$ Levels ($\mu g/m^3$) in Various Everyday Locations in Comparison to the Estimated Incremental Annual Average $PM_{2.5}$ Impact of the Four Ameren CFPPs in the St. Louis Area. $PM_{2.5}$ data for everyday locations from Levy *et al.* (2002b).

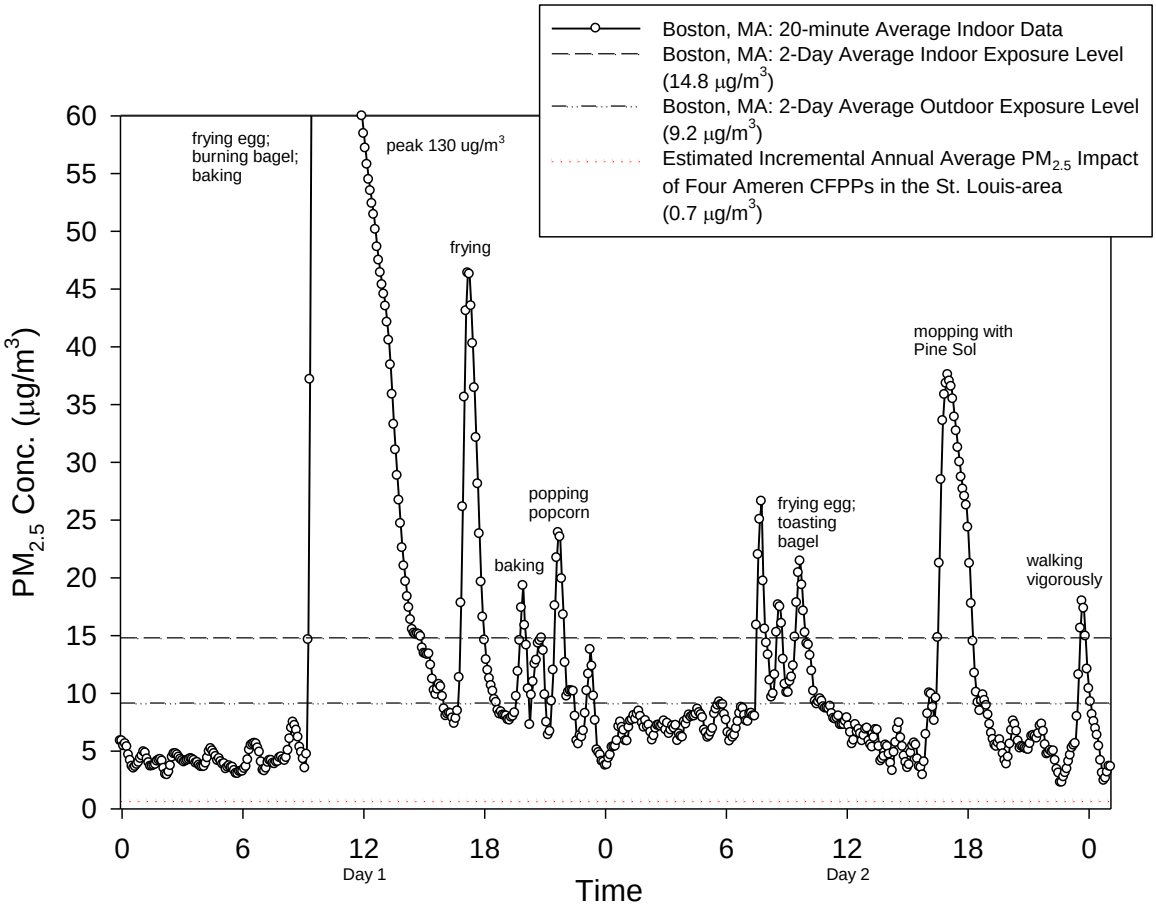


Figure 4.2 Estimated Incremental Annual Average $PM_{2.5}$ Impact of the Four Ameren CFPPs in the St. Louis Area (red dotted line) *vs*. Everyday Indoor and Outdoor $PM_{2.5}$ Exposure Levels in a Boston-area Residential Home (black solid and dashed lines, respectively). Boston indoor and outdoor $PM_{2.5}$ data from Long *et al*. (2000).

Personal outdoor activities can also be significant sources of $PM_{2.5}$ exposures. For examples, as part of the U.S. EPA Small Engine Exposure Study (SEES), Baldauf *et al.* (2006) conducted a series of measurements for $PM_{2.5}$ and other airborne contaminants (carbon monoxide [CO], aldehydes, VOCs) during personal outdoor activities such as use of gasoline-powered riding tractors, walk-behind lawn mowers, string trimmers, and chainsaws. Personal $PM_{2.5}$ concentrations measured during these test events ranged from 63 up to 3,809 $\mu g/m^3$. In addition, Quintana *et al*. (2001) reported that barbecuing was associated with an average increase in personal $PM_{2.5}$ exposure of 120 $\mu g/m^3$ over a 99-minute event period, while yard work was associated with an average increase in personal $PM_{2.5}$ exposure of 72 $\mu g/m^3$ over a 177-minute event period.

Moreover, it bears mentioning that indoor concentrations of $PM_{2.5}$ of ambient origin are often substantially reduced compared to outdoor $PM_{2.5}$ concentrations due to particle losses that occur during transport across the building shell and from indoor deposition (Diapouli *et al*., 2013; Clark *et al*., 2010; Sarnat *et al*., 2006; Wallace and Williams, 2005). In other words, exposures to $PM_{2.5}$ of ambient origin, such as $PM_{2.5}$ associated with CFPP emissions, are generally reduced for people indoors as compared to outdoors. Research has demonstrated that particle losses due to transport across the building shell and from indoor deposition are dependent on building characteristics (*e.g.*, ventilation conditions, age of construction, building size, type of heating and cooling system). Sarnat *et al.* (2006) estimated an average

$PM_{2.5}$ infiltration factor of 0.48 (*i.e.*, 48% of ambient airborne $PM_{2.5}$ penetrated into indoor spaces and remained airborne indoors) based on a study of 17 homes in Los Angeles, CA; similarly, Clark *et al.* (2010) estimated an average $PM_{2.5}$ infiltration factor of 0.52 for their study of Toronto homes. Studies such as Wallace and Williams (2005), Allen *et al*. (2003), and Long *et al*. (2001) have reported evidence that the greatest reductions in ambient particle infiltration typically occur for homes under low ventilation conditions – *i.e.*, tightly-sealed homes with closed windows and doors, such as for homes that use central air-conditioning systems in the summertime and for "winterized" homes in colder climates. For example, Allen *et al*. (2003) reported a mean $PM_{2.5}$ infiltration factor of 0.53 for Seattle homes during the heating season as compared to a mean of 0.79 during the non-heating season when windows and doors are typically opened to promote ventilation.

4.2 NO_x

As discussed in HEI (2010) and U.S. EPA (2008, 2013a), engine exhaust fumes from both on-road and non-road vehicles are the largest sources of NO_x emissions nationwide; for example, based on data from the 2008 National Emission Inventory, U.S. EPA (2013a) reported that highway and off-highway vehicles contributed 39% and 19%, respectively, of total NO_x emissions, as compared to 17% for fuel combustion by utilities. In urban areas, highway vehicles are dominant contributors of ambient NO_2 concentrations, such that NO_2 has been proposed as a surrogate for traffic emissions. Figure 4.3, which is based on data and a similar figure in HEI (2010), illustrates the significance of traffic emissions as sources of ambient NO_2 concentrations, showing that higher average NO_2 concentrations have generally been measured inside vehicles and at roadside locations as compared to other monitoring locations. U.S. EPA (2008) summarized findings from a body of studies suggesting that traffic-related NO_2 exposures, both from commuting activities and from living near heavily-trafficked roads, can dominate total personal NO_2 exposures.

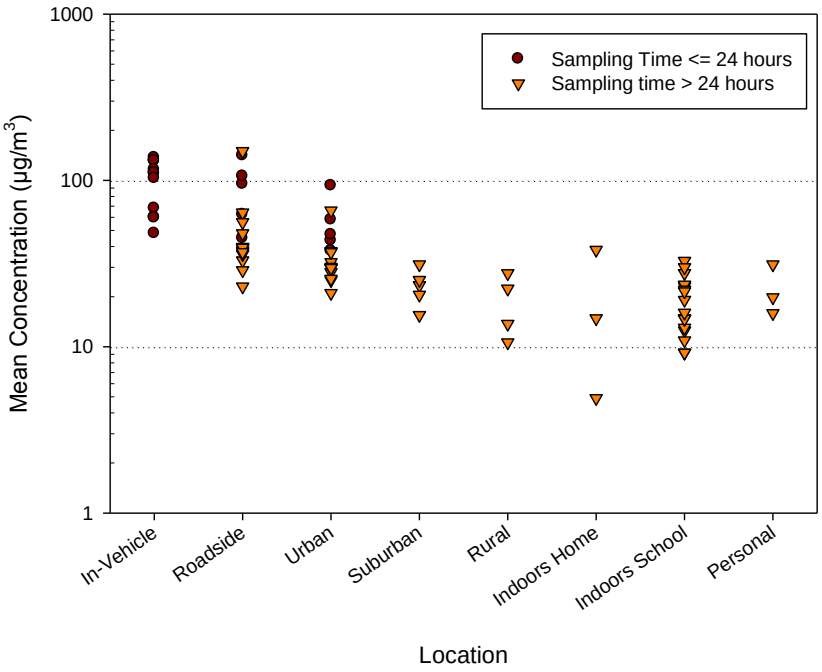


Figure 4.3 Mean NO_2 Concentrations Measured in Various Exposure Microenvironments. Figure adapted from HEI (2010). As discussed in this report, data are primarily from traffic-pollution studies published since 1998.

NO_x can also have important indoor sources, such that indoor NO_2 concentrations can be significantly higher than outdoor NO_2 concentrations. Similar to $PM_{2.5}$, the relative impacts of indoor NO_x sources *versus* ambient NO_x are affected by losses of ambient NO_x during transport indoors; as discussed in HEI (2010), study findings suggest that, in the absence of indoor sources, indoor NO_2 concentrations are approximately 50% of outdoor NO_2 concentrations. NO_x is emitted indoors by a variety of common combustion sources, including indoor combustion sources, such as gas cooking appliances (*e.g.*, natural gas cooking burners), oil furnaces, kerosene space heaters, wood-burning and natural gas fireplaces, wood stoves, candle burning, and smoking. For homes with attached garages and other structures, motor vehicles and generators can be sources of indoor NO_x.

As discussed in U.S. EPA (2008), a number of studies have demonstrated the significance of gas cooking appliances as sources of indoor NO_x exposure, with findings showing that indoor NO_2 concentrations are approximately 50 to 400% higher in homes with gas-cooking appliances than homes with electric cooking appliances. ARCADIS (2001) measured indoor NO_2 concentrations associated with simulated cooking and oven-cleaning activities using gas appliances in a small test house, reporting that average concentrations during the cooking periods ranged from 24-216 µg/m^3 (13-115 ppb) and that average NO_2 concentrations exceeded 750 µg/m^3 (400 ppb) in three rooms (kitchen, adjacent living room, bedroom) of the test house during five-hour oven self-cleaning tests. More recently, using a simulation model designed to predict time-dependent pollutant concentrations associated with residential natural gas cooking burners in Southern California homes, Logue *et al*. (2014) estimated average contributions of 25 to 39% for natural gas cooking burners to total weekly average NO_2 concentrations inside modeled homes across the four seasons. Other studies have characterized the impacts of unvented or inadequately vented gas appliances, such as gas hot water heaters and gas fireplaces, on indoor NO_2 concentrations (U.S. EPA, 2008).

4.3 O_3

Ozone exposure derives from a multitude of everyday sources, including some common indoor sources. Outdoors, ozone is rarely emitted directly; *i.e.*, ozone is not emitted from the stacks of coal-fired power plants such as the four Ameren St. Louis-area CFPPs, nor by motor vehicles. Instead, it is formed in the atmosphere *via* the action of sunlight on substances in the air emitted by a number of sources that include vehicle and engine exhaust, industrial facilities, combustion from power plants, gasoline vapors, biogenic sources, and chemical solvents. Most ambient ozone is formed when certain substances react with each other in the presence of sunlight (*e.g.*, volatile organic compounds, or VOCs, reacting with nitrogen oxides, or NO_x). Although power plant plumes can be major sources of NO_x, they are not significant sources of the VOCs that are also needed for ozone formation; as a result, formation of ozone in areas influenced by power plant emissions is dependent on the availability of VOCs from other sources. Importantly, even in the absence of anthropogenic emissions, there would still be measurable ground-level ozone concentrations due to lightning and formation from natural precursors, as well as due to transport of stratospheric ozone down to the troposphere. For example, Emery *et al*. (2012) cited study findings indicating that stratosphere-troposphere exchange can contribute to multi-day ozone enhancements in the range of 50-65 ppb at remote western and northern U.S. sites.

As outside air infiltrates indoors, there are significant reductions in ozone levels compared to outdoor levels, due to the chemical reactivity of ozone (U.S. EPA, 2013b; McKone *et al*., 2009). Specifically, studies have shown that (in the absence of indoor sources) indoor ozone concentrations are typically 10 to 40% of outdoor concentrations (U.S. EPA, 2013b; McKone *et al*., 2009). Ozone penetration is dependent on a number of factors, including season, building ventilation rate, use of air conditioning, and microenvironment. In general, higher ozone penetration is typically observed in homes with open windows and doors, while tighter homes, and in particular those relying on central air conditioning systems, have been found to have lower ozone penetration. Lee *et al*. (1999) reported nearly ten times greater ozone penetration for 17 California homes with windows open and no air conditioner (AC) use compared to three homes with AC turned on. Ozone is also efficiently removed indoors through deposition on surfaces and *via* indoor chemical reactions, further reducing indoor concentrations compared to outdoor concentrations (Weschler, 2000; U.S. EPA, 2013b).

Aside from penetration of outdoor ozone into indoor spaces, O_3 exposure indoors derives from common consumer products. Indoor sources of O_3 include ozone generators (household air purifiers), ion generators and electrostatic air cleaners (precipitators),[14] photocopiers, fax machines, laser printers, computer monitors, and ink/bubble jet printers (Weschler, 2000; U.S. EPA, 2013b; ARB, 2005). ARB (2005) reports that the number of indoor direct-emitting ozone sources has increased within the last 10-15 years. For individuals in indoor environments where these consumer and office products are used, the O_3 emissions can elevate indoor and personal O_3 concentrations well above outdoor concentrations. For example, U.S. EPA-funded studies have measured O_3 emissions from office equipment, showing that emissions increase with improper maintenance (Leovic *et al*., 1996; 1998). Based on a 1994 literature survey of office equipment emissions, U.S. EPA (1995) reported that measurement studies of photocopier machines had observed maximum breathing-level concentrations ranging from <1 to 150 ppb in poorly ventilated rooms. U.S. EPA (1995) also reported indoor ozone concentrations of approximately 50 to 2,000 ppb for laser printer usage. Other tests conducted on four laser printers by Tuomi *et al*. (2000) yielded air concentrations of up to 180 ppb ozone.

[14] Ionic air cleaners are widely available in a variety of different designs, including larger units for household use; smaller units for use in bathrooms, refrigerators, and closets; units for cars; personal wearable units, or "personal air purifiers"; ionic brushes; shoe cleaners; and toothbrush disinfectors (Britigan *et al*., 2006).

Household ozone generators (marketed as air purifiers) intentionally emit ozone into indoor air with the goal of improving air quality. Also marketed as air purifiers, ion generators emit ozone as a by-product. A number of studies (Shaughnessy and Oatman, 1992; Chessor, 1998; Mason *et al*., 2000; Britigan *et al*., 2006; Waring and Siegel, 2011) have demonstrated that certain conditions cause such air purifiers to elevate indoor ozone concentrations to levels in the range of hundreds to thousands of ppb. For example, for experiments using a human-sized mannequin wearing a personal air purifier, Britigan *et al*. (2006) reported peak O_3 concentrations near the mannequin's mouth of 700 ppb.

5 Hypothetical health impacts calculated for CFPP air emissions are unreliable because of many hidden assumptions, uncertainties, and limitations

5.1 Hypothetical health impact assessments conducted by NGOs

Hypothetical health impact calculations offered by non-governmental organizations (NGOs) like the CATF purport to present calculations that assert health harms from specific local sources, *e.g.*, from fossil-fuel power plants. Two such projections have included, among other U.S. power plants, the four Ameren CFPPs near St. Louis. The Clean Air Task Force calculated the health and economic impacts of all coal-fired power plants, with updated results reflecting their estimated projection of 2012 air emissions (CATF, 2014). A similar analysis of 51 power plants was conducted by the Environmental Integrity Project (EIP, 2012).

The CATF model calculations projected yearly premature deaths, heart attacks, asthma attacks, hospital admissions, cases of chronic bronchitis, and asthma emergency room visits attributable to the four Ameren CFPPs, with total attributed health costs of over $1.3 billion (Table 5.1). The Table 5.1 numbers were obtained from the CATF website, which has an interactive map of the U.S., showing power plants and their associated impacts on health.[15] Similarly, EIP estimated costs from "premature deaths" associated with power plants across the U.S., including three of the Ameren Missouri Plants (Labadie, Meramec, and Rush Island, Table 5.1). Both analyses projected hypothetical health and economic impacts of power plant air emissions, specifically fine particulate matter, or $PM_{2.5}$, and gaseous precursors to $PM_{2.5}$ (SO_x and NO_x). However, as we will explain in our review of the methodology, the results in Table 5.1 are speculative and should not be interpreted as reliable or even realistic predictions of actual health outcomes.

Our critical evaluation of the underlying analyses demonstrates that the health claims reported in these model exercises are hypothetical and, at best, overstated. In this section, we highlight the issues associated with the assumptions, methods, and data inputs in these projections. Specifically, in Section 5.2, we summarize the methods used to derive health impact estimates. In Section 5.3, we discuss limitations of the analyses, and in Section 5.4 we point out that experimental, non-statistical lines of scientific evidence suggest that the projections in Table 5.1 are unsupported by laboratory and clinical data.

[15] http://www.catf.us/fossil/problems/power_plants/existing/.

Table 5.1 Examples of the Translation of $PM_{2.5}$ Concentration Increments into Hypothetical Health Impacts Nationwide and Their Monetary Value

Alleged Health Statistic	Labadie Power Plant				Meramec Power Plant				Rush Island Power Plant				Sioux Power Plant	
	CATF (2014)		EIP (2012)		CATF (2014)		EIP (2012)		CATF (2014)		EIP (2012)		CATF (2014)	
	N	*Cost**	*N*	*Cost**	*N*	*Cost**	*N*	*Cost**	*N*	*Cost**	*N*	*Cost**	*N*	Cost*
Deaths	78	570,000	140-290	1,200,000-2,400,000	32	230,000	57-110	470,000-950,000	40	290,000	66-130	550,000-1,100,000	19	190,000
Heart Attacks	120	13,000	--	--	49	5,400	--	--	61	6,700	--	--	29	4,300
Asthma Attacks	1,300	69	--	--	540	28	--	--	670	35	--	--	320	23
Hospital Admissions	56	1,300	--	--	23	530	--	--	28	660	--	--	14	430
Chronic Bronchitis	48	21,000	--	--	19	8,600	--	--	24	11,000	--	--	12	7,000
Asthma ER Visits	85	31	--	--	35	13	--	--	43	16	--	--	21	10

Notes:
N = model-estimated, hypothetical annual number per year for specific power plants across the entire U.S.
Cost* = model-calculated, attributed monetary value, in thousands of dollars, of the alleged health effects.

Table 5.2 CATF (2014) Overall Hypothetical Health Impacts of All U.S. CFPPs within the St. Louis-area Counties Containing the Ameren CFPPs[1]

Alleged Health Statistic	Franklin Co, MO (Location of Labadie Power Plant)		St. Louis Co, MO (Location of Meramec Power Plant)		Jefferson Co, MO (Location of Rush Island Power Plant)		St. Charles Co, MO (Location of Sioux Power Plant)	
	N	*Cost**	*N*	*Cost**	*N*	*Cost**	*N*	Cost*
Deaths	3	25,000	20	150,000	6	44,000	8	63,000
Heart Attacks	5	580	34	3,700	10	1,100	16	1,800
Asthma Attacks	56	3	310	16	110	6	190	10
Hospital Admissions	2	55	15	350	4	99	7	170
Chronic Bronchitis	2	920	12	5,400	4	1,900	7	3,000
Asthma ER Visits	4	1	22	22	8	3	13	5

Notes:
N = model-estimated, hypothetical annual number per year for all U.S. CFPPs in just the county of interest.
Cost* = model-calculated, attributed monetary value, in thousands of dollars, of the alleged health effects.
(1) No results provided for EIP (2012) because this study did not estimate health impacts specific to the St. Louis-area counties.

5.2 Health impacts modeling by CATF and EIP targets specific sources

CATF (2014) and EIP (2012) calculated hypothetical health impact numbers for power plants across the U.S., focusing on the small increments in ambient $PM_{2.5}$ attributable to the plants' air emissions. The model used requires 1) estimating air emissions from each power plant, 2) applying atmospheric dispersion models to estimate the increment in $PM_{2.5}$ at ground-level caused by air emissions, 3) assuming a causal basis for statistical associations between changing $PM_{2.5}$ levels to changing health statistics (*e.g.*, assuming that eliminating the $PM_{2.5}$ contributions from power plants will result in avoided deaths or hospitalizations), and 4) estimating the economic benefit from eliminating such calculated hospitalizations and deaths (*e.g.*, $/death or $/hospital admissions).

In the first step of the modeling approach, air emissions for directly emitted $PM_{2.5}$ and PM-forming pollutants (sulfur dioxide [SO_2], nitrogen oxides [NO_x], volatile organic compounds [VOCs], and ammonia [NH_3]) are estimated for the modeled years. In the approach taken by CATF, the emission estimates were derived using a 2001 emissions inventory, which provided input to the Integrated Planning Model (IPM) to forecast emissions in 2012. The emissions inventory generally includes a dataset of the major pollutants that can contribute to $PM_{2.5}$, including SO_2, NO_x, NH_3, VOCs, and direct emissions of $PM_{2.5}$. The IPM is a multi-regional, dynamic model developed by ICF International and used by U.S. EPA and various private clients to calculate how environmental policies alter air emissions in the 48 contiguous states and the District of Columbia. Details on the 2001 emissions inventory and IPM analyses that were used in the CATF assessment can be found in the CAIR technical support documentation (U.S. EPA, 2005). In contrast to the approach taken by CATF, EIP obtained emissions estimates of SO_2 and NO_2 from U.S. EPA's "Clean Air Markets" website for the 51 plants that were modeled in their study. Direct emissions of PM were obtained from annual emissions inventories reports that are provided to state agencies every year. Data for EIP's calculations were based on 2011 air emissions for most power plants.

In Step 2, the air emission estimates were used as inputs in an air quality dispersion model, the so-called source-receptor (S-R) matrix, along with transfer coefficients, to estimate ground-level $PM_{2.5}$ concentrations by county. The transfer coefficients, which were derived from 1990 meteorological data, were used to calculate the proportion of direct $PM_{2.5}$ emissions and $PM_{2.5}$ precursor species (*e.g.*, SO_2, NO_x, VOCs) emissions from each source that were transported to a single hypothetical point at the center of each county across the U.S. The gross oversimplifications inherent in this modeling approach are discussed below in Section 5.3.1.

In Step 3, increments in county level $PM_{2.5}$ concentrations resulting from power plant emissions were used to calculate the hypothetical changes in health statistics for the various health outcomes, under the assumption that the statistical correlations are causal and have zero confounding, bias, and measurement error. Specifically, county-by-county population data, baseline health incidence data, the incremental $PM_{2.5}$ data, and a $PM_{2.5}$ health-effect function were used as inputs to the health impact models. The health-effect function is derived from concentration-response associations observed in epidemiology studies that correlate health statistics with increments in $PM_{2.5}$ concentrations. The model calculates the "avoided health effects," or the health effects changes assumed to occur if the modeled power plant emissions were zeroed out (*i.e.*, eliminated).

In Step 4, the model-estimated changes in health statistics are then multiplied by a unit value that assigns a dollar amount to the avoided health impact (*e.g.*, $/hospital visit or $/death).

Two distinguishing aspects of "the model" used by CATF and EIP should be noted. One is that the majority of "impacts" are calculated to occur distant to the power plant location rather in local

communities nearby to power plants. Table 5.2 shows the local county "impacts" from all U.S. power plants including the local Ameren plants, and the numbers are much lower than the "nationwide" impacts in Table 5.1. These findings thus indicate that the majority of the hypothetical health impacts and associated costs predicted by the CATF (2014) and EIP (2012) studies do not occur locally in the St. Louis-area counties and instead predominantly occur in more distant counties and states.

In addition, the "model" calculates impacts for the small increment in ambient $PM_{2.5}$ attributable to coal-fired power plants *per se*, yet ignores the much larger proportion of ambient $PM_{2.5}$ due to all other sources, both local and distant. Sections 3 and 4 discussed people's total $PM_{2.5}$ exposure, and if total personal $PM_{2.5}$ concentrations were entered into the "model," the projected mortality "impacts" would amount to a significant fraction of all mortality from all causes. For example, based on health impact calculations made using U.S. EPA's Benefits Mapping and Analysis Program (BenMAP), Fann *et al*. (2012) reported findings indicating that ambient (outdoor) $PM_{2.5}$ causes 10% of all-cause mortality in Los Angeles County. Given that U.S. EPA researchers have reported data indicating that PM of ambient origin often contributes on the order of 50% or less of total personal $PM_{2.5}$ exposures (Williams, 2005; Burke *et al*., 2001), this suggests that 20% or more of all-cause mortality in Los Angeles County is attributable to $PM_{2.5}$ exposure, which would appear to be implausible.

5.3 Limitations associated with health impact calculations

Health impact "analyses" of this nature are complex and require the use of several diverse models, model inputs, and model assumptions, each of which has numerous uncertainties. That is, many of the model inputs and assumptions do not necessarily reflect actual conditions or real causal links, and therefore the end results cannot expected to be reliable and accurate when viewed in isolation for one scenario or one source type. The sources of uncertainty include the following: inaccurate or incomplete emission inventories; inaccurate air quality model inputs; limited meteorological data; unknown causal basis for the epidemiological input to the health impact functions; debatable monetary valuation of the hypothetical health effects; incomplete population estimates and baseline disease prevalence; inaccurate projection of the future state of the world (*i.e.*, regulations, technology, and human behavior). All of these dramatically reduce the stability and reliability of the model results. Impact analyses do <u>not</u> combine the uncertainties inherent to all the input variables and assumptions that enter into the calculation to derive an overall uncertainty of the final result. Were this error propagation done, the uncertainty interval of the final result can be expected to encompass the possibility of zero health impact (see discussion below).

In addition, as shown in Sections 1 and 2, the areas around St. Louis have $PM_{2.5}$ air quality that is in compliance with the health-protective NAAQS for $PM_{2.5}$. By definition, people in attainment areas are considered to not be at risk of adverse health effects due to ambient air pollution, yet CATF and EIP have calculated hypothetical health effects in this attainment area.

As discussed in more detail below, when compliance with the $PM_{2.5}$ NAAQS and the uncertainties in the modeling approach and its estimated impacts are properly considered, it becomes clear that the health effects calculated by environmental organizations for Ameren power plant emissions are theoretical and may well be zero. In fact, the expected lack of health effects is supported by the levels of $PM_{2.5}$ that have been measured in the vicinity of the Ameren facilities, which are in attainment of the health-protective $PM_{2.5}$ NAAQS (as was discussed in Section 2).

5.3.1 Uncertainties in the emissions and $PM_{2.5}$ concentration estimates

Critical inputs to the health impact modeling are the estimated air emissions. In the CATF analysis, the emission estimates were obtained from a dated emissions inventory (reflecting 2001 emissions) and a model was used to forecast emissions 10+ years later. Any uncertainty associated with such forecasting will impact the emissions estimates, including how well the model reflects changes in regulations, combustion technology, emissions controls, electricity demand, and human behavior. For example, energy forecasts do not account for economic recessions, which would generally lead to lower energy demands and decreased air emissions. Importantly, a 2001 inventory would not reflect significant investments in emissions controls at Ameren's plants that have been made since that time. In the EIP analysis, modeling was conducted based on more recent emissions inventories, and the model runs may therefore be less uncertain in this area than the CATF estimates.

A more important source of uncertainty is associated with how emission estimates are used to determine the relative contributions to county-level, ground-level $PM_{2.5}$ concentrations. In both the CATF and EIP analyses, the S-R matrix was used to estimate concentrations of $PM_{2.5}$ at each county center. Although the model projects ambient $PM_{2.5}$ increments from emissions of various PM-forming pollutants, it relies on overly simplified algorithms to predict the conversion of these pollutants to $PM_{2.5}$. The algorithm uses fixed transfer coefficients to calculate the annual average $PM_{2.5}$ concentrations at a single receptor in each county (a hypothetical monitor located at the center of the county) and the contribution of $PM_{2.5}$ precursor species from each emission source.

Because of the simplified nature of the source-receptor model, there is significant uncertainty in the estimated contributions from secondary formation of $PM_{2.5}$ (PM formed from precursor pollutants) to the total concentration of $PM_{2.5}$. The model fails to account for all the complex chemical interactions that take place in the atmosphere in the formation of secondary PM. For example, a study by Levy *et al*. (2003) compared results using an earlier version of the S-R matrix with those of a more sophisticated model (CALPUFF). The results of this study showed that, for northern Georgia, the models yielded similar results for $PM_{2.5}$ produced from SO_2 emissions, but not for $PM_{2.5}$ produced from NO_x emissions, likely due to differences in how the models quantified the conversion of NO_x to secondary $PM_{2.5}$. In addition, Levy *et al*. (2003) cautioned that the results of his study did not validate the accuracy of the S-R matrix, but rather showed the relationship between the S-R matrix and a more sophisticated model for a single location and time period. They noted that the results would likely vary depending on the location modeled because temperature, humidity, and season are important factors impacting the formation of secondary particles. Importantly, the Levy *et al*. (2003) study also demonstrated that the estimated health impacts to the surrounding community are minimal compared to health impacts from secondary formation of $PM_{2.5}$ calculated for hypothetical populations that are far removed from the source. As pointed out earlier, a significant fraction of the Ameren CFPP "impacts" derives from tiny $PM_{2.5}$ increments multiplied by large populations long distances away from the plants.

Meteorological parameters used in the air quality modeling are also important inputs, as they are used to develop the transfer coefficients used in the S-R Matrix model. Both CATF and EIP estimated meteorological inputs based on annual averages of data collected over a single year (1990) for just 100 weather stations across the U.S. The S-R Matrix model presumably used the 1990 annual average data for weather stations closest to each county center for which ambient $PM_{2.5}$ increments were estimated, but with such few weather stations and a single year of data, these data cannot be expected to be representative of conditions for each specific locale or year being modeled (*e.g.*, each St. Louis-area county in 2011 or 2012). This is an additional and potentially large source of uncertainty; that is, variability in local weather conditions from those reflected in the data for a distant weather station, as well as variability from year to year, can be significant. To accurately model any locale, such as St. Louis,

representative meteorological data are required; *i.e.*, the gross oversimplification of meteorological data in the S-R Matrix model is a potential source of inaccurate modeling output.

This was demonstrated in a calibration exercise in which Abt Associates (2010) applied the S-R matrix methodology to all $PM_{2.5}$ sources to predict county-level $PM_{2.5}$ concentrations, which were compared to measured data from monitors in each state. The S-R air dispersion model over-predicted $PM_{2.5}$ concentrations for all states, indicating that the S-R model likely significantly over-predicted concentrations of $PM_{2.5}$ associated with emissions from power plants. In addition, this test calibration was based on a comparison of only one year of measured $PM_{2.5}$ data. This calibration approach assumes that the ratio between the modeled projection and measured data in any given county is the same for each modeled year and for each modeled source. However, it is possible that this ratio changes year to year, which would have a very significant impact on the modeled estimates of secondary $PM_{2.5}$ concentrations in each county that are attributed to specific power plants.

There are also issues and uncertainties related to the methodology that CATF and EIP used to associate the modeled secondary $PM_{2.5}$ concentrations at each county location with the source of these precursor pollutants. Specifically, secondary $PM_{2.5}$ is formed from emissions of different gaseous compounds (*e.g.*, NO_x, SO_2, NH_3, and VOCs), and these air pollutants are associated with many different sources, including, but not limited to, power plants. Some forms of $PM_{2.5}$, such as ammonium nitrate and ammonium sulfate, result from reactions among several of these pollutants (*i.e.*, NH_3 and either NO_x or SO_2, respectively). Therefore, $PM_{2.5}$ that is attributed to a specific source would be formed only if all the necessary precursor compounds are present, including precursor pollutants that may not come from the same source. This is especially true for PM from NO_x emissions, because formation of ammonium nitrate is assumed only when there is excess ammonium present, and only under low temperature conditions. As such, the modeled $PM_{2.5}$ concentrations in each county that are attributed to a specific power plant are highly uncertain because the model assumes that all of the necessary precursor pollutants are present, or that conditions are favorable for $PM_{2.5}$ formation, when this may not be the case for a given county.

5.3.2 Uncertainties with health risk estimates

5.3.2.1 Mortality

CATF (2014) reported that model predictions of nationwide impacts for the four Ameren St. Louis-area CFPPs add up to 169 premature deaths each year (Table 5.1), based on projected 2012 conditions. Although we do not have details on the updated analysis conducted by CATF, it is likely that only the emissions estimates were changed and other aspects of the analyses remained the same as in prior analyses (CATF, 2010; Abt Associates, 2010), including the assumed "effect factor" for the mortality estimates. Specifically, in 2010, CATF calculated mortality from power plant emissions based on county all-cause mortality rates (for ages 30 years and older) and a mortality effect estimate derived from the epidemiology study by Pope *et al*. (2002) (Abt Associates, 2010). The widely cited study by Pope *et al*. (2002) is one of several large cohort studies that have investigated the associations between mortality and $PM_{2.5}$. The Pope study relies on a pre-existing cohort (called "CPS-II") recruited by the American Cancer Society (ACS), composed of self-nominated volunteers residing in over 150 cities (Jacobs *et al*., 2001).

As shown in Table 5.1, EIP calculated a larger number of deaths from each of the three Ameren plants it included in its analysis as compared to CATF, providing a range of deaths based on results from two epidemiology studies. The lower-bound estimate was based on the HEI (2009) study, which is a more recent follow-up study using the ACS CPS-II cohort, and the upper-bound estimate was based on the study by Schwartz *et al*. (2008). Schwartz *et al*. (2008) presents a follow-up of another large and often cited cohort study, the Harvard Six Cities cohort, which was originally conceived in the mid-1970s. In this study, over 8,000 adults ages 25-74 years were randomly selected from six cities across the U.S.

(Watertown, MA; Harriman, TN; St Louis, MO; Steubenville, OH; Portage, WI; and Topeka, KS), and followed until 1998. Compared to CPS-II, this study was much more limited in participant number and spatial coverage. The differences in the mortality "effect factors" from the two studies highlight how different model assumptions can impact risk estimates. Specifically, the choice of epidemiology study from which the "effect factor" is derived dramatically affects the resulting risk estimate. In addition, as discussed below, numerous issues with these epidemiology studies must be considered when interpreting the hypothetical health impact assessments. Many of these issues likely lead to grossly overestimated mortality estimates. These issues are briefly discussed below, but have been the topic of many reviews (*e.g.*, see Fewell *et al*., 2007; Gamble, 1998; Valberg and Watson, 1998; Gamble and Nicolich, 2000; Stieb *et al.*, 2002; Valberg, 2004; Koop and Tole, 2004; Moolgavkar, 2005, 2006; Phalen, 2002; Taubes, 1995; Keatinge, 2002; Keatinge and Donaldson, 2001, 2006).

The major (and unproven) assumption behind calculating mortality projections from observational epidemiology is that the reported statistically significant associations represent causality, *i.e.*, that exposure to increments in $PM_{2.5}$ levels *per se* causes clinical health effects, including death. Although the particular studies that were used in the models did report statistically significant associations between $PM_{2.5}$ and mortality, many other studies have shown no such effects (*e.g*., Chay *et al*., 2003; Beelen *et al*., 2008; Brunekreef *et al.*, 2009; Enstrom, 2005; Greven *et al*., 2011; Kloner *et al*. 2009; Lipfert *et al*., 2006; McDonnell *et al*., 2000; Vedal and Dutton, 2006; Zeger *et al*., 2008), indicating that there remains significant uncertainty regarding the causal linkage between ambient PM and mortality risk. In recent health effects evaluations similar to the CATF and EIP evaluations, U.S. EPA acknowledged that "[i]f the PM/mortality relationship is not causal, it would lead to a significant overestimation of net benefits" (U.S. EPA, 2011a).

Exposure misclassification also plagues the observational epidemiology. Most epidemiology studies (including the ones providing "effect factors" for the CATF and EIP models) rely on data from central ambient monitoring sites to provide community averages of ambient pollutant concentrations. Interpretation of the statistical results requires assuming that these "community averages" reflect people's actual personal exposures. That is, in the statistical correlations, individuals are assumed to be exposed 24/7, each day of the year, to the $PM_{2.5}$ concentration measured outdoors at the nearest U.S. EPA ambient $PM_{2.5}$ monitor. In reality, people spend most of their time indoors at home or in other environments and the difference between the measured levels and actual personal exposures results in what is called exposure measurement error. That is, because people spend a large majority of their time indoors and are exposed to different air pollutant concentrations than those measured at central outdoor monitors, error is introduced in the exposure estimates (*e.g.*, Lioy *et al*., 1990; Mage and Buckley, 1995; Janssen *et al*., 1997, 1998; Ozkaynak *et al*., 1996; Dominici *et al*., 2003). In some cases, there may be additional error if there are large temporal gaps in the data from ambient air monitors, as in the case of the Pope *et al.* (2002) study, where some of the data had to be estimated. The amount and direction of the exposure measurement error in the epidemiology studies are generally unknown. But, based on analyses of the effects of this type of error, it is likely that the "effect factors" from the epidemiology studies used by CATF and EIP are unreliable and overestimated because of this issue (*e.g.*, Rhomberg *et al*., 2011).

Another common issue in air pollution epidemiology studies is confounding. A confounder is a factor associated with both the exposure and the health outcome, and therefore it is a factor that may explain in part or in full the observed association between the air pollutant and the health endpoint (in this case $PM_{2.5}$ and mortality). Confounding is a major challenge in epidemiology analyses because there are so many potential confounders, including co-pollutants, meteorological factors, societal factors related to PM emissions, and individual exposure factors, which cannot be accounted for because of a lack of data. Also, even when studies consider potential confounders (such as weather, or day of week), data may be incomplete and residual confounding remains an issue. This is a particular problem in the ACS studies, in which several potential confounding factors were considered, including smoking, education, body mass

index (BMI), diet, alcohol consumption, and occupation. However, these factors were evaluated only at the time of enrollment (1982) and not during follow-up (up to 30 years later). Therefore, there was no information on how these factors changed over time, and how these changes influenced the PM-mortality associations. Furthermore, many of these factors were collected using a self-administered questionnaire, an approach that is well known to result in errors and under-reporting of key potential risk factors for mortality (*e.g.*, smoking).

Confounding by other pollutants is of particular importance. There is evidence that these "co-pollutants" can confound associations in PM mortality studies – particularly strongly correlated pollutants. For example, HEI (2009) reported associations between several pollutants and mortality in single-pollutant models, but they did not present results from multi-pollutant models (which would account for the combined effects of multiple pollutants). Mortality risks reported for several pollutants (*e.g.*, SO_2 and summertime O_3) were of similar magnitude and statistical significance as $PM_{2.5}$ (HEI, 2009), suggesting the possibility of confounding. In fact, in an earlier re-analysis of the ACS study, HEI (2000) found that when accounting for other pollutants, $PM_{2.5}$ risk estimates were decreased. Importantly, the issue of confounding relates to both the assumption of causality, where another factor may actually be the causal agent, and the magnitude of the association, where a co-factor may account for some of the observed risk. In both instances, when confounding is not considered, the risk estimates are overestimated. Finally, many other important air pollutants – *e.g.*, the so-called 187 hazardous air pollutants (HAPs) – could play a confounding role; however, because the HAPs are not measured on a routine basis, their confounding role can neither be evaluated nor corrected for.

The interpretation of the observational epidemiology correlations is also markedly affected by which statistical models are used. To address this question, researchers have conducted extensive sensitivity analyses, including tests of the effects of various model assumptions on mortality estimates. The epidemiology studies relied upon in the modeling by CATF and EIP as the source of effect factors (*i.e.*, Pope *et al*., 2002; Schwartz *et al*., 2008; HEI, 2009) have generally used the Cox proportional hazards (PH) model, which has been widely criticized. Moolgavkar (2005) noted that the assumptions of the Cox PH model are not satisfied in studies of pollution health effects, and therefore can result in inaccurate estimates. For example, Abrahamowicz *et al*. (2003) tested the effects of alternative model assumptions in a subset of the ACS CPS-II cohort (the same cohort used by Pope *et al*., 2002 and HEI, 2009), for which 50 cities had some $PM_{2.5}$ data. The authors found that by using alternative, and likely more appropriate model assumptions, the "effect factors" generated for $PM_{2.5}$ were lower. This suggests that epidemiology studies that use the Cox PH model may overstate effect estimates. In addition, other researchers have shown that model uncertainty can be large. For example, Koop and Tole (2004) emphasized that, by neglecting the important issue of model uncertainty, or by focusing on a specific model among many possible options, "most studies overstate confidence in their chosen model and underestimate the evidence from other models," which can result in "uncertain and inaccurate results."

An additional important assumption that is common to air pollution epidemiology studies, and health impact assessments that use these studies, is linearity at low concentrations, meaning that mortality rates are presumed to be directly proportional to increments in ambient $PM_{2.5}$ levels at levels all the way down to zero exposure. Several studies provide evidence that the PM-mortality association is actually non-linear, and a threshold likely exists below which no effects are likely (*e.g.*, Smith *et al*., 2000; Abrahamowicz *et al*., 2003; Gamble and Nicolich, 2006). For example, Smith *et al*. (2000) reported evidence of PM no-effect thresholds at 20-25 $\mu g/m^3$. In addition, Gamble and Nicolich (2006) found that the data from the Harvard Six Cities study show non-linearity and evidence of a threshold below 20 $\mu g/m^3$. Also, Abrahamowicz *et al*. (2003) reported non-linearity in their re-analysis of the ACS CPS-II cohort, with a threshold for $PM_{2.5}$ at around 20 $\mu g/m^3$. This is highly important for effects alleged from CFPPs, as the concentrations resulting from CFPP emissions are, as discussed above, well below these potential thresholds.

Furthermore, a threshold for PM health effects is supported by toxicological, occupational, and controlled human exposure evidence. Toxicity studies demonstrate that the physiological impact and biological mechanism of inhaled PM effects occurs at elevated concentrations only when the lung defense mechanisms are overwhelmed due to the load of particles deposited locally onto tissues (*e.g.*, Oberdörster, 1996, 2002; Valberg and Crouch, 1999; Green *et al*., 2002; Pauluhn, 2011; Valberg *et al*., 2009). For many specific forms of PM, thresholds have been established based on animal, occupational, or controlled human exposure studies. Examples include a no observed adverse effect level (NOAEL) for diesel exhaust particulate exposures of 460 $\mu g/m^3$, yielding a no-effect reference concentration (RfC) of 5 $\mu g/m^3$ (Valberg and Crouch, 1999; U.S. EPA, 2002), and occupational standard threshold limit values (TLVs) for many types of particles (Oller and Oberdörster, 2010; ACGIH, 2014).

Assuming a linear relationship has a significant impact on mortality risk estimates: when a linear, no-threshold function is assumed in health impact modeling when the reality is non-linear, the health effects projections are overestimated; this is especially the case for vast areas of the U.S. that have lower $PM_{2.5}$ levels. For example, as shown in a health impact analysis conducted by U.S. EPA, when a threshold was included, the calculated mortality estimates significantly decreased. Specifically, when U.S. EPA considered a threshold at 20 $\mu g/m^3$ (supported by the scientific evidence discussed above), mortality projections for improvements in $PM_{2.5}$ concentrations from 2000 to 2010 decreased from about 20,000 deaths nationwide to 5,000 deaths or fewer (U.S. EPA, 1999).[16]

Finally, another indication for non-causal bases for the epidemiology correlations is that the quantitative form of the association between $PM_{2.5}$ and mortality depends on U.S. geographical region and season. Researchers have found significant differences in mortality effect estimates across cities and regions that are unexplained despite recent efforts to evaluate factors (*e.g.*, heterogeneity of PM composition) that might play a role (U.S. EPA, 2011b). Therefore, it is problematic – and likely erroneous – to apply a pooled "national effect factor" to all regions of the U.S., as was done in the modeling used by both CATF and EIP. In fact, such an approach erroneously discounts any differential toxicity of $PM_{2.5}$ components, treating all $PM_{2.5}$ components as equally toxic. This assumption is surely incorrect, and significantly impacts the mortality estimates. For example, no evidence exists, either from human exposures or animal studies, showing that inhaling secondary $PM_{2.5}$ formed from emissions of SO_2 and NO_2 (*e.g.*, sulfates, nitrates, included in the CATF and EIP models), at current ambient levels, leads to mortality or morbidity (Green *et al*., 2002; Utell *et al*., 1983; U.S. EPA, 1996).[17] A risk assessment analysis that evaluated the toxicity of ambient PM on the basis of the chemicals composing PM found that none of these chemicals were present at high enough concentrations to cause adverse health effects, let alone hospital admissions or death (Valberg, 2004). Moreover, Rohr and Wyzga (2012) performed a comprehensive review of $PM_{2.5}$ component-based studies in the epidemiological, toxicological, and controlled human exposure arenas, indicating that there was stronger evidence for some particle types (*e.g.*, carbon-containing components) and greater disparity in study findings for other particle types (*e.g.*, sulfates). Therefore, if the $PM_{2.5}$ chemical species linked with power plant emissions and entered into the models of CATF and EIP are unlikely to contribute to mortality, and if mortality numbers are based on reducing this PM below already innocuous levels, then those numbers are exaggerated and misleading.

[16] Note that the theoretical mortality projections in U.S. EPA (1999) were based on an air modeling analysis of 2000 to 2010 improvements in $PM_{2.5}$ air quality in the U.S. resulting from the Clean Air Act. Currently, based on 2013 design value reports available on the U.S. EPA Air Trends website (http://www.epa.gov/airtrends/values.html), there are no U.S. locations with annual average 2011-2013 $PM_{2.5}$ design values in excess of 20 $\mu g/m^3$.

[17] Airborne sulfate is widely used in medicine. It is a common ingredient in bronchodilators used to treat asthma. One "puff" of an albuterol sulfate inhaler delivers a concentration of about 10,000 μg of sulfate per m^3 of inhaled air, which is considered to be safe (Green *et al*., 2002).

5.3.2.2 Morbidity

Many of the same problematic issues as to the interpretation of the mortality results from epidemiology studies are also evident in studies of morbidity outcomes. CATF quantified health effects other than mortality, such as asthma attacks and asthma ER visits, heart attacks, hospital admissions, and chronic bronchitis (Table 5.1 and 5.2). Below, we discuss some of the issues specific to these morbidity outcomes.

The evidence that ambient PM affects asthma and contributes to asthma attacks or ER visits is inconsistent. Not all studies have reported a significant association. CATF based its estimates on pooled estimates from two small studies that followed panels of asthmatic children (Ostro *et al*., 2001; Vedal *et al*., 1998). The first, by Ostro *et al*. (2001), followed a small group of inner-city African-American children from central Los Angeles and Pasadena, California. The authors reported effects of $PM_{2.5}$ that were of a much lesser magnitude than those of PM_{10} and mold. While the authors considered confounding by temperature and humidity, they did not evaluate the important role of exposure to environmental tobacco smoke (ETS) in the home, which affected nearly 40% of the study population. Vedal *et al*. (1998) examined associations between PM_{10} concentrations and lung function and respiratory symptoms in 75 asthmatic children living in a kraft pulp and paper mill community on Vancouver Island. The authors reported small changes in lung function that were not statistically significant in asthmatic children with exposures to PM_{10}, but they found no clear association for many symptoms of asthma (*e.g.*, cough and wheeze). Importantly, the authors did not control for important confounders such as ETS, other air pollutants, and allergens. Overall, these studies do not support a strong association between asthma and $PM_{2.5}$ exposures. Other studies have also reported a lack of association (*e.g.*, O'Connor *et al*., 2008).

Similar issues are associated with estimates of chronic bronchitis. The evidence from studies that evaluated associations between PM exposure and bronchitis is mixed, with some studies reporting significant associations and others reporting no association (U.S. EPA, 2009). The CATF estimates were based on a study by Abbey *et al*. (1995). There are significant uncertainties associated with using this one study to quantify the effects of $PM_{2.5}$. One issue is that the Abbey *et al*. (1995) study focused on PM_{10} that in turn was estimated from total suspended particle (TSP) concentrations, and likely not representative of $PM_{2.5}$. In addition, confounding is also a significant concern in this study.

With regards to the effects of $PM_{2.5}$ on heart attacks (myocardial infarction, or MI), the CATF analysis relied on a single study by Peters *et al*. (2001) that evaluated the effects of $PM_{2.5}$ on the risk of MI among 772 patients in the greater Boston, Massachusetts, area. Problems with this study included exposure measurement error and confounding by other air pollutants. In addition, the Peters *et al*. (2001) study is only one of several major MI studies. Two other studies (Sullivan *et al*., 2005; Peters, 2005) found no association between $PM_{2.5}$ and MI using similar methods, raising uncertainties with the CATF approach of relying on a single study that observed an association between $PM_{2.5}$ and MI and disregarding other studies that reported no such association.

5.3.3 Uncertainties with health effects valuation

In the final step of the benefits evaluation, estimated costs are assigned to the modeled numbers of cases. Specifically, an estimated economic value is assigned for a particular health effect (*e.g.*, a death or hospital admission), as taken from the literature. For example, for hospital admissions, CATF used cost of illness unit values, which estimate the cost of treating or mitigating the effect. There are significant uncertainties with values related to health effects such as asthma exacerbation, for which CATF used willingness to pay (WTP) unit values. These are, for example, survey-derived estimates of what people are willing to pay to avoid an asthma exacerbation.

By far the most controversial value, which is used for premature mortality, is what is known as the value of statistical life (VSL). The VSL is based on what an individual is willing to pay to reduce the risk of death. It is not the value of an actual life or the price someone would pay to avoid certain death; rather, it is the value placed on changes in the likelihood of death, and the values in Tables 5.1 and 5.2 use $7.2 million per hypothetical death. CATF and EIP evaluations were based on using the VSL to estimate costs from "premature mortality." Since mortality is inevitable, a more appropriate approach, however, would have been to estimate gains in life expectancy, or the value of life-years gained, based on WTP. Similar analyses using this approach yield approximately tenfold lower estimated economic benefits (Cox, 2012).

Overall, problems with the valuation methods used, together with the uncertainty in the health effects estimates, indicate that the modeled health costs reported in Tables 5.1 and 5.2 are likely overestimated.

5.4 Recent epidemiologic studies continue to call into question the causal connection between small increments in ambient $PM_{2.5}$ and premature mortality

Several recent epidemiologic analyses of $PM_{2.5}$ health effects have focused on the statistical models alone, with findings casting doubt on the causal connection between increments in ambient $PM_{2.5}$ and premature mortality, as assumed by CATF and EIP. Greven *et al*. (2011) identified potentially serious flaws in assuming that the chronic $PM_{2.5}$ studies (the cohort studies) reflect causality, because confounding appears to be playing a significant role in the statistical findings of positive $PM_{2.5}$-mortality associations. The investigators used a Medicare data set comprised of 18.2 million records over the period 2000-2006 to investigate associations between long-term $PM_{2.5}$ exposure and changes in life expectancy. The authors divided the PM exposure variable into components, separating how $PM_{2.5}$ varies over space (location) and how it varies over time. They estimated two regression coefficients: a "global" coefficient that reflects the association between national time trends in PM and mortality and a "local" coefficient that measures the location-specific time trends in PM and mortality, adjusted by the national trends. If confounding were not an issue, one would expect these two coefficients to be similar. However, Greven *et al*. found differences between the two coefficients, with no associations for the local coefficient reflecting the relationship between PM reduction and change in life expectancy. That is, mortality risk did not respond to changes in $PM_{2.5}$, and thus, risk analyses based on such non-causal risk coefficients will project health effects where there, in fact, are none.

Cox *et al*. (2013) examined correlations between changes in average $PM_{2.5}$ and corresponding changes in average daily mortality rates, from 1999 to 2000, in each of 100 U.S. cities in the National Mortality and Morbidity Air Pollution Study (NMMAPS) data base. The authors found that "reductions in $PM_{2.5}$ do not appear to cause any reductions in mortality rates," and they concluded "it is crucial to use causal relations, rather than statistical associations, to project the changes in human health risks due to interventions such as reductions in particulate air pollution."

Cox and Popken (2015) examined the mortality effect of the nationwide "natural experiment" whereby $PM_{2.5}$ levels for 483 counties in the 15 most populated U.S. states declined as much as 30% between 2000 and 2010. They compared county-level changes in average annual ambient $PM_{2.5}$ levels to corresponding changes in all-cause and cardiovascular disease (CVD) mortality rates over the course of the decade. Even though some studies have demonstrated statistical associations (which have been interpreted causally or relied upon in the type of calculations made by CATF and EIP), they found that the historical data did not predict a causal basis for the epidemiological associations. The authors concluded: "[Our]

findings suggest that predicted substantial human longevity benefits resulting from reducing $PM_{2.5}$ and ozone may not occur, or may be smaller than previously estimated."

5.5 Experimental data do not support ambient $PM_{2.5}$ causing mortality

The correlations that CATF and EIP use to model reductions in health statistics are from observational studies that examine two factors, which, in small part, seem to go up and down together, namely, changes in mortality (either temporally, say, on a day-by-day basis, or geographically, say, on a city-by-city basis) and changes in levels of ambient $PM_{2.5}$ measured at central monitors from day-to-day, or from city-to-city. Small correlations between $PM_{2.5}$ levels and mortality are reported by the epidemiology studies, but it is not correct to assume the statistical deaths reflected in the PM-mortality correlations are caused by changes in the levels of outdoor $PM_{2.5}$.

The claim of mortality risk being due to present-day ambient outdoor levels of $PM_{2.5}$ is based on assuming a causal link behind the observational epidemiology. This logical leap is taken despite the widespread recognition that statistical correlations are not equivalent to causation (Hill, 1965; U.S. EPA, 2009, p. 1-14: "Causality determinations are based on the evaluation and synthesis of evidence from across scientific disciplines" and "An association…is insufficient proof of a causal relationship between exposure and disease or health effect").

Interpretation of correlations between air pollution levels and health effects needs to be supported by experimental science and clinical evidence. Although we can clearly identify who dies of car accidents, food poisoning, firearms, viral and bacterial infections, and so forth, for the CATF and EIP model predictions of deaths from breathing outdoor air, not a single individual has been identified, or reported in the medical literature, whose death would have been prevented, but for him/her inhaling low levels of ambient $PM_{2.5}$. Laboratory experiments have carefully examined animals exposed to high levels of airborne PM, and human volunteers have breathed PM in clinical settings, including at $PM_{2.5}$ levels hundreds of times higher than found in outdoor air, with no evidence of sudden death or any life-threatening effects (Schlesinger and Cassee, 2003).

The major amount of ambient $PM_{2.5}$ generated from power plant emissions is secondary sulfate (SO_4^{2-}), and sulfate is suggested as one of the primary chemical components in PM that is responsible for the mortality effects of $PM_{2.5}$. However, biological evidence for mortality or life-threatening health effects being caused by sulfate at ambient concentrations is entirely lacking. Clinical experiments have exposed human volunteers (including children and asthmatics) to sulfate particulates at high concentrations (35 to 2,000 $\mu g/m^3$) in combination with heavy exercise (Linn *et al.*, 1981, 1989, 1994, 1995, 1997; Hackney *et al.*, 1989). These laboratory concentrations were about 6 to 330 times greater than St. Louis ambient levels of sulfate (~6 $\mu g/m^3$). Even exposures at such elevated SO_4 levels resulted only in modest and reversible changes in pulmonary function measurements, and nothing has ever been observed even approximating life-threatening effects in these laboratory experiments on humans breathing high levels of SO_4 (compared to ambient).

Furthermore, sulfate is a common constituent of airborne medications that are repeatedly inhaled by asthmatics to alleviate airway narrowing. Personal inhalers for asthmatics (inhalers that produce an aerosol of a bronchodilator drug) use a sulfate salt of the active drug (*e.g.*, albuterol sulfate, metaproterenol sulfate, terbutaline sulfate). For example, a typical dosage of albuterol sulfate inhalation solution administered by nebulization three to four times a day corresponds to daily inhalation of approximately 1.3 to 1.7 mg of sulfate, or over 10 times greater than the average daily amount inhaled from ambient air (at 6 $\mu g/m^3$). Inhalation of the "sulfate" in asthma medications is not known to cause harm to asthmatics, let alone precipitate an asthma attack or hasten death.

In summary, the CATF and EIP calculated increases in deaths and other health effects (Tables 5.1 and 5.2) lack plausibility and experimental support, and their hypothetical nature must be recognized.

5.6 Comprehensive risk evaluations of hazardous air pollutant (HAP) emissions from U.S. coal-fired power plants do not provide support for significant health risks from primary PM emissions from CFPPs

If dramatic health effects, such as those listed in Table 5.1, are to be attributed to airborne particles deriving from coal-fired power plants, there must be some chemical constituent(s) contained within the particulate matter that is(are) highly toxic. While the studies discussed above bear on the potential health risks related to secondary $PM_{2.5}$ (sulfates and nitrates) formed from CFPP emissions, this question of "mode of action" has been exhaustively addressed for primary PM emissions by several health risk assessments of HAPs present in "utility boiler" air emissions. The possibility of health effects from coal-fired power plant air emissions has been examined by U.S. EPA and others using quantitative health risk assessment (French *et al*., 1997; U.S. EPA, 1998, 2011c; EPRI, 2009). Overall, these studies have demonstrated that ambient air levels of hazardous air pollutant (HAP) chemicals, including trace metals in particulate form, emitted by power plants are below thresholds of health concern.

The results have been remarkably consistent for all these evaluations. In its 1998 assessment, U.S. EPA considered the 67 most potentially toxic chemicals that could be present in gas and particle air emissions from power plants. The agency concluded that, on either a plant-by-plant basis or on a nationwide basis, the "potentially hazardous" coal-fired power-plant air emissions were not expected to lead to significant health impacts. In its assessment, EPRI (2009) concluded that "Under realistic exposure scenarios, inhalation risks of cancer are below one in one million for all power plants, and inhalation risks of non-cancer toxic effects are well below federal threshold levels for all power plants." EPRI also concluded that population-weighted inhalation risks were "insignificant."

U.S. EPA's recent (2011c) "Mercury and Air Toxics Rule" for hazardous air pollutant emissions from fossil-fuel power plants found minimal expectation of potential non-cancer health effects from power plant HAPs emissions: U.S. EPA found that "All of the facilities had non-cancer target-organ-specific hazard index values less than one…" and that the coal units never exceeded a non-cancer hazard index of 0.05[18] (whereas the level of potential concern would be HI >1.0). In addition, the highest cancer risk that was estimated, 5 in one million lifetime risk, was well within U.S. EPA's acceptable cancer risk range (1 in 10,000 lifetime risk to 1 in 1,000,000 lifetime risk).

[18] A hazard index (HI) is the ratio of the received dose divided by a dose that is low enough to be health protective, *e.g.*, a "reference dose." Hence, HI values that are less than 1.0 indicate that no adverse health effects are anticipated.

6 Asthma is a complex, multi-factorial disease, with a multitude of known triggers and risk factors. Scientific studies provide evidence that asthma prevalence and morbidity are more closely linked to allergic status, lifestyle factors, and indoor air pollution than to indicators of outdoor air pollution.

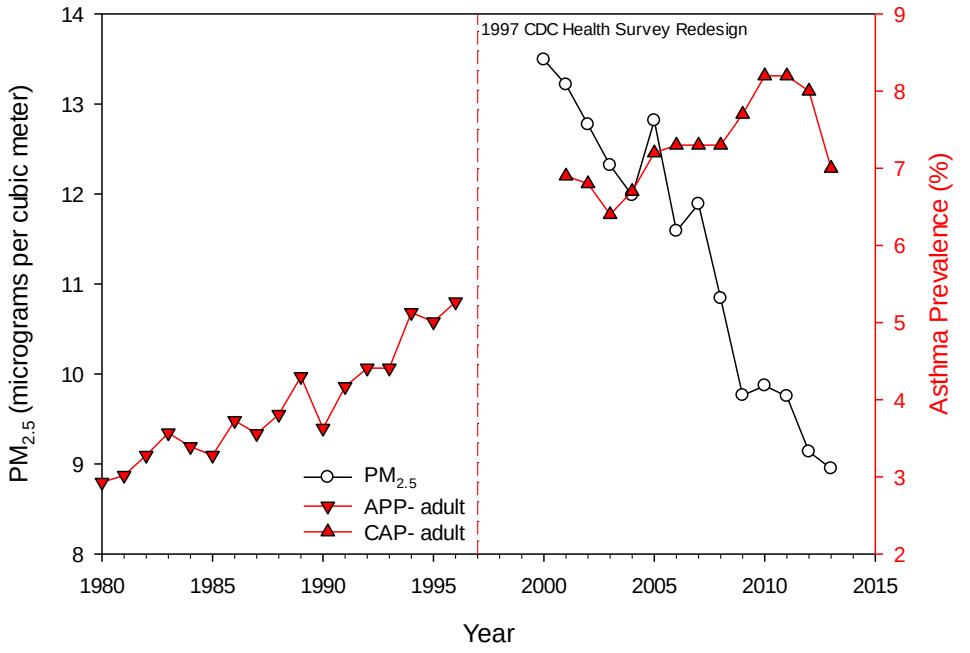
Over the past several decades, asthma prevalence[19] in the U.S. and other countries has been rising, but the cause is unclear. Although there has been an increase in asthma prevalence over the past 20 to 30 years, this increase has occurred during a period of time when concentrations of anthropogenic air pollutants in outdoor air have been decreasing (Anderson, 1997; Anderson *et al*., 2012). This is illustrated by Figure 6.1 that compares national-scale trends in several criteria air pollutants ($PM_{2.5}$, PM_{10}, O_3, SO_2) over the past two to three decades with national-scale trends in asthma prevalence for both adults and children. As reflected by this graph, ambient air pollutant emissions and concentrations in the United States have decreased significantly over the past several decades while the prevalence of asthma has increased, providing support for the conclusion that factors other than exposure to outdoor air pollutants are more important risk factors underlying the trends in increased asthma prevalence.

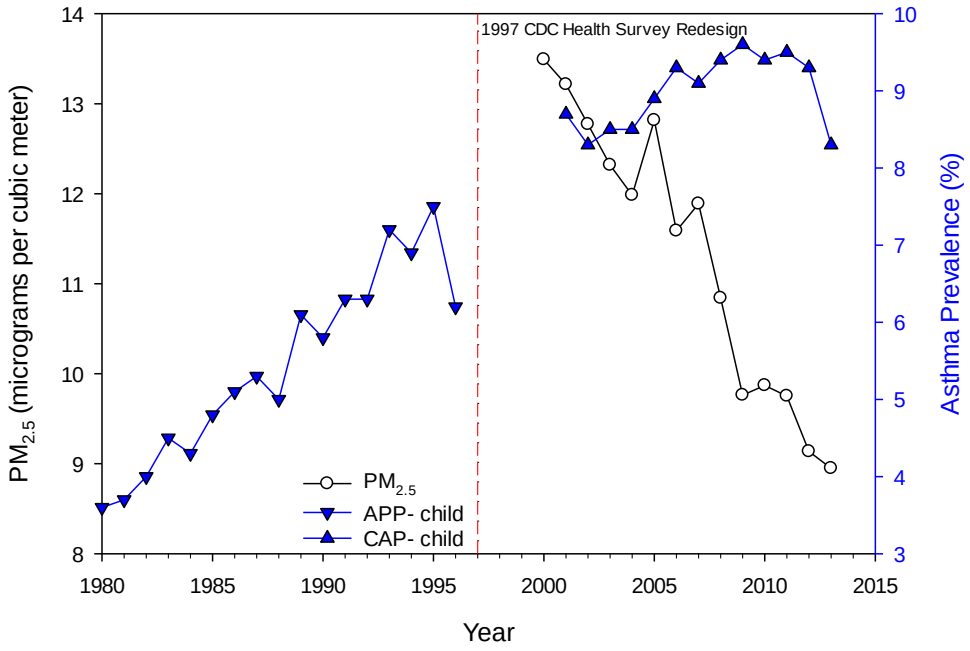
Studies of within-city trends in asthma statistics illustrate the conflicting evidence regarding the nature of the linkage between ambient air pollution and asthma morbidity and mortality. For example, in a study of asthma hospitalizations across communities in the City of Boston conducted by researchers at Boston University, School of Public Health, Gottlieb *et al.* (1995) observed dramatic differences in asthma hospitalizations among different communities. Some areas had asthma hospitalization rates as high as 10 per 1,000, but despite similar ambient air quality in nearby neighborhoods, others had much lower rates of about 1.3 per 1,000.

In New York City, investigators have analyzed the distribution and factors affecting asthma mortality and hospitalization (Carr *et al.*, 1992; De Palo *et al.*, 1994). These studies have shown asthma prevalence in New York City to be related strongly to socioeconomic status, with several factors linking asthma with poor housing conditions. Factors that related to asthma risk in low-income areas were the number of occupants (related to bacterial and viral exposures), water leaks (related to fungi exposures), moist basements (related to fungi exposures), deteriorating building materials (related to fungi and mite exposures), and house dust (related to insects, animal dander, and exposures to animal excreta).

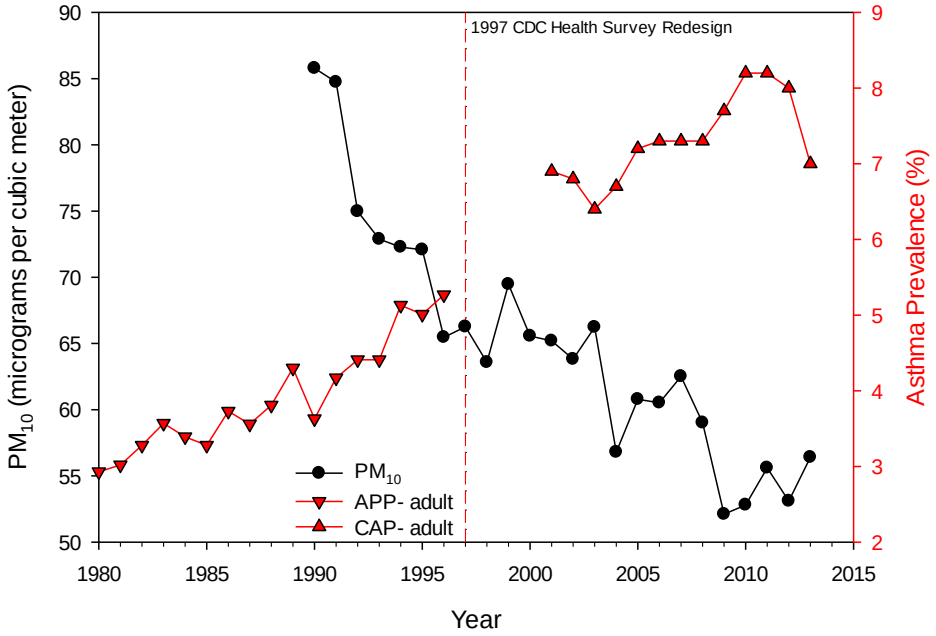
[19] Asthma prevalence is defined as the percentage of people who have ever been diagnosed with asthma and still have asthma at a particular point in time.

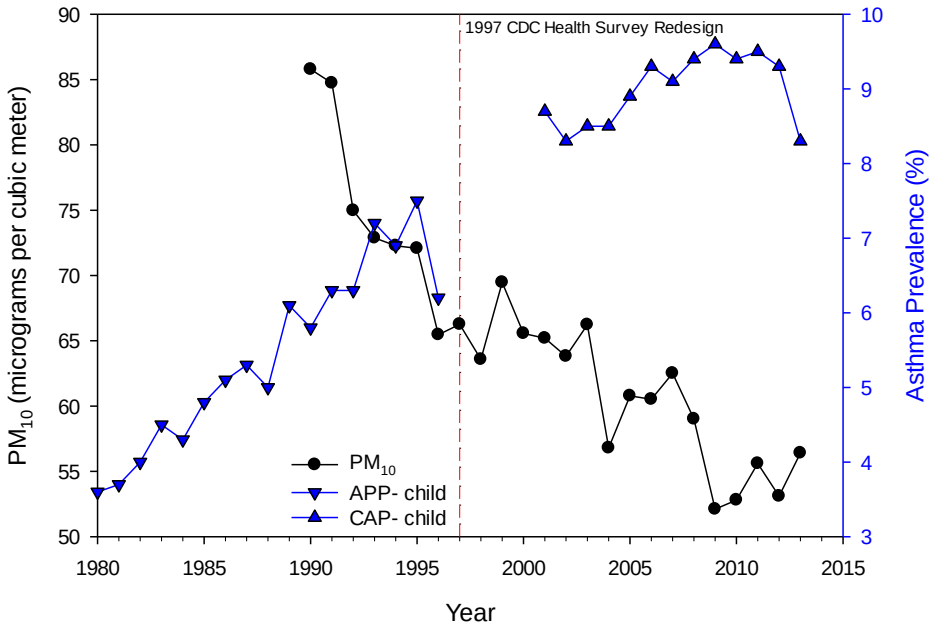
(a) As nationwide $PM_{2.5}$ levels have declined, nationwide adult and pediatric asthma prevalence has increased.



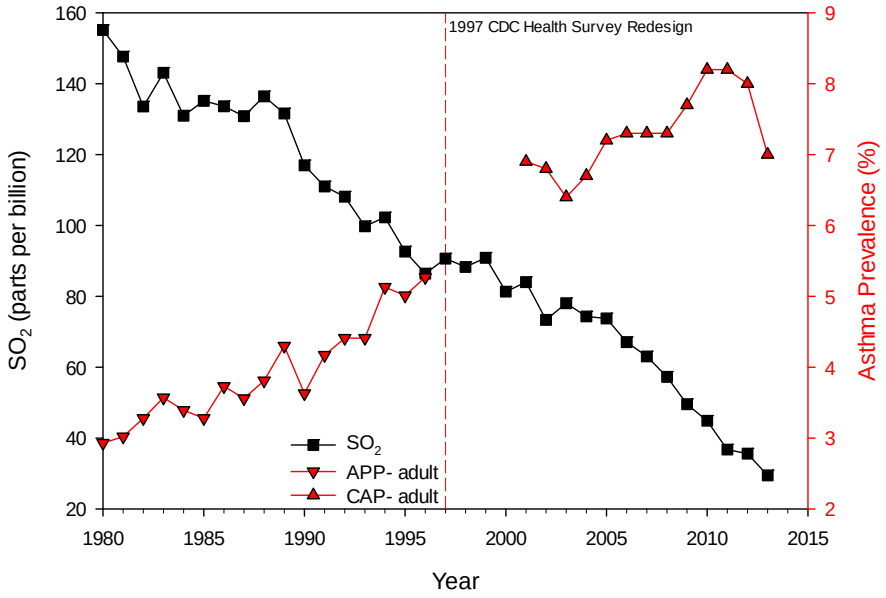


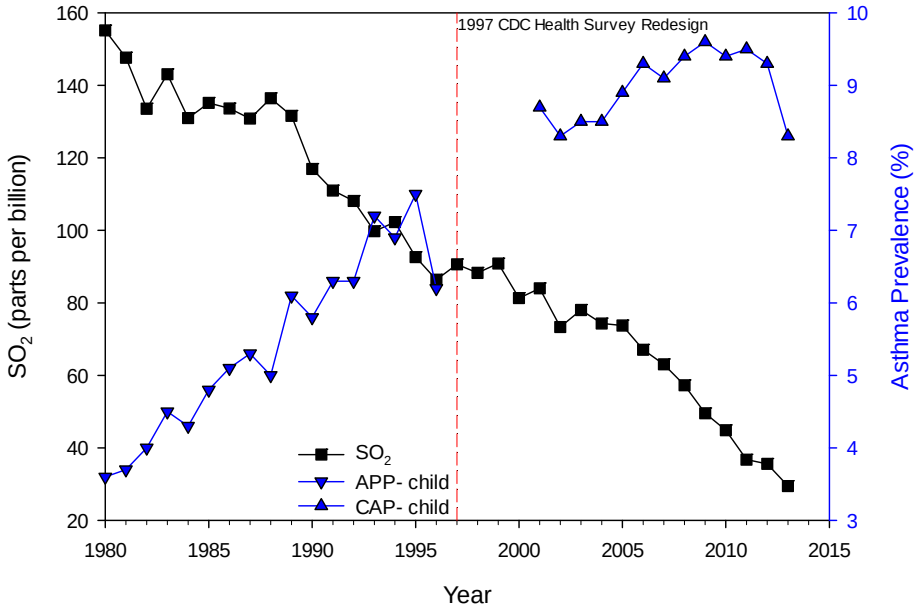
(b) As nationwide PM_{10} levels have declined, nationwide adult and pediatric asthma prevalence has increased.





(c) As nationwide SO_2 levels have declined, nationwide adult and pediatric asthma prevalence has increased.





(d) As nationwide O_3 levels have declined, nationwide adult and pediatric asthma prevalence has increased.

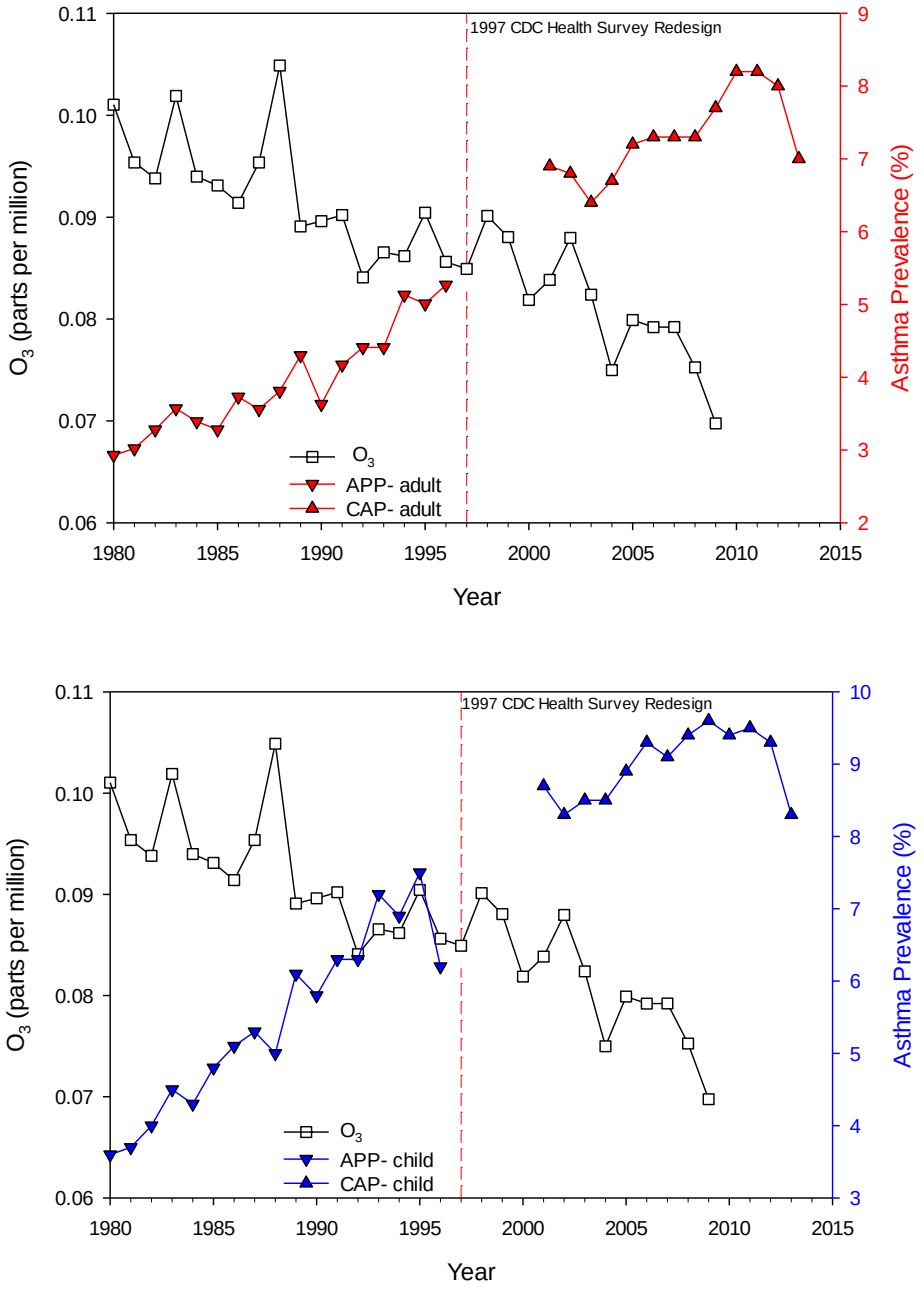


Figure 6.1 National-scale Trends in Several Criteria Air Pollutants with National-scale Trends in Asthma Prevalence for both Adults and Children. Criteria air pollutant data obtained from the U.S. EPA AirTrends website (http://www.epa.gov/airtrends/) and asthma prevalence data obtained from the U.S. Centers for Disease Control and Prevention (CDC) Asthma Surveillance Data website (http://www.cdc.gov/asthma/asthmadata.htm). Prior to 1997, CDC collected data on asthma period prevalence (APP), which

reflects the percentage of the U.S. population having asthma in the previous 12 months. CDC redesigned its health survey in 1997, and in 2001, began to collect data on current asthma prevalence (CAP), which represents the percentage of the U.S. population diagnosed with asthma and having asthma at the time of the survey.

Gupta *et al*. (2008) have demonstrated large geographic variability in childhood asthma prevalence across different Chicago neighborhoods. As illustrated in Figure 6.2 below, the Gupta *et al*. (2008) data for childhood asthma prevalence (green to orange shading) show no apparent relationship between either ambient $PM_{2.5}$ levels (numbers next to green diamonds) or proximity to two coal-fired power plants (purple squares) that were in operation at the time of the study.

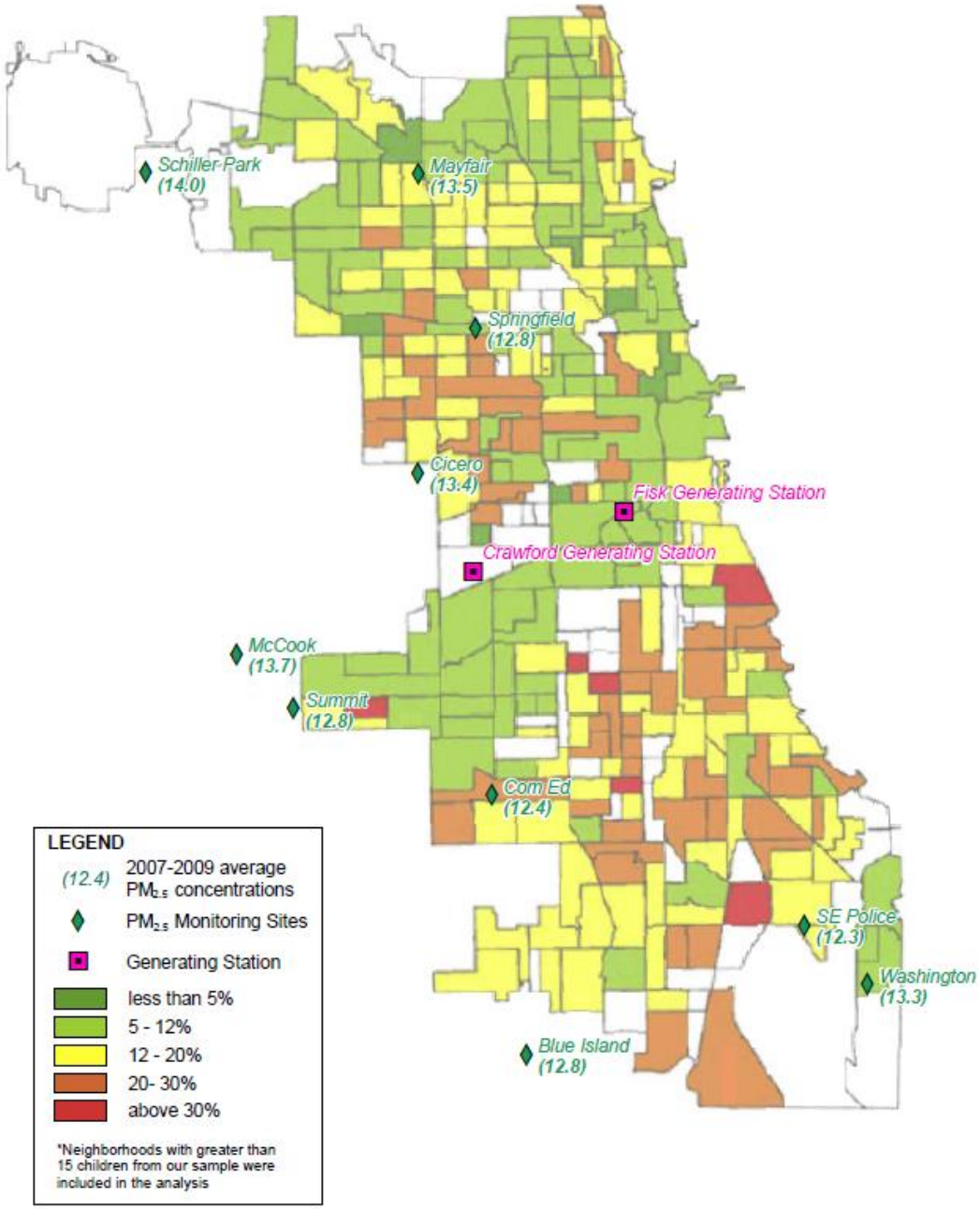


Figure 6.2 Childhood Asthma Prevalence in Chicago Neighborhoods (adapted from Gupta *et al*., 2008)

Importantly, studies worldwide report low prevalence of asthma in countries with high ambient air pollution, such as Mexico, Eastern Europe, China, and Greece, whereas asthma rates are nearly 10 times higher in countries that have very good air quality and much less industry, for example, New Zealand, Australia, and Canada (Peat and Li, 1999; ISAAC, 1998). In the United Kingdom, regional differences in ambient air pollution do not correlate with asthma prevalence (Anderson, 1997; Anderson *et al*., 2012). In Europe, asthma rates are lower in more polluted regions than in regions with cleaner air (Björkstén, 1997; Nicolai, 1997). Also, during the time period following unification, East Germany (a more polluted region) had less asthma than West Germany (Krämer *et al*., 2010).

It is now widely recognized that asthma is a complex, multi-factorial disease, with a multitude of known triggers and risk factors. As reflected in Eggleston (2007), clinicians feel that indoor air is likely much more important than outdoor air for triggering asthmatic symptoms, in part because people spend so much of their time indoors (Eggleston, 2007):

> Our understanding of the environmental influences [of asthma] is still in its infancy, but we can say that indoor exposures are more important than ambient pollutants and that bioaerosols containing allergenic proteins are especially important.

For example, some of the most potent asthma-inducing allergens (such as spores, mold, pollen, and allergens from rodents, pets, fungi, cockroaches, and dust mites) can be found in indoor environments (Carr *et al.*, 1992; De Palo *et al.*, 1994; Leaderer *et al*., 2002; Belanger *et al*., 2003; Teach *et al.*, 2006). A recent review of scientific studies published between 2000 and 2013 regarding indoor environments and asthma found a causal relationship with asthma exacerbation by indoor dampness-related agents, endotoxin, and environmental tobacco smoke (Kanchongkittiphon *et al*., 2015). The authors also reported suggestive evidence for asthma exacerbation by indoor fungi, NO_2 (which is emitted from natural gas appliances such as natural gas cooking burners), presence of rodents, feather/down pillows; and dust mite, cockroach, dog, and dampness-related agents. Also, other studies have linked fragranced consumer products, such as air fresheners, deodorizers, and household cleaning products, with asthma exacerbation (Steinemann *et al*., 2011).

Well-respected authorities on asthma, when describing factors that contribute to asthma prevalence and exacerbation, list many more indoor-air / lifestyle factors as opposed to man-made substances in the outdoor air. For example, collecting asthma risk factors and asthma triggers from the American Lung Association (ALA, 2014), National Heart, Lung, and Blood Institute (NHLBI, 2014), and National Institute of Environmental Health Sciences (NIEHS, 2014) websites yields the list given below. Each person reacts differently to the various risk factors and asthma triggers, but the primary established factors include:

- **colds,** *i.e.*, viral respiratory infections;
- pets and **animals**;
- **cigarette** smoke, environmental tobacco smoke (ETS);
- allergens in **fragrances** (*e.g.*, lilacs), hairspray, and cleaning products;
- **plant materials,** pollens, mold, fungus, mildew, grasses, flowers, house dust;
- **foods,** *e.g.*, sulfites;
- being overweight;
- **animal materials,** animal fur / dander, **dust mites,** cockroaches, feathers;
- indoor and outdoor air pollutants such as **ozone, nitrogen dioxide, sulfur dioxide**;

- exposure to **cold air** or sudden temperature change;
- **maternal stress** during pregnancy period when child was *in utero*;
- excitement/**stress**;
- **exercise,** physical activity; and
- over the counter **medications**, *e.g.*, aspirin or other non-steroidal anti-inflammatory drugs.

It remains unclear as to why the prevalence of childhood asthma has increased over the decades. There is the usual issue of increased diagnostic awareness, and lowered thresholds for what is classified as asthma. Also, the fraction of children who are overweight has increased over time, and high body mass index is a risk factor for asthma (Kwon *et al*., 2006; LeMasters *et al*., 2015). Episodes of high maternal stress during the prenatal period when the child is *in utero* have also been shown to increase the risk of developing asthma (Cookson *et al*., 2009; Turcotte-Tremblay *et al*., 2014). Interestingly, reduced exposure to bacteria and allergens in the first year of life has been linked to an increased risk of developing asthma later in life, suggesting that immune system hypersensitivity (atopy) is exacerbated by infants not experiencing exposure to common antigen challenges (Lynch *et al*., 2014). Exposure to allergens subsequent to the first year of life, however, increased risk of sensitization, wheeze, and asthma.

A number of investigators have examined indoor-environment and lifestyle factors and have found them to play an important part in asthma prevalence and occurrence of asthma attacks. That is, exposures to airborne allergens, dusts, and cooking-related emissions (*e.g.*, NO_2 from natural gas cooking burners) are linked to socioeconomic status and population density in homes, and these exposures increase allergic sensitization and asthma development and exacerbation (Leaderer *et al*., 2002). Teach *et al*. (2006) studied a cohort of children with moderate to severe asthma who were treated in an urban pediatric emergency department, and they correlated these exposures with household income, prior asthma morbidity, health care utilization, and quality of life. They found that increased home exposure to ETS, allergens, and cockroach allergen was significantly associated with low household income. In the presence of ETS, cockroach antigen exposure was associated with adverse effects on asthma morbidity.

Belanger *et al*. (2003) reported that home indoor-air exposures (mold, cockroach allergen, NO_2, fungi) increased the frequency of infant wheeze and persistent cough. A study that collected dust samples, taken from living-area floors and from the child's bed, showed house dust mite allergen concentrations that increased asthma severity (Gent *et al*., 2009). Indoor NO_2 levels, which are known to have gas cooking stoves as primary indoor emission sources, have been linked to asthma morbidity and an asthma severity score (frequency of wheeze, night symptoms, and use of rescue medication) (Belanger *et al*., 2013). The authors reported that asthmatic children exposed to NO_2 concentrations common in urban and suburban homes are at risk for increased asthma morbidity. Some investigators have tested indoor asthma triggers using an intervention approach. For example, in a Michigan study, staff assessed homes for asthma triggers and subsequently provided products and services to reduce exposures to cockroaches, dust mites, mold, tobacco smoke, and other triggers (Largo *et al*., 2011). The investigators found that these interventions substantially reduced the impact of asthma on the children studied, and the proportion of asthmatic children who sought acute unscheduled health care for their asthma decreased by more than 47%.

In a U.S. National Institutes of Health (NIH) study, Arbes *et al*. (2007) reported findings suggesting that more than 50 percent of current asthma cases in the United States can be attributed to specific allergies. Another recent study (Mitchell *et al*., 2009) identified "obesity, antibiotics use, and television watching [sedentary lifestyle]" as risk factors for asthma in children.

In summary, asthma is a multi-factorial disease that has shown significant increases in prevalence over the past few decades, yet, outdoor air pollution, such as ambient $PM_{2.5}$, has been trending downwards over the same time period. The time trends are in opposite directions, and therefore, completely contrary to the hypothesis of a causal link. Data such as these raise questions regarding the impacts of further reductions in outdoor air pollutant levels on asthma prevalence.

As shown in the graphs below (Figures 6.3 through 6.7), there are in fact data specific to the St. Louis area showing that over the many years that $PM_{2.5}$ air quality has been steadily improving, various asthma statistics have remained relatively unchanged. These asthma statistics include:

- In Figure 6.3, weighted prevalence of adult lifetime doctor-diagnosed asthma and crude prevalence of pediatric doctor-diagnosed asthma for the St. Louis Region from the Missouri Behavioral Risk Factor Surveillance System (BRFSS).
- In Figure 6.4, rates (per 10,000) of total all-age asthma inpatient hospitalizations for St. Louis City from the Missouri Information for Community Assessment (MICA) system.
- In Figure 6.5, rates (per 1,000) of total all-age asthma emergency room (ER) visits for St. Louis City from the Missouri Information for Community Assessment (MICA) system.
- In Figure 6.6, rates (per 10,000) of pediatric (<15 years of age) asthma inpatient hospitalizations for St. Louis City from the Missouri Information for Community Assessment (MICA) system.
- In Figure 6.7, rates (per 1,000) of pediatric (<15 years of age) asthma emergency room (ER) visits for St. Louis City from the Missouri Information for Community Assessment (MICA) system.

Moreover, similar to what has been shown for other U.S. cities, there is also evidence that asthma statistics are highly variable between different St. Louis City zip codes despite similarities in outdoor air quality. This is illustrated by Figures 6.8 through 6.10 that examine the following asthma statistics by zip code:

- In Figure 6.8, 2010 rates (per 10,000) of total all-age asthma inpatient hospitalizations for St. Louis City zip codes from the Missouri Information for Community Assessment (MICA) system.
- In Figure 6.9, 2010 rates (per 1,000) of total all-age asthma emergency room (ER) visits for St. Louis City zip codes from the Missouri Information for Community Assessment (MICA) system.
- In Figure 6.10, 2006-2008 rates (per 1,000) of pediatric (<15 years of age) asthma emergency room (ER) visits for St. Louis City zip codes from an undated Missouri DHSS Report (only includes zip codes with more than 300 asthma ER visits during 2006-2008 three year-period).

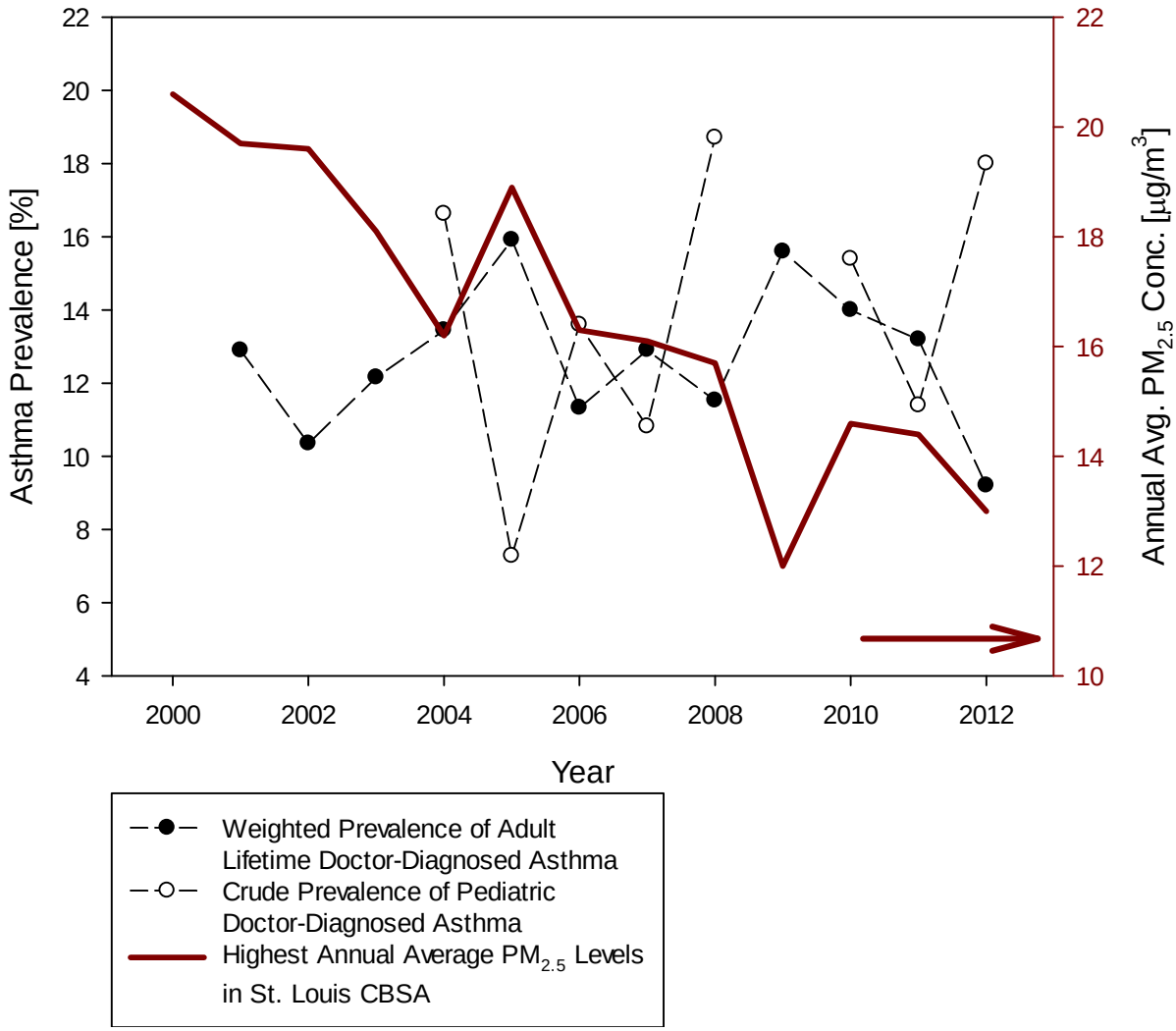


Figure 6.3 St. Louis-area $PM_{2.5}$ Levels Have Dropped by More than 25% While Adult and Pediatric Asthma Prevalence in the St. Louis Region Has Remained Relatively Unchanged. Highest annual average $PM_{2.5}$ concentrations in the St. Louis CBSA obtained from the U.S. EPA AirData website, and doctor-diagnosed adult and pediatric asthma prevalence data in the St. Louis Region obtained from the Missouri Behavioral Risk Factor Surveillance System (BRFSS).

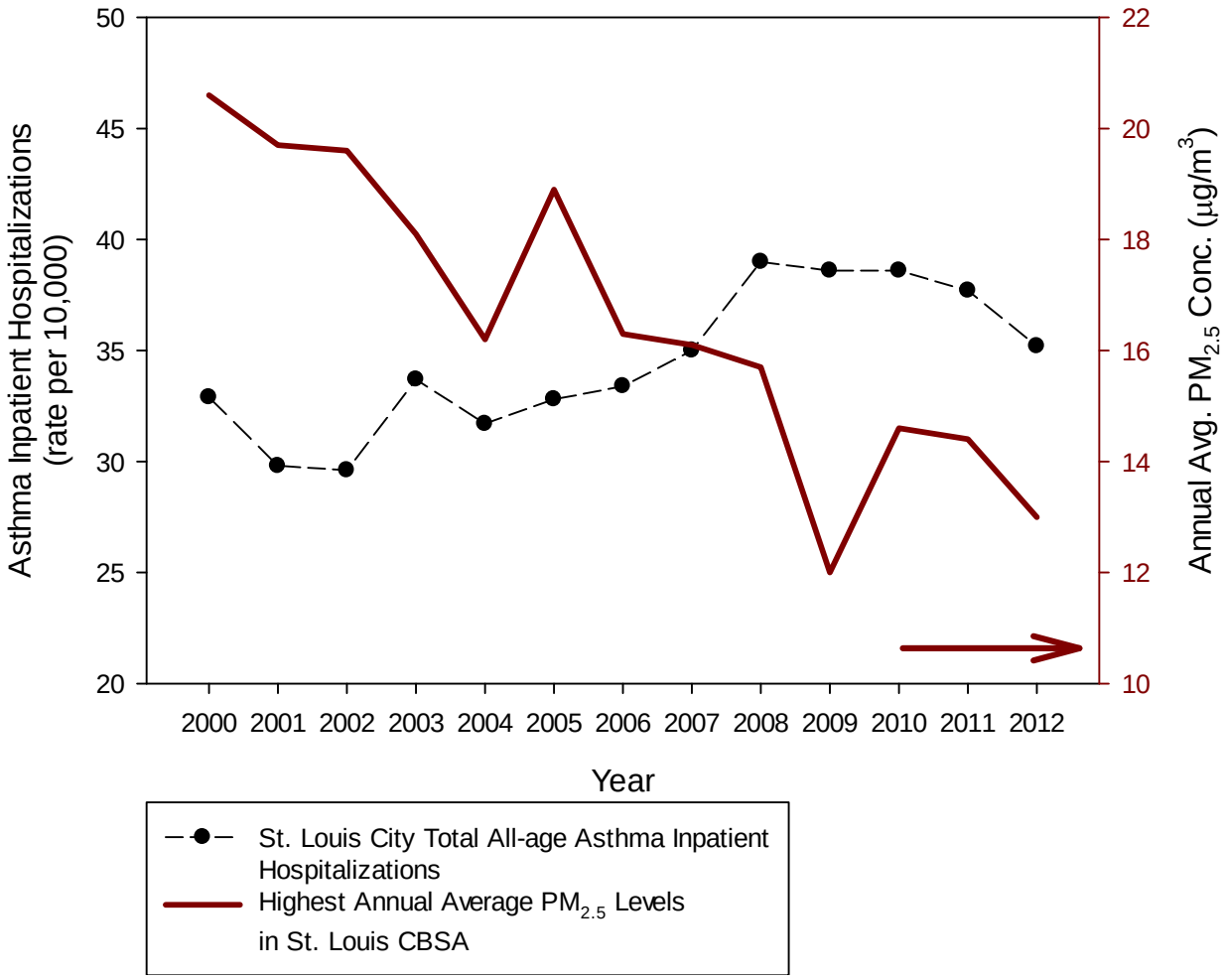


Figure 6.4 St. Louis-area $PM_{2.5}$ Levels Have Dropped by More than 25% While All-age Asthma Inpatient Hospitalization Rates in St. Louis City Have Slightly Increased. Highest annual average $PM_{2.5}$ concentrations in the St. Louis CBSA obtained from the U.S. EPA AirData website, and rates (per 10,000) of total all-age asthma inpatient hospitalizations in St. Louis City obtained from the Missouri Information for Community Assessment (MICA) system.

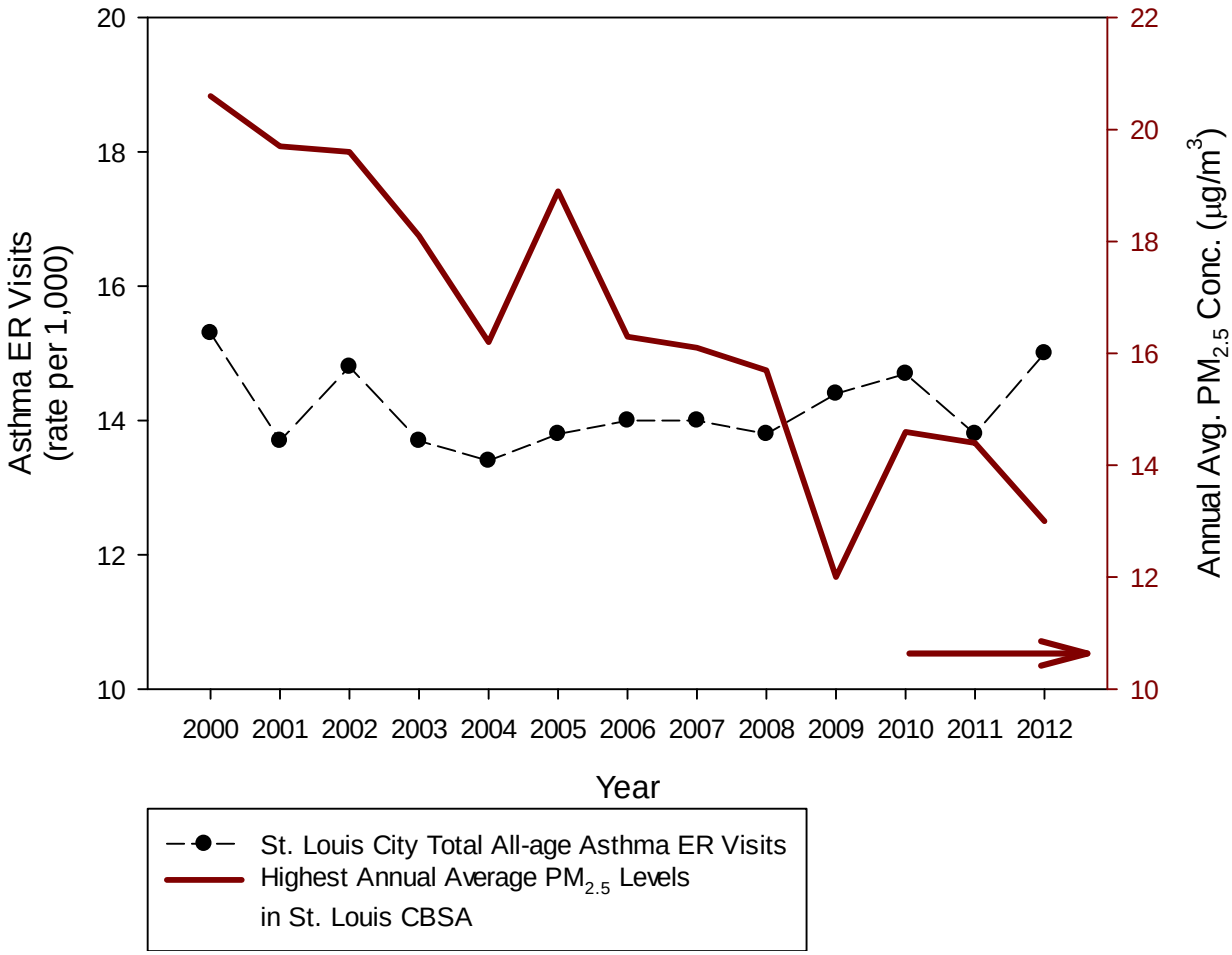


Figure 6.5 St. Louis-area $PM_{2.5}$ Levels Have Dropped by More than 25% While Rates of All-age Asthma ER Visits Have Remained Steady. Highest annual average $PM_{2.5}$ concentrations in the St. Louis CBSA obtained from the U.S. EPA AirData website, and rates (per 1,000) of total all-age asthma emergency room (ER) visits in St. Louis City obtained from the Missouri Information for Community Assessment (MICA) system.

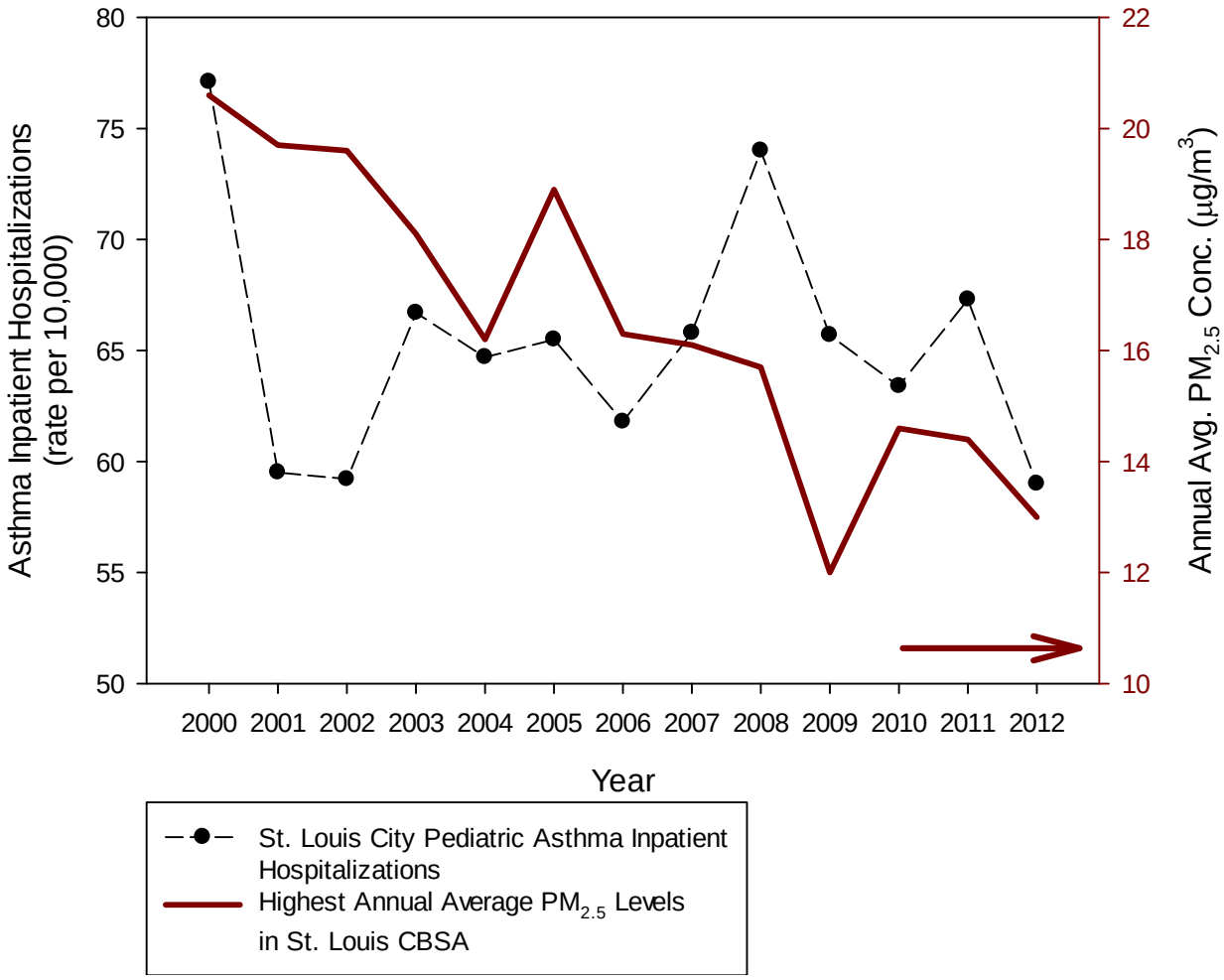


Figure 6.6 St. Louis-area $PM_{2.5}$ Levels Have Dropped by More than 25% While Pediatric Asthma Inpatient Hospitalization Rates in St. Louis City Have Remained Relatively Unchanged. Highest annual average $PM_{2.5}$ concentrations in the St. Louis CBSA obtained from the U.S. EPA AirData website, and rates (per 10,000) of pediatric (<15 years of age) asthma inpatient hospitalizations in St. Louis City obtained from the Missouri Information for Community Assessment (MICA) system.

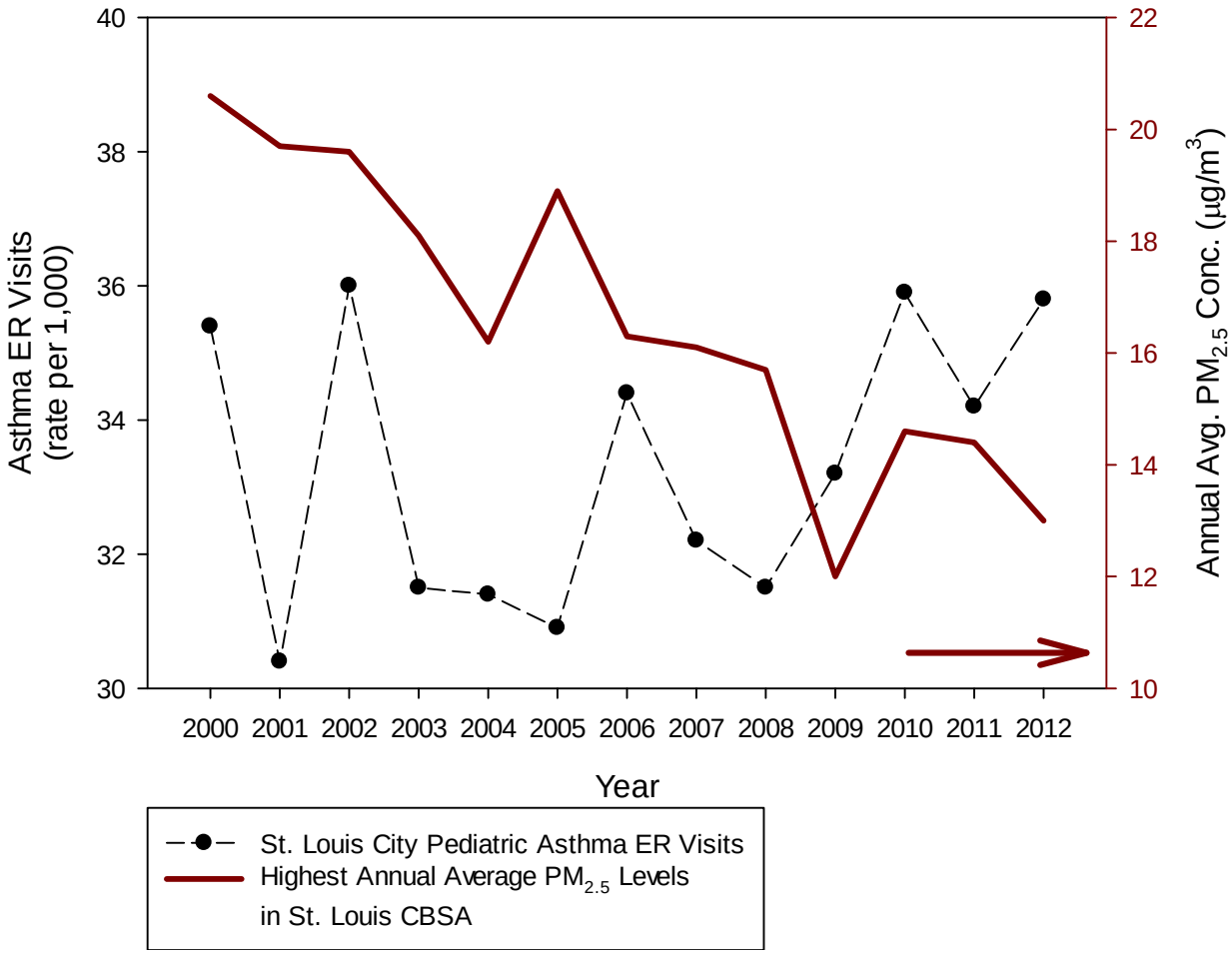


Figure 6.7 St. Louis-area $PM_{2.5}$ Levels Have Dropped by More than 25% While Rates of Pediatric Asthma ER Visits Have Remained Relatively Unchanged. Highest annual average $PM_{2.5}$ concentrations in the St. Louis CBSA obtained from the U.S. EPA AirData website, and rates (per 1,000) of pediatric (<15 years of age) asthma emergency room (ER) visits in St. Louis City obtained from the Missouri Information for Community Assessment (MICA) system.

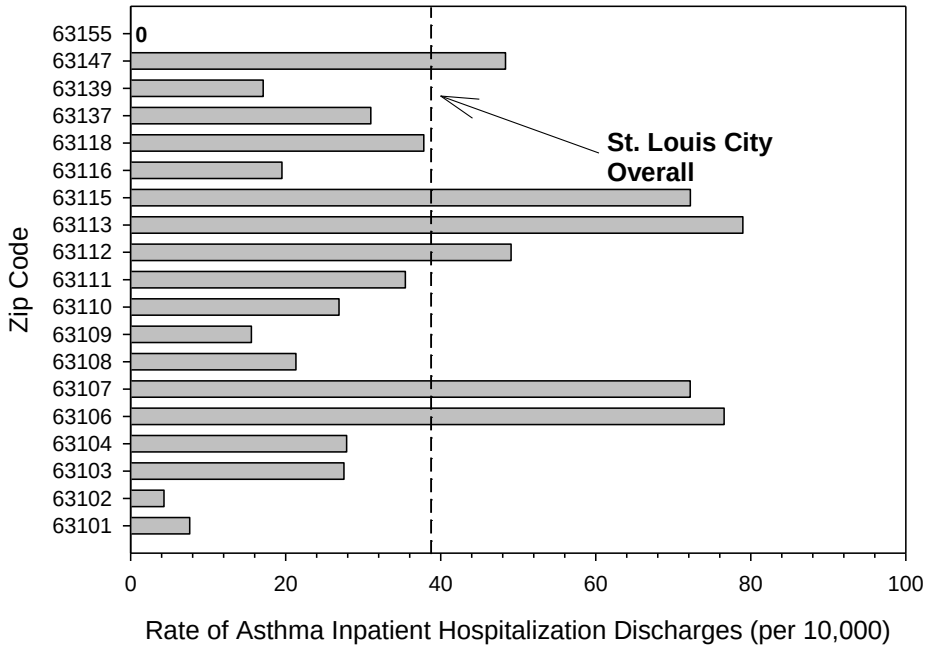


Figure 6.8 Despite Being Located Within the Same Airshed, Rates of Total All-age Asthma Inpatients Hospitalizations Vary Significantly by Zip Code in the St. Louis City Area. 2010 rates (per 10,000) of total all-age asthma inpatient hospitalization discharges for St. Louis City zip codes obtained from the Missouri Information for Community Assessment (MICA) system; numbers of cases converted to rates using 2010 census data.

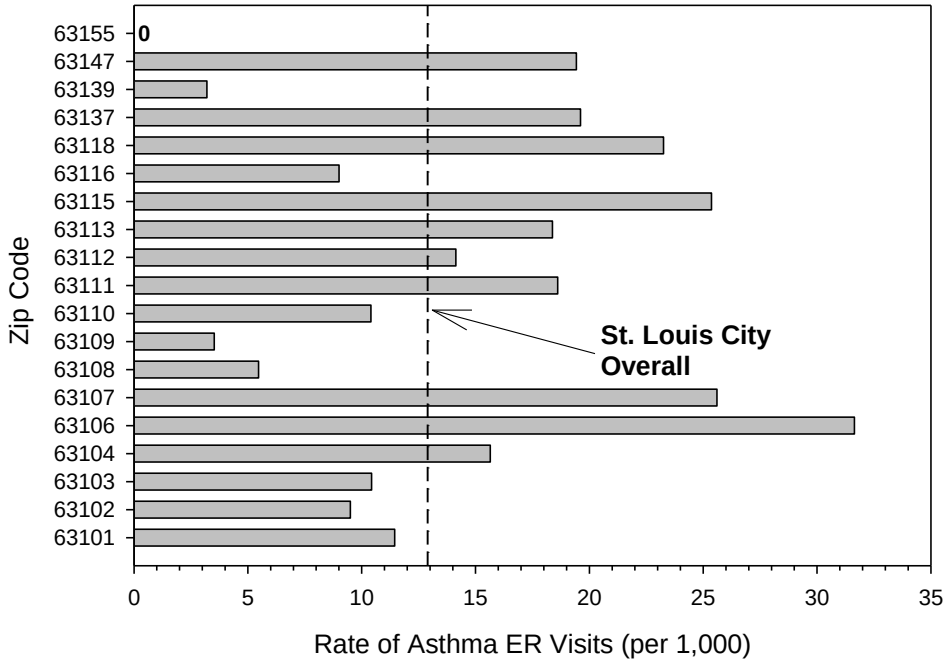


Figure 6.9 Despite Being Located Within the Same Airshed, Rates of Total All-age Asthma Emergency Room (ER) Visits Vary Significantly by Zip Code in the St. Louis City Area. 2010 Rates (per 1,000) of total all-age asthma emergency room (ER) visits for St. Louis City zip codes obtained from the Missouri Information for Community Assessment (MICA) system; numbers of cases converted to rates using 2010 census data.

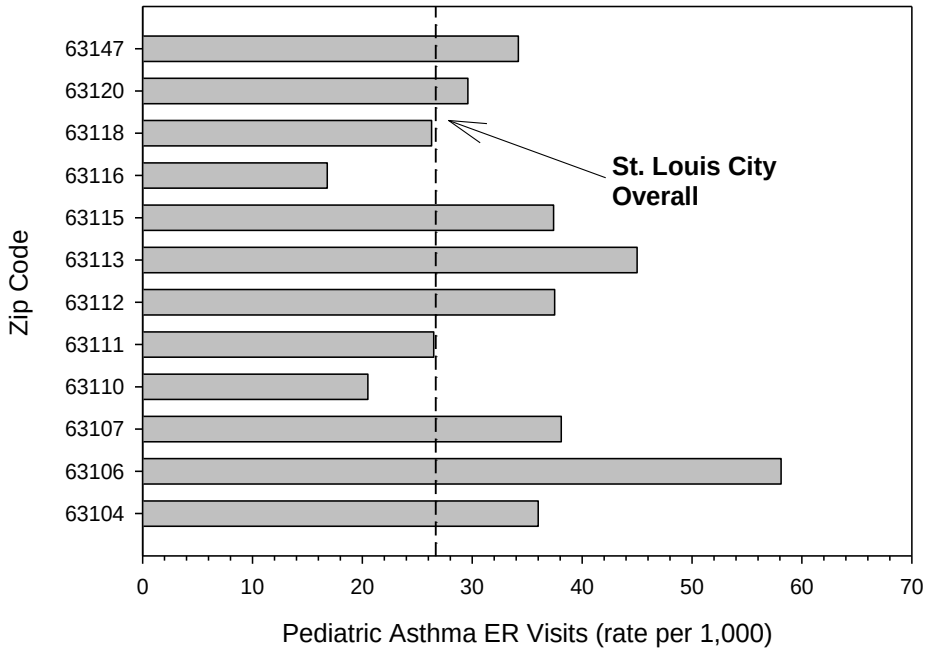


Figure 6.10 Despite Being Located Within the Same Airshed, Rates of Pediatric Asthma Emergency Room (ER) Visits Vary Significantly by Zip Code in the St. Louis City Area. 2006-2008 rates (per 1,000) of pediatric (<15 years of age) asthma emergency room (ER) visits for St. Louis City zip codes obtained from an undated Missouri DHSS Report (only includes zip codes with more than 300 asthma ER visits during 2006-2008 three year-period).

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