A TOXICOLOGIST'S PERSPECTIVE ON SO_2 HEALTH EFFECTS

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CONTENTS

FIGURES

TABLES

APPENDICES

1.0 INTRODUCTION

The purpose of this report is to provide additional detail and documentation for opinions that I expressed regarding the potential health effects associated with SO_2 at the August 27, 2015 hearing on area designations for the 2010 1-hour SO_2 NAAQS. In addition, I provide a toxicologist's perspective on statements made at the hearing by the Sierra Club and other local environmental groups about the potential health effects associated with SO_2. The comments made by these groups misconstrue, mischaracterize or simply ignore the scientific body of evidence that is currently available on health effects associated with SO_2 exposure.

A copy of my resume is provided in **Appendix A** of this report.

2.0 NATIONAL AMBIENT AIR QUALITY STANDARDS (NAAQS) ARE DESIGNED TO PROTECT HUMAN HEALTH AND WELFARE WITH A MARGIN OF SAFETY

Not all air emissions constitute air pollution. According to 10 CSR 10-6.020, the Missouri Department of Natural Resources (MDNR) defines air pollution as:

> "The presence in the ambient air of one (1) or more air contaminants in quantities, of characteristics, and of a duration which directly and approximately cause or contribute to injury to human, plant, or animal life or health, or to property or which unreasonably interfere with the enjoyment of life or use of property."

National Ambient Air Quality Standards (NAAQS) are standards set by the United States (US) Environmental Protection Agency (EPA) for specific emissions that are determined to be protective of human health and the environment *with a margin of safety* (i.e., a safety buffer).

There are two types of NAAQS—primary and secondary NAAQS. Primary NAAQS provide public *health* protection, including protecting the health of "sensitive" populations *such as asthmatics, children, and the elderly*. Secondary NAAQS provide public *welfare* protection, including protection against damage to crops, vegetation, and materials (ferrous and non-ferrous metals, zinc and other protective coatings, and inorganic building materials [e.g., concrete and limestone]) and certain textiles. Thus, evaluating whether ambient SO_2 concentrations exceed the SO_2 NAAQS is a quantitative way to answer the question of whether adverse health or welfare effects can be expected and whether ambient air concentrations represent pollution.

Each NAAQS consists of a *level* and a *form*. The "level" is the concentration of the pollutant in parts per billion (ppb). The "form" of the NAAQS defines the air quality statistic or metric used to determine whether the NAAQS is met. The level and form of the NAAQS for SO_2 are provided in **Table 1**.

TABLE 1: NATIONAL AMBIENT AIR QUALITY CRITERIA FOR SULFUR DIOXIDE (SO_2)

Sulfur Dioxide (SO_2) Standards	Averaging Time	Level ppb	Form
Primary	1-Hour	75	99th percentile of 1-hour daily maximum concentration, averaged over 3 years
Secondary	3-Hour	500	Not to be exceeded more than once per year

The Primary SO_2 NAAQS is largely based on controlled human exposure studies of SO_2-induced respiratory effects among exercising asthmatics following peak exposures (defined as 5- to 10-minute exposures to relatively high concentrations, e.g., 200 to 1,000 ppb) (EPA, 2008). Details on how the 1-hour SO_2 Primary NAAQS was established, including a discussion on how both the

level and the form were determined and why the NAAQS is highly conservative, is provided in **Appendix B** of this report.

3.0 HEALTH EFFECTS ASSOCIATED WITH SO_2 ARE PRIMARILY LIMITED TO EXACERBATION OF RESPIRATORY SYMPTOMS IN EXERCISING ASTHMATICS

Contrary to the assertions made by environmental groups, health effects associated with SO_2 are primarily limited to exacerbation of respiratory symptoms in asthmatics that are engaged in physical exercise. Assertions have also been made that secondary formation of particulate matter (PM) with a mean diameter equal to or less than 2.5 micrometers ($PM_{2.5}$) from gaseous pollutants, including SO_2, is responsible for the majority of the alleged health effects that environmental groups attribute to coal-fired power plants (CFPPs). However, these claims are entirely reliant on highly uncertain modeled health impacts that are not supported by the evidence.

This section of my report focuses on health effects associated with SO_2 and does not address potential health effects that may be associated with $PM_{2.5}$, for which secondary formation may be partially dependent on SO_2 emissions. Allegations of health effects associated with $PM_{2.5}$ formed from gaseous pollutants emitted by CFPPs are addressed in **Section 5.2**.

3.1 ONLY EFFECT THAT EPA CONCLUDED SO_2 "CAUSES" IS SHORT-TERM RESPIRATORY EFFECTS

EPA evaluated potential health effects associated with SO_2 exposure during the most recent SO_2 NAAQS review and identified only one causal relationship: short-term respiratory effects. At levels in the range of the 1-hour SO_2 NAAQS, SO_2 causes bronchoconstriction (tightening of the airways in the lungs) in asthmatics, while studies in healthy people provide little evidence of respiratory effects at concentrations more than 10 times the NAAQS. Therefore, claims made by citizen and environmental groups that SO_2 causes other health effects, such as cardiovascular effects (including stroke) and premature mortality, should be viewed with skepticism because a causal link between SO_2 exposure and these effects has not been clearly established.

With regard to cardiovascular effects, the EPA (EPA, 2008) specifically concluded that, as a whole, the evidence from animal toxicological, human clinical, and epidemiologic studies is ***"inadequate"*** to infer a causal relationship between short-term SO_2 exposure and cardiovascular effects (see p. 3-42). EPA further concluded that the evidence for a ***causal link*** between short-term SO_2 exposure and mortality at current ambient SO_2 levels is limited by potential confounding and lack of understanding regarding the interaction of SO_2 with co-pollutants (see p. 3-52). Moreover, EPA concluded that the available evidence for a causal link between long-term SO_2 exposure and any type of morbidity or mortality is inadequate (see p. 5-8).

3.1.1 Asthma Severity Is Not a Key Determinant of Responsiveness to SO_2

The response of asthmatics to SO_2 has been extensively studied in controlled human exposure studies. Evidence from studies conducted in adult asthmatics have demonstrated that asthmatics are more sensitive to the effects of SO_2 than healthy individuals (and possibly individuals with

Chronic Obstructive Pulmonary Disease or COPD). However, even amongst asthmatics, elevated ventilation (i.e., exercise) is required to provoke a respiratory response at SO_2 concentrations that are anywhere close to ambient levels.

Most SO_2 is absorbed by the nasal passages when breathing through the nose, as is common during rest and normal activity levels (i.e., resting ventilation rates). During exercise, ventilation increases and shifts from nasal to oronasal or mouth breathing. When this occurs, the pattern of SO_2 absorption switches from the upper airways to the lower airways. SO_2 must reach the lower airways to cause bronchoconstriction and trigger an asthma attack.

For ethical reasons, the most severe asthmatics are not evaluated in controlled human exposure studies on the effects of SO_2 (Linn, Avol, Peng, Shamoo, & Hackney, 1987; Linn, et al., 1988; Linn, et al., 1990). Environmental groups often argue that because subjects participating in these human exposure studies do not include the most severe asthmatics, that the thresholds observed in these studies may not represent the lowest threshold level to which more severe asthmatics may respond. However, limited studies conducted to examine the effect of asthma severity on SO_2-induced respiratory effects suggest that asthma severity does not affect the responsiveness of asthmatics to SO_2. While severe asthmatics could be more susceptible to the adverse effects of SO_2, it may be that the most severe asthmatics are less likely to experience SO_2-induced bronchoconstriction due to exercise limitations (Johns & Linn, 2011).

3.1.2 Non-Asthmatics Are Much Less Susceptible to the Respiratory Effects of SO_2

In healthy adults, SO_2-induced bronchoconstriction is generally only observed after exposures to 5,000 ppb (for periods of 10 to 60 minutes) or more. Even during exercise, studies in healthy people provide little evidence of respiratory effects at concentrations below 1,000 ppb (Amdur, Melvin, & Drinker, 1953) (Lawther, 1955) (Frank, Amdur, Worcester, & Whittenberger, 1962) (Nadel, Salem, Tamplin, & Tokiwa, 1965).

Studies in which healthy individuals and individuals with COPD at rest were exposed to SO_2 concentrations of 300 to 4,000 ppb failed to show bronchoconstrictive effects (Reichel, 1972; Weir & Bromberg, 1972; Weir & Bromberg, 1973). Another study exposed volunteers with COPD to SO_2 concentrations of 400 and 800 ppb for 1 hour, with two 15-minute periods of light exercise, neither significantly affecting lung function nor the severity of respiratory symptoms (Linn, et al., 1985). While these studies suggest that individuals with COPD are not more susceptible to the effects of SO_2, it may simply be the case that very little of the SO_2 reached the lower airways, which is necessary for bronchoconstriction.

3.2 1-HOUR PRIMARY SO_2 NAAQS IS DESIGNED TO PREVENT ASTHMA EXACERBATION

Studies of controlled human exposures are described as providing the "definitive" evidence of the relationship between short-term SO_2 exposure and respiratory symptoms (EPA, 2008, Section 5-

2). Controlled human exposure studies are studies conducted with human volunteers in a laboratory setting.

3.2.1 1-Hour SO_2 NAAQS Level Is Based on Controlled Human Exposure Studies that Evaluated Respiratory Effects in Exercising Asthmatics

Because asthmatics are more susceptible to the respiratory effects of SO_2 and respiratory effects occur more readily at elevated ventilation rates, EPA relied almost exclusively on human exposure studies conducted with asthmatics who were exposed to SO_2 while exercising in establishing the level (i.e., concentration) of the 1-hour SO_2 NAAQS.

EPA adopted an extraordinarily conservative approach in setting the most recent SO_2 NAAQS standard. Despite the conclusion of the World Health Organization (WHO) that changes in lung function observed during 5- to 10-minute exposures of 200 ppb SO_2 are similar to those seen in asthmatics with exercise alone (WHO, 2006), the fact that a single study (Linn, et al., 1983) reported statistically significant increases in respiratory symptoms below a concentration of 400 ppb, and no studies reported clinically relevant decreases in lung function below 400 ppb (Goodman, Dodge, & Bailey, 2010), EPA identified a short-term (5-minute) exposure threshold for SO_2 of 200 ppb in the most recent decision on the SO_2 NAAQS (75 CFR 35220, Jun 22, 2010).

3.2.1.1 Measurement Error and Exposure Misclassification Are Reduced in Controlled Human Exposure Studies Relative to Epidemiology Studies

Human chamber studies are powerful in assessing acute, reversible effects from short-duration exposures in humans, such as those associated with SO_2 exposure. The major advantage of human exposure studies is that the conditions of exposure are carefully controlled (i.e., air concentrations, breathing rates, exposure durations, etc.) and health outcomes can generally be measured more precisely, which reduces measurement errors and exposure misclassifications, both of which are common to epidemiology studies. A further advantage of clinical studies is the random assignment of subjects to treatment groups, which reduces both confounding[1] and selection bias[2], also common in epidemiology studies.

3.2.1.2 EPA Judged the Available Epidemiology Studies Inappropriate for Use in Its Risk and Exposure Assessment Due to Their Substantial Uncertainties

In contrast with most recent NAAQS reviews, EPA did not use the results of epidemiology studies in its quantitative risk assessment because of the substantial uncertainties associated with them. Unlike clinical studies, epidemiological studies are not conducted in a controlled environment, but rather are observational studies designed to evaluate whether ambient SO_2 levels are associated with increases in adverse health effects at the population level. Observational epidemiological

[1] A confounder is an extraneous variable in a statistical model that correlates (directly or inversely) with both the dependent variable (e.g., asthma ED visits) and the independent variable (e.g., air concentrations).

[2] Selection bias refers to the selection of individuals, groups or data for analysis in a way that proper randomization is not achieved, thereby making the sample unrepresentative of the population to be analyzed

studies attempt to determine which factors are associated with diseases, but cannot prove that a specific risk factor actually causes the disease being studied. This is because epidemiological studies cannot control for, nor can they necessarily identify all of the factors that may influence a health outcome. Therefore, they are plagued with issues of confounding. For example, if coffee drinkers are more likely to also be cigarette smokers, and a study was conducted to explore potential associations between coffee drinking and lung cancer, without taking the smoking habits of the coffee drinkers into account, smoking would be a confounder and the results may seem to show that coffee drinking increases the risk of lung cancer.

When associations are observed in epidemiology studies, the first question that should be asked is "Is it real?" Epidemiology studies test whether there is an *association* between exposure and disease, not whether exposure *causes* a disease. If there is an *association*, the exposure is called a risk factor of the disease. However, the risk factor can either be:

- A predictor (i.e., a marker or a proxy), such as employment in a specific industry or socioeconomic status; or
- A causal factor, such as cigarette smoking for lung cancer.

In addition, if a non-representative population was chosen for study, the association may have occurred by *chance*. This is why it is important to pay attention to whether results from studies (epidemiology and clinical) are statistically significant because results that are statistically significant are not likely to have occurred by *chance alone*.

Although epidemiological studies have the advantage of studying the population of interest (including sensitive individuals) at ambient pollutant levels, their utility is limited because invariably the study population is exposed to mixtures of pollutants from which health effects associated with a particular pollutant are difficult to disentangle. Sophisticated statistical models that few people adequately understand are required to separate (even partially) the effects of individual pollutants. Furthermore, relevant information is rarely collected for individuals admitted to the hospital or emergency department (ED), which precludes ruling out other factors, such as workplace exposures, pre-existing disease, or lifestyle factors as being the causative agent for the health effects observed. **Due to confounding factors and a lack of individual exposure measurements, it is widely recognized that observational epidemiology studies alone cannot be used to infer causality**. According to the *Reference Guide on Epidemiology in* the National Academy of Science's Reference Manual on Scientific Evidence (NAS, 2011):

"Epidemiology is concerned with the incidence of disease in populations and does not address the question of the cause of an individual's disease. This question, sometimes referred to as specific causation, is beyond the domain of the science of epidemiology. **Epidemiology has its limits at the point where an inference is made that the relationship between an agent and a disease is causal** (general causation) and where the magnitude of excess risk attributed to the agent has been determined; that is, epidemiology addresses whether an agent can cause a disease, not whether an agent did cause a specific plaintiff's disease."

Unfortunately, environmental advocates typically rely upon and misuse such epidemiologic studies in presenting their views and making unsupported claims about health effects.

3.2.2 Form and Level of the 1-Hour SO_2 NAAQS Are Designed to Limit 5-Minute Peak Concentrations

As discussed previously, EPA identified a short-term (5-minute) exposure threshold for SO_2 of 200 ppb, despite the fact that changes in lung function observed at this level are similar to those seen in asthmatics with exercise alone. Using data from monitors that voluntarily reported 5-minute data, EPA demonstrated that there is a high correlation between the 5-minute maximum level and the corresponding 1-hour average SO_2 concentration (section 2.5.2 of EPA, 2009) and used this information to support the establishment of a 1-hour standard that limits peak (5-minute) exposures of 200 ppb. Since the 1-hour 75 ppb NAAQS for SO_2 is designed to prevent peak exposures of 200 ppb, it represents a highly health-protective level.

Therefore, even though the SO_2 NAAQS does not have a 5-minute averaging time, the form and level were established specifically to limit 5-minute peak concentrations (section 10.5.3 and Figures 7–27 and 7–28 of EPA, 2009). EPA concluded that a concentration-based NAAQS that is averaged over a three year period better reflects the continuum of health risks of increasing SO_2 concentrations by weighting years when 1-hour daily maximum SO_2 concentrations are well above the level of the standard more heavily than those when 1-hour daily maximum SO_2 concentrations that are just above the level of the standard. Because it is designed to give greater weight to days with high levels of SO_2 in determining compliance, the form of the NAAQS is also conservative.

4.0 TRENDS IN ASTHMA RATES DO NOT CORRELATE WITH OUTDOOR AIR CONCENTRATIONS

There are many studies showing that regional differences in outdoor air concentrations do not correlate with asthma prevalence (Anderson H. , 1997; Anderson, et al., 2012). Moreover, there are also many studies that show 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 & Li, 1999; ISAAC, 1998 as cited in Gradient, 2015).

4.1 ASTHMA RATES ARE HIGHLY VARIABLE AMONG DIFFERENT COMMUNITIES WITHIN CITIES DESPITE SIMILARITIES IN OUTDOOR AIR QUALITY

In a study of Boston asthma hospitalizations, researchers (Gottlieb, Beiser, & O'Connor, 1995) observed dramatic differences in asthma hospitalizations among different communities despite similar ambient air quality. Similarly, Gupta, Zhang, Sharp, Shannon, & Weiss (2008) have shown that neither ambient $PM_{2.5}$ levels nor proximity to two CFPPs in operation at the time of the study explain the large geographic variability in childhood asthma prevalence across different Chicago neighborhoods.

Similar to what has been shown for other US cities, Gradient (2015) showed that there is also evidence that asthma statistics are highly variable between different St. Louis City zip codes despite similarities in outdoor air quality. The Gradient report can be found at http://www.americaspower.org/sites/default/files/FinalGradientReport_090115.pdf.

Figures 6.8 and 6.9 from the Gradient report, which are reproduced as **Figures 1** and **2** below, show that, despite being located within the same airshed, rates of total all-age asthma inpatient hospitalizations and ED visits vary significantly by zip code in the St. Louis City area. The Gradient results are based on 2010 data for asthma inpatient hospitalization discharges for St. Louis City obtained from the Missouri Information for Community Assessment (MICA) system available on the Missouri Department of Health and Senior Services website[3] (numbers of cases converted to rates using 2010 census data).

The examples discussed in this section support the conclusion that asthma rates do not correlate with criteria pollutant levels in outdoor air.

[3] http://health.mo.gov/data/mica/MICA/.

FIGURE 1: VARIATION IN ALL-AGE ASTHMA INPATIENT HOSPITALIZATIONS BY ZIP CODE IN THE ST. LOUIS CITY AREA

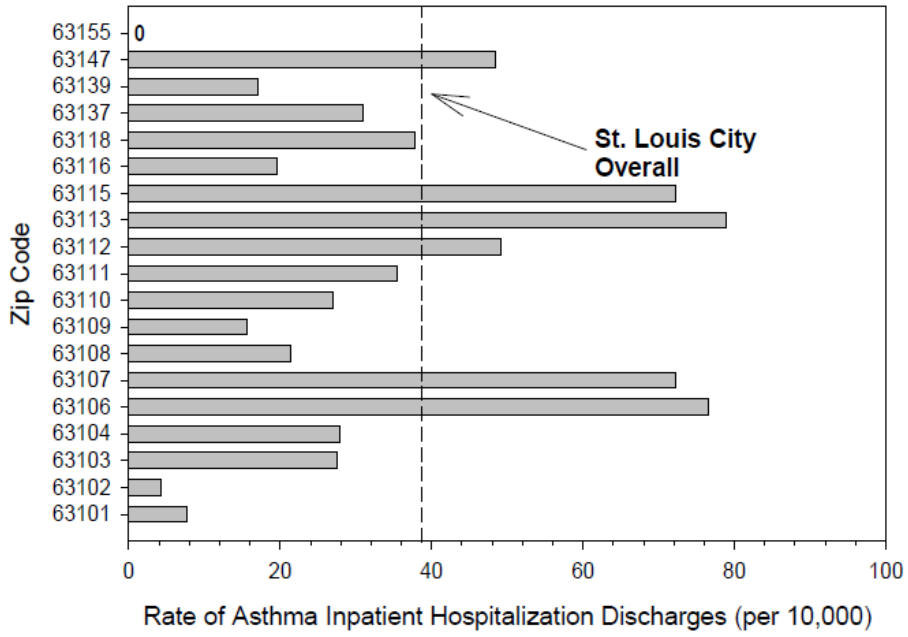
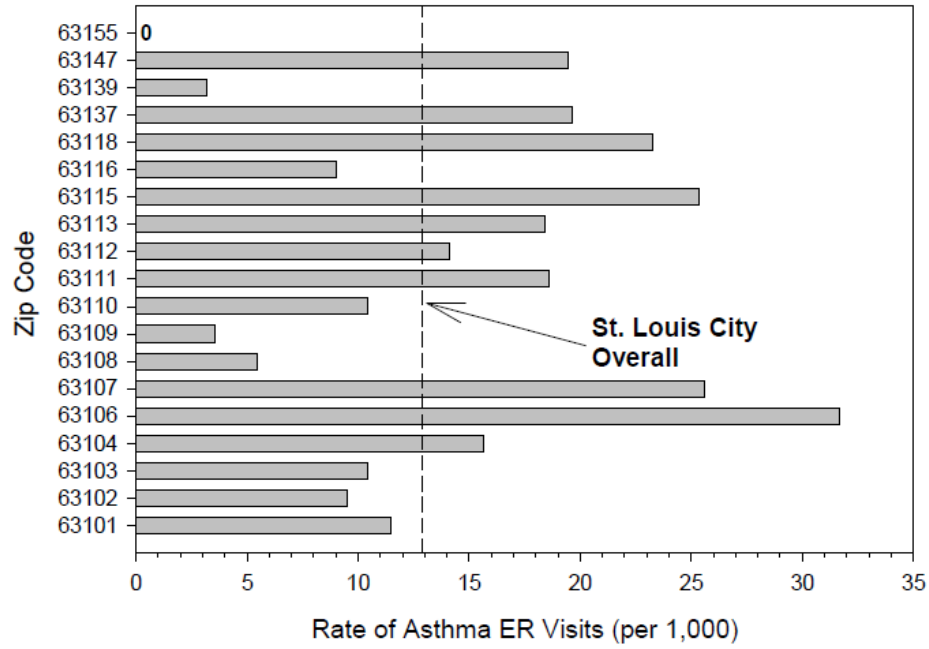


FIGURE 2: VARIATION IN ALL-AGE ASTHMA EMERGENCY DEPARTMENT VISITS BY ZIP CODE IN THE ST. LOUIS CITY AREA



Source: Gradient, 2015. A Case Study: The Public Health Consequences of Air Emissions from Coal-Fired Power Plants in the St. Louis Area

4.2 ASTHMA PREVALENCE HAS INCREASED OVER SAME TIME PERIOD THAT OUTDOOR POLLUTANT LEVELS HAVE DECREASED

Asthma prevalence has risen over the last several decades and this increase is frequently used by the Sierra Club and other environmental groups to advocate for additional reductions in outdoor pollutant levels. However, this increase in asthma has coincided with a time during which concentrations of anthropogenic air pollutants in outdoor air have been decreasing. **Figures 3** and **4** below (reproduced from Gradient, 2015) compare national-scale trends in SO_2 over the past two to three decades with national-scale trends in asthma prevalence in adults and children, respectively.

FIGURE 3: TRENDS IN AMBIENT SO_2 CONCENTRATIONS AND ADULT ASTHMA PREVALENCE

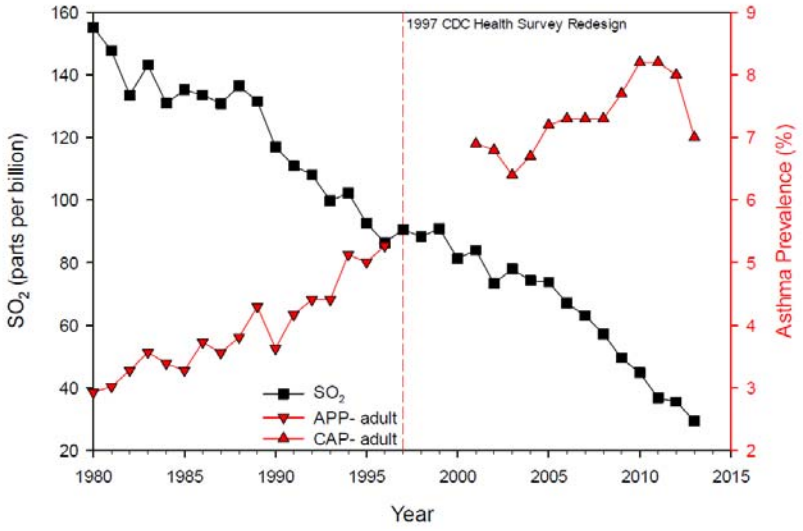
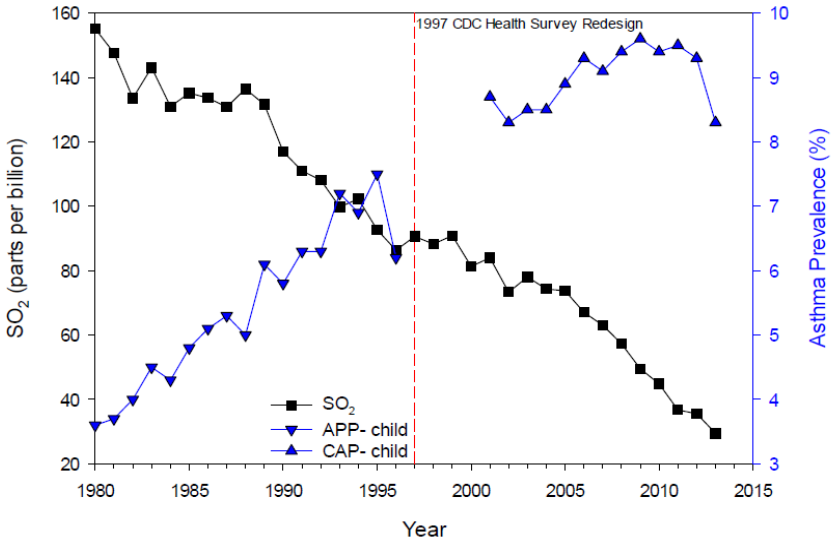


FIGURE 4: TRENDS IN AMBIENT SO_2 CONCENTRATIONS AND CHILD ASTHMA PREVALENCE



Source: Gradient, 2015. A Case Study: The Public Health Consequences of Air Emissions from Coal-Fired Power Plants in the St. Louis Area

In both figures, the black line with the boxes represents the falling SO_2 concentrations over the past 35 years. The red line with triangles represents the rising asthma prevalence in adults in the first figure and the blue line with the triangles in the second figure represents the rising asthma prevalence in children. The break in the asthma prevalence lines (red line in first graph and blue line in the second) reflects the fact that prior to 1997, the Centers for Disease Control and Prevention (CDC) collected data on asthma period prevalence (APP), which reflects the percentage of the US population with asthma in the previous 12 months. CDC redesigned its health survey in 1997, and in 2001, started collecting data on current asthma prevalence (CAP), which represents the percentage of the US population diagnosed with asthma and having asthma at the time of the survey.

As shown in the figures, SO_2 concentrations in the US have decreased significantly over the past several decades while the prevalence of asthma has increased. Although not shown here, the Gradient report (2015) shows similar trends for other criteria pollutants ($PM_{2.5}$, PM_{10}, and O_3).

4.3 FACTORS OTHER THAN OUTDOOR AIR CONCENTRATIONS ARE MORE LIKELY RESPONSIBLE FOR TRENDS IN INCREASED ASTHMA PREVALENCE

The only logical conclusion that can be drawn from the data presented in **Figures 1** through **4** is that factors other than outdoor air concentrations are triggering factors underlying the trends in increased asthma prevalence. Several possible partial explanations for the increase in asthma prevalence are explored in the paragraphs that follow.

4.3.1 Changes in Diagnostic Coding May Be Responsible for Some of the Apparent Increase in Asthma Prevalence

Changes in the diagnostic coding of asthma and survey questions in self-reporting asthma questionnaires over the last 30 years have likely altered the diagnosis of asthma cases and caused changes in prevalence and incidence statistics. The International Classification of Diseases (ICD) provided by the WHO was revised in 1978 (9^{th} revision) and 1990 (10^{th} revision) resulting in a change to the coding of asthma. In the ICD 8, a patient with "asthmatic bronchitis" would have been coded under bronchitis, while in ICD 9 this same person would be coded under asthma. One study that analyzed asthma patient records found an increase in patients with an asthma classification that had a history of smoking in the 1980s versus the 1970s. The cause of this difference was attributed to the change in classification of asthmatic bronchitis from a bronchitis heading to an asthma heading, resulting in asthmatic bronchitis patients now falling under the umbrella of asthma in the 1980s (Marcus & Braman, 2010). This change in coding may also influence the validity of epidemiology studies that look at hospital ED visits for asthma as potential indicators of an association between ambient pollutant concentrations and respiratory effects over years during which changes in the asthma definition has changed.

4.3.2 Changes in Survey Questionnaires May Be Responsible for Some of the Apparent Increase in Asthma Prevalence

A large source of asthma surveillance data is compiled by the National Center for Health Statistics of the CDC under the National Health Interview Survey (NHIS). As previously described, the NHIS questions used to evaluate asthma prevalence changed in 1997 and 2001, resulting in three separate types of questions that could impact asthma prevalence estimates from 1980 to 1996, 1997 to 2000, and 2001 to the present according to the National Heart, Lung, and Blood Institute website.[4] These changes prevent direct comparisons of reported asthma rates from 1980 to 1997 to the more recent data set from 1997 to 2001 and illustrate the potential variability in reported asthma prevalence depending on how asthma questions are phrased, and what sort of asthma information is requested (lifetime incidence versus episodes in the past 12 months, for example).

4.3.3 Changes in Health Care Access and Physician Perceptions May Be Responsible for Some of the Apparent Increase in Asthma Prevalence

The increase in asthma cases may also be partially explained by factors relating to changes in health care access and physician perceptions. The diagnosis of asthma may have become more likely than a similar diagnosis of bronchitis or COPD among patients with similar symptoms. One study looked at healthcare data from Manitoba, Canada from 1980-1990 and found a statistically significant increase in asthma diagnosis above background increases found for other diseases with similar symptoms over that time period. The study attributed some of the increase to an increase in the likelihood of asthma diagnoses (Manfreda, Becker, Wang, Roos, & Anthonisen, 1993).

4.4 ASTHMA APPEARS TO BE MORE CLOSELY LINKED TO ALLERGIC STATUS, LIFESTYLE FACTORS, AND INDOOR AIR POLLUTANTS

The reason for the surge in asthma prevalence over the past several decades is unclear, but it is well known that asthma is a complex disease with a multitude of triggers, of which air pollutants can be one. **However, scientific evidence indicates that asthma is more closely linked to allergic status, lifestyle factors, and indoor air pollutants than to outdoor air concentrations**.

According to the American Lung Association website,[5] common asthma triggers include:

- Medical Conditions
  - Colds, i.e., viral respiratory infections
  - Sinus infections
- Allergens
  - Allergens in fragrances, hairspray, and cleaning products
  - Food allergies (peanuts, shellfish)

[4] www.nhlbi.nih.gov/health/prof/lung/asthma/surveil.htm.

[5] http://www.lung.org/lung-disease/asthma/taking-control-of-asthma/reduce-asthma-triggers.html.

  - Animal fur/dander, dust mites, rodents, cockroaches, feathers
  - Over the counter medications, (aspirin or other non-steroidal anti-inflammatory drugs)
- Lifestyle Factors
  - Being overweight
  - Exercise, physical activity
  - Excitement/stress
  - Maternal stress during pregnancy period when child was in utero
- Weather, Pollen, Air Pollution
  - Exposure to cold air or sudden temperature change
  - Outdoor - pollens, grasses, flowers, pollutants
  - Indoor - mold, fungus, mildew, house dust, cigarette smoke, smoke from fireplace

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, Zeitel, & Weiss, 1992; De Palo, Mayo, & Friedman, 1994; Belanger, et al., 2003; Leaderer, et al., 2002; Teach, Crain, Quint, Hylan, & Joseph, 2006 as cited in Gradient, 2015).

Children spend much more time indoors today than they did 30 years ago. In addition to contributing to the development of asthma, exposure to various indoor air irritants can also exacerbate asthma symptoms. Cat, cockroach, and house mite dust allergens have all been causally linked to exacerbation of asthma symptoms in sensitive individuals, and environmental tobacco smoke exposure has also been causally linked to exacerbation of asthma symptoms in young children (IOM, 2000).

5.0 CLAIMS THAT COMMUNITIES NEAR THE LABADIE PLANT BEAR A DISPROPORTIONATE SHARE OF THE BURDEN OF DISEASE ARE UNFOUNDED

Despite claims by environmental and citizen groups that areas with the highest concentration of CFPPs bear a disproportionate share of the burden of disease, the facts do not support this claim. Such claims are based on overly simplistic formulas that distort the underlying scientific principles.

To support assertions of illness and mortality, environmental and citizen groups ignore the scientific research studies cited in this report and instead rely on reports from interest groups such as the Clean Air Task Force's *Toll From Coal* (CATF, 2010) and the Environmental Integrity Project's (EIP) *Net Loss: Comparing the Cost of Pollution vs. the Value of Electricity from 51 Coal-Fired Plants*, along with health projections placed on websites (CATF, 2014) of such groups. These organizations support their advocacy positions by *estimating* impacts of CFPP emissions on ambient air quality and then applying results from epidemiology studies to *approximate* changes in the incidence of adverse health outcomes, such as hospital admissions, asthma attacks, and premature deaths. Unfortunately, there is currently neither an accepted gold standard nor even a simple, reliable, and evaluated method for carrying out such assessments. As a result, the hypothetical health impacts estimated using models such as those employed by CATF (2010 and 2014) and EIP (2012) are highly uncertain and easily manipulated through choice of input parameters.

The gross oversimplifications and highly uncertain estimates made by these groups are discussed in **Section 5.2** and in much more detail in **Appendix C**.

5.1 *REAL* DATA DEMONSTRATE THAT THE LABADIE AREA IS IN COMPLIANCE WITH THE NAAQS AND THAT ASTHMA RATES ARE NOT HIGHER NEAR COAL-FIRED POWER PLANTS (CFPPS)

The areas around St. Louis have air quality that is in compliance with the health-protective $PM_{2.5}$ NAAQS. The same is true for the areas immediately surrounding Labadie and the other Missouri CFPPs[6]. Therefore, people living in these communities are not at risk for adverse health effects potentially associated with particulates, regardless of their origin.

Fortunately, the Missouri Department of Health and Senior Services (MDHSS) provides age-adjusted asthma ED visits and hospitalizations by county for the entire state (MDHSS, 2010), which shows that asthma rates are not correlated with proximity to CFPPs. The availability of county-specific asthma rates from the MDHSS allows ***real*** data on asthma ED visits and hospitalizations for counties in which CFPPs are located and/or that are being considered for designation to be compared to asthma rates for the state of Missouri and the nation as whole.

[6] http://www.epa.gov/airquality/greenbk/rncty.html.

The MDNR is considering designating portions of Franklin and St. Charles counties as either unclassifiable or non-attainment based on air dispersion modeling. **Table 2** shows asthma ED visits per 1,000 people in the population and asthma hospitalizations (inpatient) per population of 10,000 from the *Missouri Asthma Surveillance Report* (MDHSS, 2010) for Franklin and St. Charles counties and the state of Missouri as whole, and US Data on asthma rates from the CDC (CDC, 2012).

TABLE 2: ASTHMA RATES IN FRANKLIN AND ST. CHARLES COUNTIES

Geographic Area	2008 Asthma Trends		Reference
	Emergency Department Visits	Hospitalizations	
	Rate Per 1,000	Rate per 10,000	
Franklin County, MO	3.8	9.4	MDHSS, 2010
St. Charles County, MO	3.2	8.3	
State of Missouri	5.2	13.8	
United States	6.5	14.4	CDC, 2012

Sources: MDHSS, 2010. Missouri Asthma Surveillance Report: The Burden of Asthma in Missouri. http://health.mo.gov/living/healthcondiseases/chronic/asthma/pdf/burdenreport.pdf and CDC, 2012. National Surveillance of Asthma: United States, 2001–2010. http://www.cdc.gov/nchs/data/series/sr_03/sr03_035.pdf.

The first column shows the geographical area. The second column shows the age-adjusted ED visit rates per 1,000 people in the population and the third column shows age-adjusted asthma hospitalization rates per 10,000 people in the population.

As shown in **Table 2**, the age-adjusted ED visit rate for the entire state was 5.2 per 1,000 people in the Missouri population. The age-adjusted asthma hospitalization rate for all Missouri residents was 13.8 per 10,000 in the population. The Missouri asthma ED visit rate and hospitalization rate are both lower than the national average rates, which are shown in the bottom line of the table as 6.5 per 1,000 for asthma ED visits and 14.4 per 10,000 for asthma hospitalizations.

In contrast, the asthma ED visit and hospitalization rates in Franklin and St. Charles counties are well below the rates for the state of Missouri as a whole and the nation. Although not shown here, similar results are reported for other counties in which CFPPs are located. Asthma incidents are higher within metropolitan areas where other risk factors (e.g., housing conditions, inadequate access to health care etc.) could influence asthma rates. Therefore, it is clear from the Missouri asthma data that asthma ED visit and hospitalization rates simply do not correlate with proximity to power plants.

5.2 HIGHLY UNCERTAIN HEALTH IMPACT ESTIMATES MADE IN CLEAN AIR TASK FORCE (CATF) AND ENVIRONMENTAL INTEGRITY PROJECT (EIP) REPORTS ARE UNRELIABLE

The estimation of health impacts made by the CATF and EIP require multiple models, numerous model inputs, and a variety of model assumptions, each of which has many associated uncertainties. Notably, the underlying model used by these groups, the Climatological Regional Dispersion Model (CRDM), uses outdated algorithms and assumptions that are not in line with state-of-practice dispersion modeling approaches (both short-range and long-range transport and dispersion) and, according to EPA (EPA, 2015) are only capable of providing "crude estimates" of air quality changes due to emissions. Perceived determinants of health are then employed in the form of concentration-response functions from epidemiology studies that may not reflect causal relationships to *estimate* changes in the incidence of adverse health outcomes. The models predict the largest impacts from the secondary formation of $PM_{2.5}$ from gaseous pollutants (SO_2, nitrogen oxides, volatile organic compounds [VOCs], and ammonia), which is *estimated* and not only highly uncertain, but also likely substantially overestimated (Gradient, 2015).

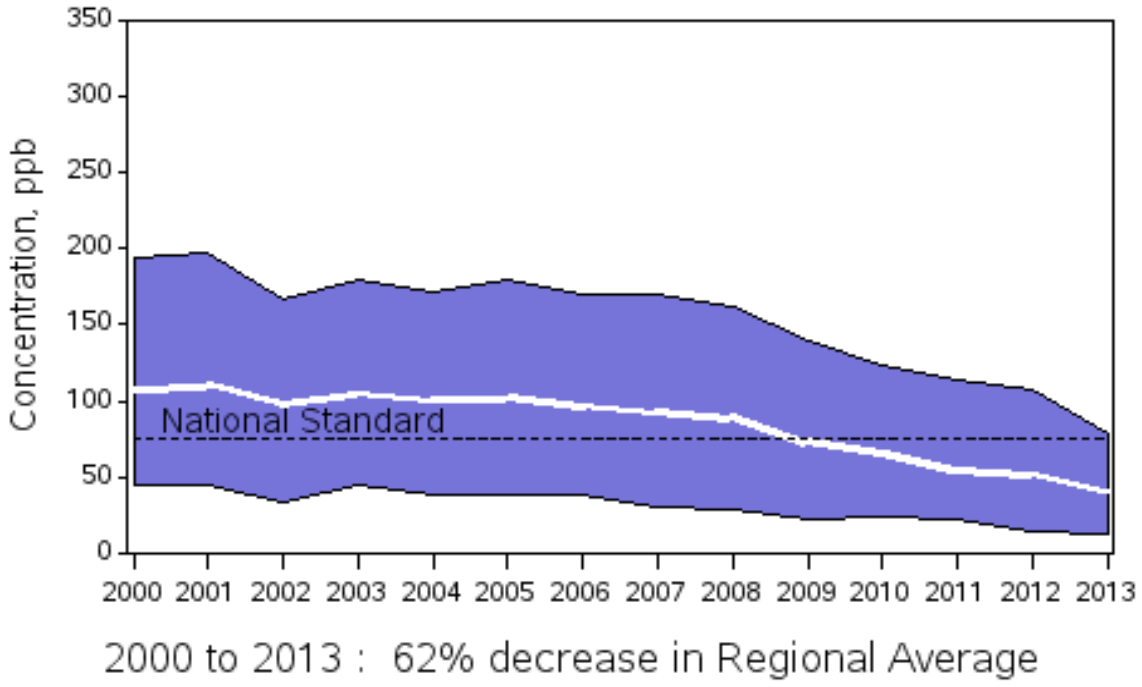
These models estimate huge impacts in terms of hypothetical lives lost or hospitalizations from incremental additions of $PM_{2.5}$ from CFPPs because large populations that are located long distances from the plants modeled are assumed to be exposed to $PM_{2.5}$ in a manner that is highly unrealistic. The majority of the impacts from such models are estimated at locations distant from the plants modeled, a fact that is rarely appreciated by citizens that live near the modeled plants and whose fears these groups exploit with their unsubstantiated claims about health effects attributable to CFPPs.

More technical detail on the issues associated with the CATF and EIP models, many of which have been identified by others (Gradient, 2015; Abt, 2010) are discussed in more detail in **Appendix C**.

6.0 CRITERIA POLLUTANT CONCENTRATIONS HAVE DECREASED DRAMATICALLY IN THE ST. LOUIS REGION

Consistent with the drop in SO_2 levels that have occurred across the US, the Central region (Missouri, Illinois, Indiana, Ohio, West Virginia, Kentucky, and Tennessee) saw a 62% decrease in ambient SO_2 levels between 2000 and 2013, as shown in **Figure 5** below.

FIGURE 5: ANNUAL 99TH PERCENTILE DAILY MAXIMUM 1-HOUR AVERAGE SO_2 CONCENTRATION



Source: EPA Sulfur Dioxide Air Trends. http://www.epa.gov/airtrends/sulfur.html.

The blue band shows the distribution of SO_2 levels among the trend sites, displaying the middle 80%. The white line represents the average among all the trend sites. Ninety percent of sites have concentrations below the top line, while 10% of sites have concentrations below the bottom line.

According to the EPA Air Trends website (EPA, 2015), decreases in Central regional averages have been observed for other criteria pollutants between 2000 and 2013, as summarized below:

- Annual $PM_{2.5}$ – 38% decrease
- 24-hour PM_{10} – 33% decrease
- 1-hour NO_2 – 22% decrease
- 8-hour ozone – 21% decrease

6.1 ST. LOUIS CORE-BASED STATISTICAL AREA

Air monitoring data from all of the central-site ambient monitors in the St. Louis Core Based Statistical Area (CBSA) were compiled and summarized in a case study of the air quality impacts of Ameren's four St. Louis-area CFPPs (Gradient, 2015). 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). According to the case study, ***the highest 2011-2013 1-hour SO_2 design value for all monitors in the St. Louis CBSA,* excluding Herculaneum, was approximately 50 ppb, which is well below the 75 ppb NAAQS.** Herculaneum was excluded because the Doe Run smelter, which shut down at the end of 2013, was a major source of SO_2 in the area and since its closure, SO_2 levels have fallen precipitously (2014 99^{th} percentile 1-hour SO_2 concentration was 18 ppb). Therefore, a 2013-2015 design value for monitors in the St. Louis CBSA including Herculaneum (using unofficial data for the Herculaneum monitor through April 13, 2015 available on the US AirData website and assuming that concentrations will remain similar for the remainder of 2015) was estimated at 60 ppb, which is also well below the current NAAQS. Therefore, the air concentrations monitored in the St. Louis CBSA are below levels demonstrated to cause respiratory effects in healthy individuals and asthmatics.

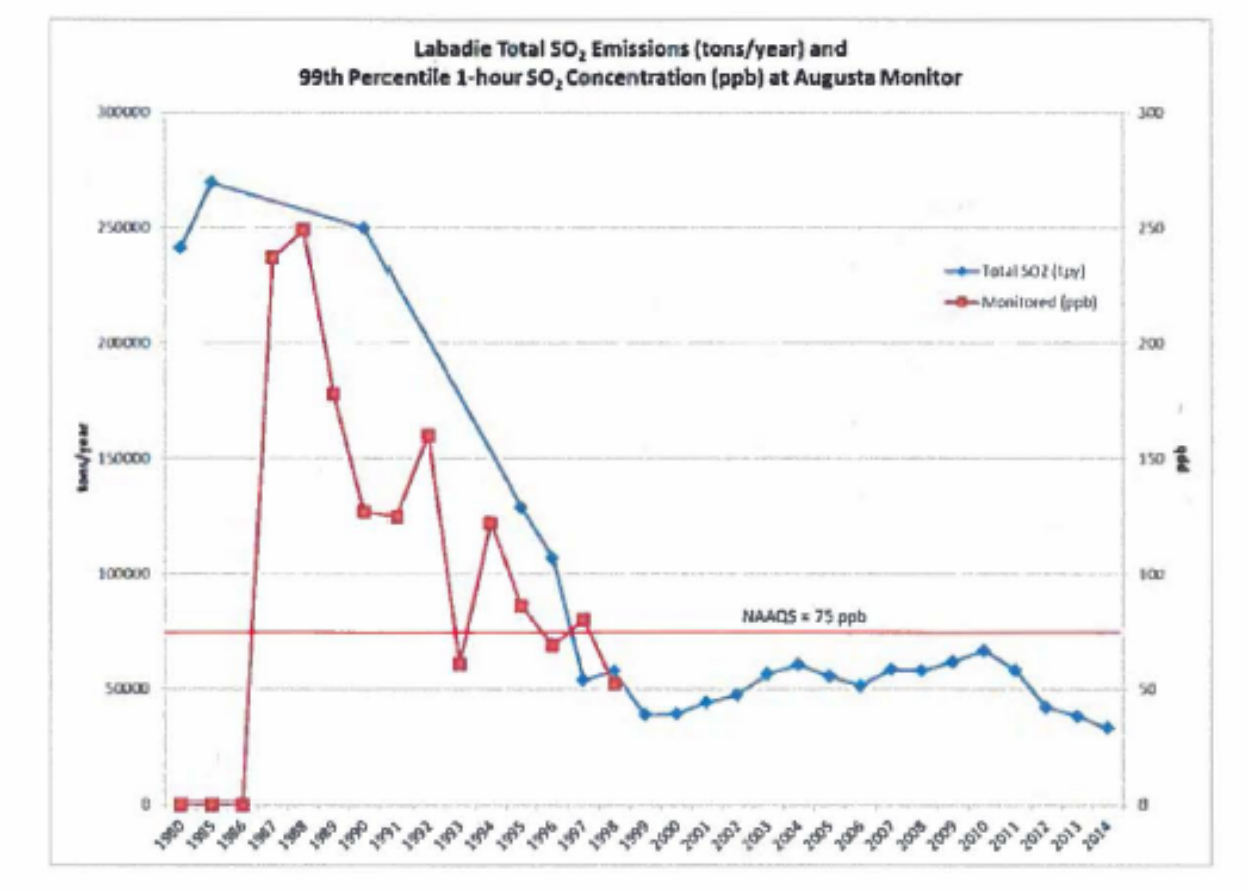
As shown in the Gradient (2015) report, 24-hour and annual concentrations of $PM_{2.5}$ (Figures 1.2 and 1.3 of Gradient report) and hourly and annual concentrations of NO_2 (Figures 1.6 and 1.7 of Gradient report) concentrations are also below their respective NAAQS in the St. Louis CBSA.

6.2 FRANKLIN COUNTY

SO_2 monitoring data in the vicinity of Ameren's Labadie plant in Franklin County are available for two different time periods: 1) current air monitoring initiated in April of 2015; and 2) previous multiple-year monitoring conducted by MDNR from 1987 to 1998. During the last few years of the MDNR monitoring (1995-1998), Plant emissions were significantly reduced as a result of the switch to low-sulfur coal obtained in response to the Clean Air Act's Acid Rain Phase 1 requirements (AECOM, 2015).

According to the *Analysis of SO_2 NAAQS Compliance for the Labadie Energy Center* conducted by AECOM (AECOM, 2015), emissions at the Labadie Generating Station decreased by about 75% from the time that the monitoring began in 1987 to when it ended in 1998, coinciding with the switch from higher sulfur coal to Powder River Basin (PBR) coal. As shown in **Figure 6** below (Figure 3 of the AECOM report), the 99th percentile 1-hour SO_2 monitored values also decreased in a similar manner during this period.

FIGURE 6: TRENDS IN SO_2 EMISSIONS AND SO_2 MONITORING FOR LABADIE ENERGY STATION



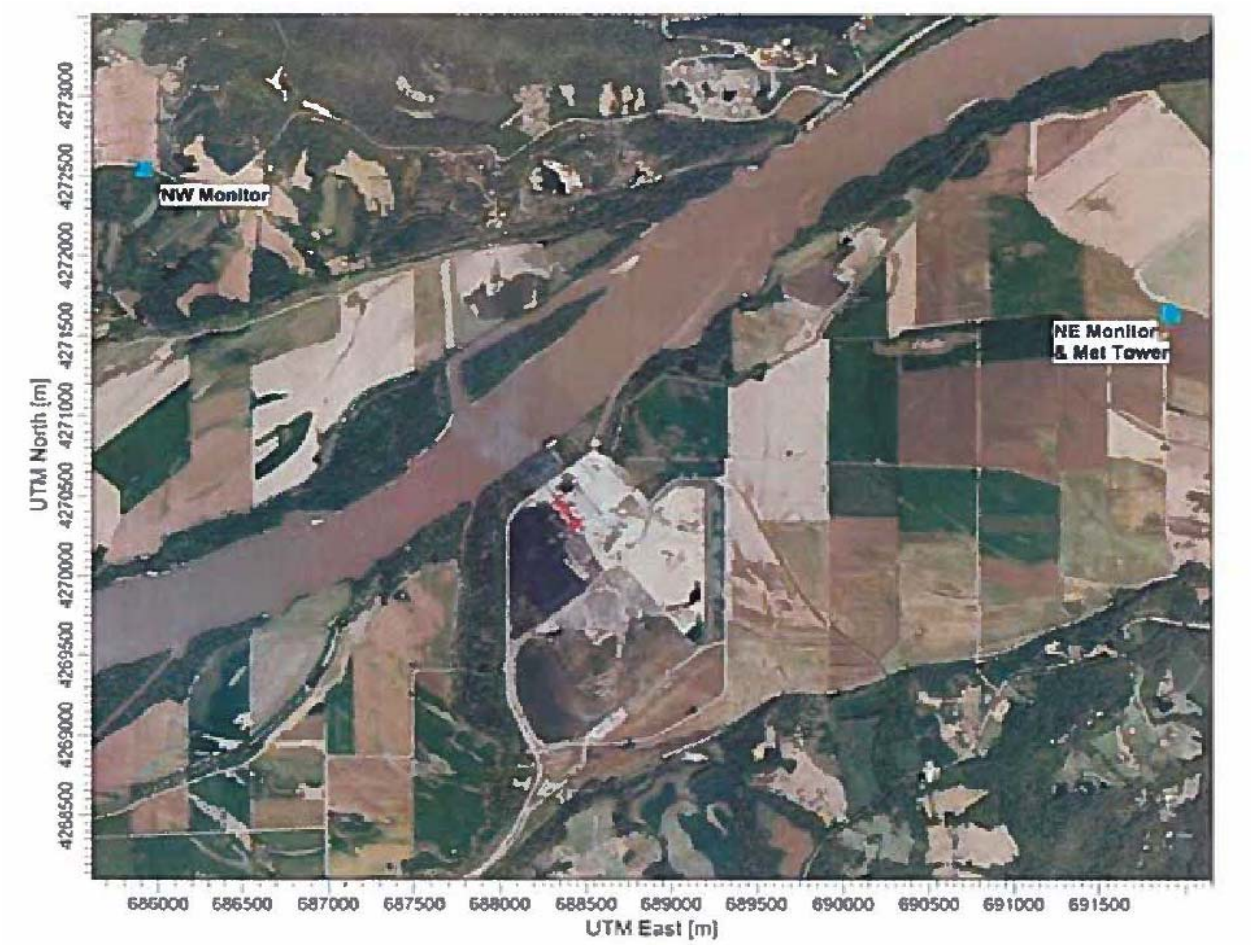
Source: AECOM, 2015. *SO_2 NAAQS Compliance for the Labadie Energy Center*.

According to the AECOM report (AECOM, 2015), the former Augusta MDNR SO_2 monitor, which was about 2 km from the Plant, was at a distance consistent with peak impacts measured near similar facilities in past field studies (Liu & Moore, 1984). In addition, it was sited in a direction with frequent winds based on Spirit of St. Louis airport wind rose (see Figure 4 of AECOM report). Therefore, the most recent data from the former MDNR Augusta monitor location, which was collected after the switch to low sulfur coal, are most relevant to current air quality.

During the last 36 months of monitoring (September 1995 - August 1998) at the Augusta monitor, a 99th percentile peak daily 1-hour maximum concentration (the "design concentration") of 69 ppb was measured. This is comfortably below the SO_2 NAAQS of 75 ppb.

Ameren initiated a new SO_2 monitoring program in 2015 to evaluate the air quality impact of the Labadie Energy Center. The monitoring plan was approved by the MDNR. Two monitors, corresponding to distances and directions expected to be in peak impact locations became operational in April (2015). As shown in **Figure 7**, one monitor (Ameren Northwest) is located across the river at the approximate location of the former MDNR Augusta monitor. The other monitor is located slightly north of the plant and approximately three kilometers to the east.

FIGURE 7: CURRENT SO_2 MONITORS IN THE VICINITY OF LABADIE GENERATING STATION



Source: Source: AECOM, 2015. *SO_2 NAAQS Compliance for the Labadie Energy Center*.

Based on the available data (about 3 months), peak (maximum) measured hourly SO_2 concentrations at the two sites were 38 ppb at the Northwest monitor and 21 ppb at the Northeast monitor. Both concentrations are well below the SO_2 NAAQS of 75 ppb.[7]

[7] For consistency with the presentation given, monitoring data collected prior to the August 27, 2015 public hearing was used for this report. On August 27th, a SO_2 concentration of 51 ppb was recorded at the Northeast monitor. This level is well below the 1-hour SO_2 NAAQS and does not change any of the opinions expressed at the hearing or in this report.

7.0 CONCLUSION

Available monitoring data indicate that the area surrounding the Labadie plant is currently in compliance with the primary SO_2 NAAQS, which provides public *health* protection, including protecting the health of "sensitive" populations *such as asthmatics, children, and the elderly*, and has been since at least 1998. In addition, the available monitoring data also indicate that the area is in compliance with the secondary NAAQS, which provides public *welfare* protection, including protection against damage to crops, vegetation, and materials. Therefore, there is no reason to fear that emissions from the Labadie plant have adversely affected air or land in the immediate vicinity of the Plant or beyond since the SO_2 impacts from the Plant are clearly localized.

8.0 RESPONSES TO SELECT COMMENTS MADE DURING THE AUGUST 27, 2015 MDNR PUBLIC HEARING

Several representatives of environmental groups at the August 27th, 2015 MDNR Public Hearing on Proposed Options for Area Boundary Recommendations for the 2010 1-Hour Sulfur Dioxide Standard: July 2016 Designations made allegations about health effects attributable to emissions from the Labadie Energy Center, the adequacy of the monitoring near the Labadie Energy Center, and the legitimacy of an "Unclassifiable Designation" for portions of Franklin and St. Charles Counties.

In the responses provided below, I have attempt to correct misrepresentations of the emissions from the Labadie Energy Center and mischaracterizations of the potential for health effects associated with emissions from the Plant by citing to *real* data to show that the claims made are not supported by the evidence. The Commission is urged not to allow unsubstantiated claims from environmental activists to substitute for credible evidence and data.

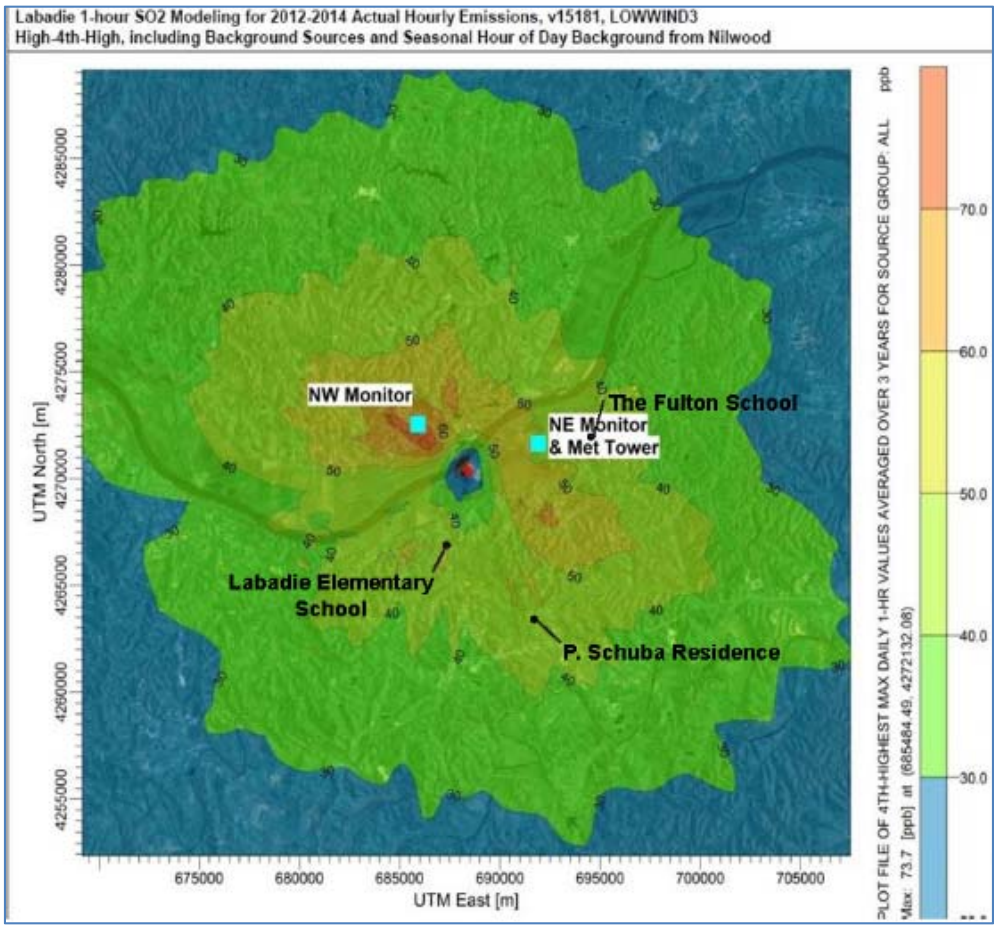
8.1 COMMENTS RELATING TO MONITOR PLACEMENT AND HISTORICAL EMISSIONS

Sierra Club contends that the former MDNR Augusta monitor showed exceedances of the current SO_2 NAAQS for most of the years that it was operated and that MDNR failed to share that in its discussion of options.

Response: In accordance with the form of the NAAQS, the 99th percentile 1-hour SO_2 concentration, a near maximum value, was calculated using data from the former MDNR Augusta monitor during the last 3 years of operation (between September 1995 and August 1998). The 99th percentile 1-hour SO_2 concentration during this period was 69 ppb, which is below the current NAAQS. As discussed in **Section 6.2**, SO_2 emissions were significantly reduced during this period as a result of Labadie's switch to low-sulfur coal in response to the Clean Air Act's Acid Rain Phase 1 requirements. Only those SO_2 levels monitored after the switch to low-sulfur coal are relevant to current air quality conditions. It is ludicrous to suggest that SO_2 levels monitored before Labadie switched to low-sulfur coal is in any way representative of current conditions and, therefore, the MDNR was correct in focusing its discussion on SO_2 levels monitored during the last period of monitoring for the Augusta monitor.

Patricia Schuba, a resident of Franklin County who lives near the Labadie Energy Center, made comments on behalf of the Labadie Environmental Organization (LEO). Ms. Schuba contends that in living within "the impact zone" of the Labadie Energy Center, she has been exposed to high levels of SO_2, PM, and airborne mercury.

Response: The location of Ms. Schuba's residence as well as nearby schools have been plotted onto a map of SO_2 concentration isopleths and depicted below. As shown in the figure, the air dispersion modeling results indicate that 1-hour concentrations of SO_2 at these locations are expected to be in the 40-50 ppb range, well below the health-protective SO_2 NAAQS of 75 ppb.



Ms. Schuba also asserted unsubstantiated claims that emissions from the Labadie plant are responsible for the loss of 140 – 210 lives per year, citing to the EIP (2012) report for the proposition that the Plant is killing people.

Response: Claims regarding mortality due to power plant emissions are based on health impact estimates made by the CATF (CATF, 2014) and EIP (EIP, 2012). I address the deficiencies of such analyses in **Appendix C**.

Ms. Schuba also opined that SO_2 is not the only contaminant of concern from a health perspective.

Response: SO_2 can react with other pollutants to form $PM_{2.5}$ in the atmosphere. However, despite claims by the CATF and EIP that the great majority of the impact of power plants is due to $PM_{2.5}$ formation from gaseous pollutants, the increment of secondary $PM_{2.5}$ theoretically contributed by CFPPs is small by comparison to $PM_{2.5}$ exposures that we all experience from everyday sources (primarily indoors) such as cooking, burning candles, commuting via bus or subway, or walking vigorously (Gradient, 2015; Levy, Dumyahn, & Spengler, 2002; Levy, Spengler, Hlinka, Sullivan, & Moon, 2002).

Given the many sources of $PM_{2.5}$, including natural sources in the environment, it can be concluded that direct emissions of $PM_{2.5}$ from CFPPs and the formation of $PM_{2.5}$ from other gaseous pollutants emitted from CFPPs contribute a miniscule portion of our everyday $PM_{2.5}$ exposures.

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APPENDIX A

RESUME

LUCY H. FRAISER, PH.D., DABT
Principal

AREAS OF EXPERTISE

- Toxicological Evaluations
- Risk Assessments
- Risk Communication
- Litigation Support
- Agency Negotiations
- Development of Innovative Risk-Based Approaches
- Exposure Modeling

EDUCATION

Ph.D., Toxicology, *University of Texas at Austin, 1992*

B.A. Psychology, *University of Texas at Austin, 1985*

REGISTRATIONS/AFFILIATIONS/CERTIFICATIONS

Diplomate of the American Board of Toxicology

American College of Toxicology

National Society of Toxicology

Lone Star Society of Toxicology

National Member, Air & Waste Management Association

Dr. Lucy Fraiser is a board certified toxicologist with over 24 years of experience in the areas of exposure and risk assessment, health effects and toxicology evaluations, development of quantitative toxicity criteria, development of risk-based air quality guidelines and soil cleanup criteria, and risk communication. While Dr. Fraiser works with all environmental media, she specializes in air quality health evaluations, including assessment of whether criteria pollutant emissions cause or contribute to a condition of air pollution and determination of the likelihood that air toxics will adversely impact health or welfare.

Dr. Fraiser has worked in both the public and private sectors over the last 24 years. She has conducted and managed multi-pathway exposure and human health risk assessments for a wide variety of environmental pollutants and sources. Dr. Fraiser has, on many occasions, examined the scientific foundation on which exposure assumptions and toxicity criteria are based on behalf of private and public sector clients and trade organizations. Her leading work on these issues has resulted in corrections to regulatory guidance and risk-based criteria on a number of occasions. She has conducted hundreds of exposure assessments for chemicals used in pharmaceutical laboratories and industrial processes, chemicals applied to control pests and unwanted vegetation, and chemicals released as unwanted by-products of chemical and product manufacturing, combustion of fossil and waste-derived fuels, generation of electricity, petroleum refining, smelting, rock crushing, and activities at military installations.

Litigation Experience

Dr. Fraiser has been qualified as an expert, deposed, and has provided expert testimony in contested case hearings, criminal case hearings, Federal Civil suits, and toxic tort litigation on numerous occasions. She has testified before the Texas State Legislature, in public meetings, and before numerous state regulatory agencies on behalf of commercial clients. Dr. Fraiser also conducted a televised press conference on behalf of a state and a national trade organization regarding mercury emissions from power plants.

Dr. Fraiser recently provided critical expert testimony in a high-profile toxic tort case involving a flaring event at a multi-national petrochemical company that resulted in a jury verdict for the defense. She also recently provided critical testimony in a citizen suit against a Texas energy company in which a judge from the Western District of Texas ruled from the bench that there were no violations of the Clean Air Act and later ordered the Plaintiff to pay $6.4 million in defense attorneys' fees. Dr. Fraiser also recently provided critical expert testimony in another citizen suit against a Texas petrochemical company involving excess air emission and maintenance, startup, and shutdown events. The federal cases

involved alleged violations of opacity standards, National Ambient Air Quality Standards, and in the case of the petrochemical plant, screening levels for compounds considered to be hazardous air pollutants.

She has also provided testimony on potential risks associated with permitting of rock crushers (silica, limestone, PM10/2.5), a concrete batch plant (silica, PM10/2.5), hazardous waste combustion units (polycyclic aromatic hydrocarbons, polychlorinated biphenyls (PCBs), dioxins), and a copper smelter (PM10/2.5, NO2, SO2, sulfuric acid, arsenic, lead, and cadmium). Dr. Fraiser has also developed opinions in cases that did not go to hearing regarding the likelihood that exposure to H2S/SO2 from a from a Sulfur Recovery Unit release was sufficient to cause known health effects, the potential for health effects associated with relatively short-term exposure to benzene concentrations in drinking water above the Maximum Contaminant Level, and potential risks associated with lead and total petroleum hydrocarbon (TPH) levels detected in street sweepings.

Regulatory Experience

As a Senior Toxicologist with the TCEQ, Dr. Fraiser conducted and managed risk assessments for incinerators and industrial boilers seeking permits to burn hazardous waste, provided support to the US Environmental Protection Agency (EPA) as they formulated national policies related to combustion risk assessment, provided critical input into the development of protective concentrations under the TRRP, served as an external peer reviewer for risk assessment guidance documents developed by EPA Region 6 and adopted as national guidance, and represented the Agency on EPA workgroups and in contested case hearings.

Dr. Fraiser recently provided comments to EPA on behalf of commercial clients questioning the extent to which health studies support the need for a tighter ozone NAAQS. She also provided comments on the Boiler MACT Health-Based Emissions Limitations on behalf of a trade organization. In the past, she has developed technical comments on EPA Risk Assessment Protocols for Hazardous Waste Combustion Facilities on behalf of the Louisiana Chemical Association and the Cement Kiln Recycling Coalition, and completed formal technical comments on behalf of a power generation client on a risk-based program intended to significantly reduce levels of toxic air contaminants in Kentucky.

Air Quality Health Impact Evaluations

Dr. Fraiser was the health risk assessment advisor for a study recently completed on behalf of the Electric Power Research Institute (EPRI) that evaluated the potential health risk from emissions of coal fired power plants throughout the U.S. She recently served as project manager responsible for multi-pathway risk assessment updates for a specialty chemical company to support permitting activity that reflected the installation of new sulfur dioxide (SO2) abatement equipment, served as the risk assessment team lead for a vapor intrusion evaluation using crawl-space soil vapor and ambient air samples collected beneath and near a house in the vicinity of a crude oil release, and performed a health risk assessment using indoor and ambient air samples from a manufacturing facility.

Dr. Fraiser has conducted or served as task leader on more than two dozen human health risk assessments conducted in support of Resource Conservation Recovery Act (RCRA) Part B permit applications for hazardous waste combustion units at chemical plants, waste management facilities, army depots, and cement kilns.

Risk-Based Corrective Action and Risk Assessment

Dr. Fraiser has conducted and/or served as task leader for over 75 human health risk assessments and/or risk-based corrective action (RBCA) evaluations in support of RCRA closures or under the Comprehensive Environmental Response, Compensation, and Liability Act for both commercial companies and government clients.

Dr. Fraiser has substantial experience performing risk evaluations under the Texas Risk Reduction Rule and the Texas Risk Reduction Program (TRRP), as well as other state RBCA programs. She has completed and received Texas Commission on Environmental Quality (TCEQ) approval for several Affected Property Assessment Reports and has provided support on the successful completion of several Response Action Completion Reports.

Dr. Fraiser recently completed a multi-media human health risk assessment for high school at which placement of fill material to build up the area for sports fields resulted in PCB contamination. She also recently completed a toxicity assessment and fish cooking loss study for dioxins and PCBs for a contaminated river segment in the northeast.

Toxicological Evaluations and Risk-Based Regulatory Criteria Development

Dr. Fraiser has developed numerous health-based criteria for compounds lacking published values using toxicity studies, structure activity relationships, and her knowledge of pharmacokinetics. She has developed risk-based regulatory criteria including emergency response planning guidelines, inhalation reference concentrations, water quality criteria, and acceptable ambient air levels, including Effects Screening Levels (ESLs), for a number of compounds. Based on her understanding of the human health underpinnings of federal regulations and state corrective action and air quality guidelines, Dr. Fraiser has assisted many clients wishing to challenge health-based criteria during public comment periods and in identifying adjustments to existing criteria.

Publications, Presentations and Training Courses

Fraiser L. EPA May Go Beyond Law and Science in Setting NAAQS. Natural Gas & Electricity, 30(3):1-8. October, 2014.

Fraiser L., and Karen Olson. Ozone NAAQS – Where is it Headed? Texas Association of Business, Austin TX. July 24, 2014.

Fraiser L. Ozone NAAQS – Where is it Headed? Houston Regional Monitoring Association, Houston, TX. July 9, 2014.

Fraiser L., and Davis B. Ozone NAAQS – Where is it Headed? Clean Air Force of Central Texas, Austin TX. April 24, 2014.

Fraiser L., and Karen Olson. Ozone NAAQS – Where is it Headed? Winstead PC, Austin TX. May 27, 2014.

Fraiser L., and Sullivan, T. Ozone NAAQS – Where is it Headed? Texas Pipeline Association, Austin TX. April 9, 2014.

Fraiser, L. Recent Reductions in NAAQS – Good Science or Perpetuation of Dogma on Health Consequences of Low-Level Air Pollutants? Energy Utility Environment Conference, Phoenix AZ. February 3 -5, 2014.

Fraiser, L.H. and Bradley, L.J.N. Key Decisions in Establishing National Ambient Air Quality Standards. 52nd Annual Meeting of the Society of Toxicology. San Antonio, Texas. March 10 – 14, 2013.

Fraiser, L.H. Health Basis for EPA's 1-Hr SO2 NAAQS. Alamo Chapter AWMA Meeting, January 10, 2013.

Ruffle, B., Fraiser, L., Kaczmar, S., Schew, W. Update on Cooking Loss Factors for PCDD/PCDFs, PCBs and Chlorinated Pesticides. Passaic River Symposium V. Passaic River Institute of Montclair State University. October 19, 2012.

Fraiser, L.H. and Vosnakis, K.A.S. Evolution of PCB Regulations and Toxicity Assessment: Impact on Environmental Management. 27th Annual International Conference on Soils, Sediments, Water and Energy, Amherst, Massachusetts. October 17 – 19, 2011.

Fraiser, L. Toxicology & Risk Assessment in the News: Recent EPA Proposals with Broad Implications. Invited Presented at the Gulf Coast Air & Waste Management Association Meeting. Houston, Texas. June 08, 2010.

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Fraiser, L.H. Trends in International Risk-Based Screening Levels (RBSLs). Society of Toxicology and Chemistry, New Orleans, Louisiana. November 19 – 23, 2009.

Fraiser, L.H. Risk Assessment: How it Can Inform Site Closure Decisions. Invited Short Course presented to the Department of Environment Malaysia, Kuala Lumpur. March 4 – 5, 2009.

Fraiser, L.H. Incinerator Risk Assessment: Principles and Practices, Hong Kong. Regional Conference on Sustainable Waste Management in Carbon-Conscious Cities. December 2008.

Site-Specific Risk Assessments, RCRA Omnibus Provision and Combining Risk Burns and Comprehensive Performance Tests. MACT EEE EPA Training Workshop, Dallas, TX. November 3 – 8, 2008.

Fraiser, L.H. Involvement of Local Governments in Air Toxics Regulation. Texas Chemical Council/ Association of Chemical Industry of Texas's EH&S Seminar Moody Gardens Hotel, Galveston Texas. June 10, 2008.

Fraiser, L.H., and Chaudhuri, I. Short-Term Toxicity Benchmark for Nickel Oxide. 42nd Annual Society of Toxicology Meeting. March 9 – 14, 2002. Salt Lake City, Utah.

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Fraiser, L., Barnett, J.W., and Hixson, E.J. 'Toxicity Equivalents for Chlorinated Hydrocarbon Pesticides Lacking EPA-Verified Toxicity Values.' Toxicologist 14: 1540, 1994.

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APPENDIX B

DETAILS ON THE ESTABLISHMENT OF THE 1-HOUR PRIMARY SO_2 NAAQS LEVEL AND FORM

DETAILS ON THE ESTABLISHMENT OF THE 1-HOUR PRIMARY SO2 NAAQS LEVEL AND FORM

B.1 EPA's Short-Term SO_2 Exposure Threshold Level

There are many controlled human studies that have exposed asthmatic test populations to SO_2 and that have measured small lung function decrements in the asthmatic population, particularly at higher than normal exertion levels. However, most fail to show a statistically significant responses, and even in asthmatics (a sensitive subpopulation), responses are only seen at relatively high concentrations, usually 400 ppb or more, over a 5 to 10-minute period. According to the World Health Organization (WHO), the mean response in asthmatics at 400 ppb over a 5 to 10-minute exposure has been definite though small, whereas at 200 ppb, any change has been minimal and similar in magnitude to effects of exercise alone in clean air.[8]

Despite the conclusions of the WHO, the fact that a single study[9] reported statistically significant increases in respiratory symptoms below a concentration of 400 ppb, and no studies reported clinically relevant decreases in lung function below 400 ppb,[10] EPA identified a short-term exposure threshold for SO_2 of 200 ppb in the most recent decision on the SO_2 NAAQS.[11] One study[12] did report statistically significant lung function declines at 250 ppb, but the change was not large enough to be considered clinically relevant (i.e., increase in specific airway resistance [sRaw] of 100%) and the participants in the study were also required to engage in a higher level of exertion (i.e., ventilation rate of 50 – 60 L/min) than generally used (usually ≈ 40 L/min) in most other studies.

EPA's *Integrated Science Assessment (ISA) for Sulfur Oxides – Health Criteria (Final Report)*[13] (hereafter referred to as the ISA) (Section 4.2.2) indicates that studies have consistently demonstrated that exposure to SO_2 concentrations as low as 200-300 ppb for 5-10 minutes can result in moderate or greater decrements in lung function, evidenced by a ≥15% decline in forced expiratory volume in one second (FEV_1) and/or ≥ 100% increase in sRaw, in a significant percentage of exercising asthmatics. However, lung function decrements have not been consistently shown at 200 – 300 ppb range. In fact, there is only one study that shows a 15% decrease in FEV_1 and that same study shows a 15% increase in FEV_1 in an equal number of test subjects.[14] Furthermore, lung function decrements observed following exposures at these levels have not been shown to be statistically significant at the group mean level.

[8] WHO, 2006. World Health Organization. Air Quality Guidelines Global Update 2005.

[9] Linn, Venet, Shamoo, Valencia, Anzar, Spier, Hackney, 1983. Respiratory effects of sulfur dioxide in heavily exercising asthmatics. *Am Rev Respir Dis* 127:278–283.

[10] Goodman, Dodge, and Bailey, 2010. A framework for assessing causality and adverse effects in humans with a case study of sulfur dioxide. *Reg Toxicol Pharmacol* 58:308–322.

[11] 75 CFR 35220, Jun 22, 2010.

[12] Bethel, Sheppard, Geffroy, Tam, Nadel, and Boushey, 1985. Effect of 0.25 ppm sulfur dioxide on airway resistance in freely breathing, heavily exercising, asthmatic subjects. *Am Rev Respir Dis* 131(4):659–661.

[13] EPA, 2008. Integrated Science Assessment (ISA) for Sulfur Oxides – Health Criteria (Final Report). EPA/600/R-08/047F.

[14] Linn, 2010. A critical review of selected documents concerning proposed revision of the National Ambient Air Quality Standard for sulfur dioxide. Prepared for American Petroleum Institute.

Statistical significance is a key consideration for determining whether an exposure and effect are associated. If the difference between exposed and unexposed groups is not statistically significant, then the exposure of interest was either insufficient to cause the adverse effect (i.e., the effects are caused by something other than the exposure of interest) or the study does not have enough power (i.e., test population was too small, an insufficient number of exposure doses were tested) to establish an association. Because biological effects can be caused by a number of factors in any particular person, causality cannot be inferred simply because an individual has experienced an adverse effect following exposure. Isolated effects (occurring in very few study subjects) and independent effects (occurring in the absence of other effects that are expected via the same mode of action) occur inconsistently and are, thus, more likely to reflect biological effects from another cause or a measurement error than to be exposure-related (Goodman et al., 2010).

The lung function decrements reported by EPA after exposure to 200 ppb SO_2 do not necessarily indicate an effect from SO_2 exposure, any more than lung function improvements of equal magnitude in the same number of individuals (5) signifies a beneficial effect of SO_2. It is unlikely that SO_2 is a causal factor for these effects (decrease or increase in FEV_1) and that instead, these findings simply represent the type of variability typically seen in lung function amongst the test population.

B.1.1 Controlled Exposure Studies Evaluated in Support of the 1-Hour NAAQS

Table 9-3 of EPA's *Risk and Exposure Assessment to Support the Review of the SO_2 Primary National Ambient Air Quality Standards: Final Report*[15] (hereafter referred to as the REA) and Table 3-1 of the ISA indicate that there is only limited evidence of SO_2-induced increases in respiratory symptoms in some asthmatics in the 200 to 300 ppb SO_2 concentration range, stronger evidence with some statistically significant increases in respiratory symptoms at SO_2 concentrations ranging from 400 to 500 ppb, and clear and consistent increases in SO_2 induced respiratory symptoms at concentrations between 600 to 1,000 ppb. Controlled studies of asthmatics exposed to SO_2 for 5 to 10 minutes at increased ventilation evaluated by EPA as part of the ISA (EPA, 2008) are summarized in **Table B-1**.

[15] EPA, 2009. Risk and Exposure Assessment to Support the Review of the SO2 Primary National Ambient Air Quality Standards: Final Report. EPA-452/R-09-007.

TABLE B-1: CONTROLLED HUMAN EXPOSURE STUDIES RELIED UPON BY EPA IN ESTABLISHING THE 1-HOUR SO2 NAAQS THRESHOLD LEVEL

Study	*N*	SO2 Exposure (ppb)	Ventilation Rate (L/min)	Statistically Significant Lung Function Changes	Clinically Relevant Lung Function Changes	Significantly Increased Respiratory Symptoms	NOAEL	LOAEL
Roger et al. (1985)	28	0, 250, 500, 1000	40	500, 1000	1000	1000	500	1000
Bethel et al. (1985)	19	0, 250	50-60	250	None	Symptoms not evaluated	250	None
Kehrl et al. (1987)	9	0, 250	80-90	None				
	10	0, 1000	41	1000	1000	Symptoms not evaluated	None	1000
Linn et al. (1987)	40	0, 200, 400, 600	40	Not reported	400, 600	Not reported	200	400
Linn et al. (1988)	20	0, 300, 600	50	Not reported	Insufficient data	Not reported	Unknown	Unknown
Linn et al. (1990)	21	0, 300, 600	50	Not reported	Insufficient data	Not reported	Unknown	Unknown
Magnussen et al. (1990)	46	0, 500	30[a]	500	500	Symptoms not evaluated	None	500
Schacter et al. (1984)	10	0, 250, 500, 750, 1000	35	750, 1000	750, 1000	Not reported	500	750
Balmes et al. (1987)	8	0, 500, 100	60[a]	500, 1000	500, 1000	Not reported	None	500
Linn et al. (1983)	23	0, 200, 400, 600	48	400, 600	600	200, 400, 600	None	200
Linn et al. (1984)	14	0, 600	50	600	600	600	None	600
Gong et al. (1995)	14	0, 500, 1000	30, 36, or 43	Not reported	Insufficient data	Not reported	Unknown	Unknown
Horstman et al. (1988)	12	0, 1000	40	1000	1000	1000	None	1000

Source: Goodman, Dodge, and Bailey, 2010. A framework for assessing causality and adverse effects in humans with a case study of sulfur dioxide. *Reg Toxicol Pharmacol* 58:308-322.

As shown in **Table B-1**, the only controlled studies in which a statistically significant and clinically relevant lung function decrement accompanied by significantly increased respiratory symptoms were observed included Roger et al. (1985)[16], Linn et al. (1983)[17], Linn et al. (1984)[18] and Horstman et al. (1988)[19]. However, the combination of lung function decrement and increased respiratory symptoms did not occur in the 200 to 300 ppb range in any of these studies. However, EPA reasoned that it is highly likely that these decrements in lung function would result in increased medication use and a disruption of normal activities for a significant percentage of these asthmatics to support its decision to develop a new 1-hour SO_2 NAAQS in 2010 (section 4.3 of REA). The ISA describes the controlled human exposure studies as being the "definitive evidence" for its causal determination between SO_2 exposure and short-term respiratory morbidity (section 5.2).

[16] Roger, Kehrl, Hazucha, Horstman, 1985. Bronchoconstriction in asthmatics exposed to sulfur dioxide during repeated exercise. J Appl Physiol 59:784–791.

[17] Linn, Venet, Shamoo, Valencia, Anzar, Spier, Hackney, 1983. Respiratory effects of sulfur dioxide in heavily exercising asthmatics. Am Rev Respir Dis 127:278–283.

[18] Linn, Avol, Shamoo, Venet, Anderson, Whynot, Hackney, 1984. Asthmatics' responses to 6-hr sulfur dioxide exposures on two successive days. Arch Environ Health 39:313–319.

[19]

B.1.2 Epidemiological Studies Evaluated in Support of the 1-Hour NAAQS

There is a relatively small body of epidemiologic evidence describing positive associations between 1-hour maximum SO_2 levels and respiratory symptoms as well as hospital admissions and Emergency Department (ED) visits for all respiratory causes and asthma.

Ten key epidemiological SO_2 studies conducted in the US were relied upon by EPA in their evaluation of the need for a 1-hour NAAQS for SO_2. Those studies are summarized in **Table B-2**.

TABLE B-2: EPIDEMIOLOGY STUDIES RELIED UPON BY EPA IN ESTABLISHING SUPPORT FOR THE 1-HOUR SO_2 NAAQS THRESHOLD LEVEL

Study	Outcome	Location	99th percentile 1-h max SO_2[a] (ppb)	Pollutants in model	Unit SO_2 Increase	SO_2 RR	95% CI	Notes
Wilson et al. (2005)	Daily ER visits for all respiratory effects and asthma	Portland, ME	47	SO_2	10 μg/m³	1.03	1.01, 1.04	Portland (all respiratory)
		Manchester, NH	69		(3.8 ppb)	1.04	1.00, 1.07	Portland (asthma)
						1.00	0.99, 1.02	Manchester (all respiratory)
						1.02	0.99, 1.06	Manchester (asthma)
Lin et al. (2004)	Childhood asthma hospitalizations	Bronx, NY	119	SO_2	10 ppb	1.19 (OR)	1.11, 1.29	
Jaffe et al. (2003)	Asthma ER visits	Cincinnati, OH	457	SO_2	50 μg/m³ (19 ppb)	1.21[b]	1.09, 1.35	Cincinnati
		Cleveland, OH	211			1.06	0.93, 1.21	Cleveland
		Columbus, OH	51			1.26	0.75, 2.13	Columbus
Sheppard (2003)	Non-elderly hospital admissions for asthma (re-evaluation of)	Seattle, WA	84	SO_2	4.9 ppb	1.01	0.98, 1.03	
Peel et al. (2005)	ER visits for respiratory effects, including asthma, chronic obstructive pulmonary disease (COPD), upper respiratory infection, and pneumonia	Atlanta, GA	81	SO_2	20 ppb	1.008	0.997, 1.019	
Tolbert et al. (2007)	ER visits for respiratory effects, including asthma, COPD, pneumonia, and bronchitis	Atlanta, GA	76	SO_2	16 ppb	1.003	0.997, 1.009	
Schwartz et al., 1996	Daily counts of respiratory related mortality and hospital admissions of elderly	Cleveland, OH	170	SO_2	38 ppb	1.03	0.99, 1.06	
Ito et al. (2007)	ER visits for asthma	New York City	82	SO_2, SO_2, NO_2	6 ppb	1.20 NS	1.13, 1.28 0.9, 1.1	
Schwartz (1995)	Respiratory hospital admissions	New Haven, CT	150	SO_2	50 μg/m³	1.03	1.02, 1.05	New Haven
		Tacoma, WA	100	SO_2	(19 ppb)	1.06	1.01, 1.12	Tacoma
				SO_2, O_3		1.02	0.96, 1.08	New Haven
				SO_2, O_3		0.93	0.83, 1.04	Tacoma
		South Bronx, NY	78	SO_2	10 ppb	1.11	1.06, 1.17	Bronx
NYSDOH (2006)	Asthma ER visits	Manhattan, NY	80	SO_2, O_3		1.11	1.05, 1.17	Bronx
				SO_2, $PM_{2.5}$		1.11	1.04, 1.18	Bronx
				SO_2, NO_2		1.11	1.04, 1.17	Bronx
				SO_2		0.99	0.88, 1.11	Manhattan
				SO_2, O_3		0.99	0.88, 1.12	Manhattan
				SO_2, $PM_{2.5}$		0.97	0.85, 1.11	Manhattan
				SO_2, NO_2		1.01	0.87, 1.16	Manhattan

Source: Goodman, Dodge, and Bailey, 2010. A framework for assessing causality and adverse effects in humans with a case study of sulfur dioxide. *Reg Toxicol Pharmacol* 58:308-322.

While the observational epidemiological studies of SO_2 were designed to address whether SO_2 can cause an increase in adverse health effects at the population level, they do not provide strong evidence for an association between peak ambient SO_2 exposure and lung function in either children or adults (see pg. 3-31 of the ISA).

Determining whether results of epidemiology studies are statistically significant is similar requires some understanding of the risk statistics used. Relative risks (RRs) are used to compare the difference in results between two groups and odds ratios (ORs) compare the odds that an outcome will occur given a particular exposure to the odds of the outcome occurring in the absence of that exposure. Confidence intervals (CIs) for relative risks and odds ratios provide both the risk measure and a range within which the risk or odds likely would fall if the study were repeated numerous times. A relative risk of 1 means there is no difference in risk between the two groups (i.e., no association or no increased risk). Therefore, a relative risk of 1.8 indicates an 80% increased relative risk of disease and a relative risk of 0.8 indicates a decreased risk of 20%. The odds ratio is essentially equivalent to the relative risk, so an odds ratio of 1 means that

exposure does not affect the odds of the outcome. Therefore, if the confidence interval contains "1", it is not statistically significant.

As can be seen in **Table B-2**, more studies than not have failed to find statistically significant associations between long-term and short-term SO_2 concentrations and adverse health outcomes (i.e., most CIs contain 1). Of the 10 epidemiological studies, most found either no association or very small positive associations (i.e., less than 10% increase in relative risk). The highest relative risks are reported by Ito et al. (2007)[20], Lin et al. (2004)[21] and Jaffee et al. (2003),[22] although even those studies only indicate approximately 20% increase in relative risk.

Among the studies for which weak positive associations were observed, conclusions were either: 1) based only on results from single-pollutant models (i.e., multi-pollutant models were not applied to address potential confounding by other pollutants); or 2) were based on results from single-pollutant models that were not statistically significant in multi-pollutant models.[23] Only two studies provided evidence of statistically significant associations in multi-pollutant models (Bronx, NYDOH, 2006[24] and NYC, Ito, 2007).

EPA used the 1-hour daily maximum air quality data from epidemiology studies presented in Figures 5-1 to 5-5 of the REA to inform both the upper and lower ranges of alternative SO_2 standards for analysis in the REA. However, despite statements that the new 1-hour SO_2 NAAQS is informed by both clinical and epidemiological studies showing an association between short-term exposures to SO_2 and adverse respiratory effects, in its REA, EPA did not ultimately find the overall breadth of the epidemiological evidence robust enough to support a quantitative assessment of risk (pg. 58 of the REA) because of the limited number of studies focused on SO_2-concentration-response relationships, lack of statistically significant findings, and the fact that inclusion of other pollutants in multi-pollutant models resulted in a loss of statistical significance for the SO_2 effect estimate in about half of the studies, indicating that an independent effect of SO_2 on ED visits and/or hospitalizations was not consistently observed.

B.2 EPA's Risk and Exposure Assessment

EPA conducted a series of three analyses to estimate risks associated with 5-minute SO_2 exposures ranging from 100-400 ppb in exercising asthmatics in the REA:

[20] Ito, Thurston, and Silverman, 2007. Characterization of PM2.5, gaseous pollutants, and meteorological interactions in the context of time-series health effects models. *J Expo Sci Environ Epidemiol* 17 (Suppl. 2), S45–S60.

[21] Lin, Hwang, Pantea, Kielb, and Fitzgerald, 2004. Childhood asthma hospitalizations and ambient air sulfur dioxide concentrations in Bronx County, New York. *Arch Environ Health* 59(5):266–275.

[22] Jaffe, Singer, and Rimm, 2003. Air pollution and emergency department visits for asthma among Ohio Medicaid recipients, 1991–1996. *Environ Res* 91:21–28.

[23] Goodman, Dodge, and Bailey, 2010. A framework for assessing causality and adverse effects in humans with a case study of sulfur dioxide. Reg Toxicol Pharmacol 58:308-322.

[24] NYSDOH, 2006. New York State, Department of Health. A study of ambient air contaminants and asthma in New York City. Part A: a comparison of ambient air quality in the Bronx and Manhattan. Part B: air contaminants and emergency department visits for asthma in the Bronx and Manhattan (Final). Report to Agency for Toxic Substances and Disease Registry (ATSDR); New York State, Energy Research and Development Authority. NTIS PB2006-113523, July, 260p.

1) the first analysis estimated the number of days per year that measured or statistically estimated 5-minute daily maximum SO_2 concentrations equaled or exceeded health benchmark values of 100, 200, 300 and 400 ppb;

2) the second analysis involved exposure analysis case studies conducted in the St. Louis modeling domain and Greene County Missouri to provide estimates of the number and percent of asthmatics residing within 20 kilometers (km) of SO_2 sources that experience 5-minute exposures to 100, 200, 300, and 400 ppb SO_2, while at elevated ventilation rates; and

3) the third analysis was a quantitative risk assessment to estimate health risks for the number and percent of asthmatics estimated to experience moderate or greater lung function responses.

The third approach combined results from the exposure analysis (i.e., the number of exposed total asthmatics or asthmatic children in St. Louis and Green County Missouri while at moderate or greater exertion) with exposure-response functions derived from individual level data from controlled human exposure studies to estimate the percentage and number of exposed asthmatics and asthmatic children in St. Louis and Greene County Missouri likely to experience a moderate or greater lung function response (i.e., decrements in lung function defined in terms of forced expiratory volume (FEV_1) and specific airway resistance (sRAW). These results are particularly relevant here because they were conducted in Missouri.

B.2.1 Study Areas

The selection of areas to include in the exposure analysis considered the availability of ambient monitoring, the presence of significant and diverse SO_2 emission sources, population demographics, and results of the ambient air quality characterization. Missouri was one of only a few states that reported both 5-minute maximum and continuous 5-minute SO_2 ambient monitoring data (14 total monitors) and had over thirty 1-hour SO_2 monitors in operation at some point during the period from 1997 to 2007. Two counties in Missouri were modeled, St. Louis and Green, which included the greater St. Louis and Springfield Metropolitan Statistical Areas (MSAs), respectively. Because of the complexity of the modeling analysis, the modeling domain was limited to three (those directly surrounding the city of St. Louis) of the 16 counties in the St. Louis MSA: St. Louis City, St. Louis County, and St. Charles County. Calendar year 2002 was simulated for both modeling domains to characterize the most recent year of emissions data available for the study locations.

For the analysis, major facilities were defined as those with an SO_2 emission total exceeding 1,000 tons per year in 2002. This resulted in the identification of 11 (combined) stacks in Greene County and 38 (combined) stacks in St. Louis, with an additional 45 (combined) stacks identified across the state border that could influence concentrations in St. Louis. Activity from the Port of St. Louis was modeled as fourteen area sources along the waterfront in the St. Louis analysis. Non-point sources, constituting industrial, commercial and institutional facilities, were also modeled.

B.2.2 Exposure Analysis

The exposure assessment included the total population residing in each modeled area and susceptible subpopulations including: asthmatic children (5-18 years) and all asthmatics (all ages). The proportion of the population of children characterized as being asthmatic was estimated by statistics on national asthma prevalence rates recently used in the NAAQS review for ozone.[25] EPA indicates that these data were used rather than the aggregate data available at the Missouri county level, to retain the variability in asthma prevalence observed with children of different ages. Adult asthma prevalence rates were estimated by gender and for each particular modeling domain based on Missouri regional data.[26]

The Air Pollutants Exposure (APEX) model, an EPA human exposure model, was used to estimate 5-minute population exposures using census block level hourly SO_2 concentrations estimated by AERMOD. Population demographics were drawn from the year 2000 Census at the tract, block-group, or block-level, and a national commuting database based on 2000 census data provides home-to-work commuting flows.

APEX simulates the movement of individuals through time and space and estimates their exposure to a given pollutant in indoor, outdoor, and in-vehicle microenvironments. Daily activity patterns for individuals in a study area were obtained from detailed diaries that are compiled in the Consolidated Human Activity Database (CHAD).

Asthmatic exposures were characterized only when the individual was at moderate or greater exertion levels during the exposure events. Exposure profiles were used to calculate the number of days per year an individual had at least one 5-minute exposure above the potential health effect benchmark levels of 100, 200, 300, and 400 ppb. Exposures were calculated in APEX by identifying concentrations in the microenvironments visited by the person according to the composite diary. Estimated micro-environmental concentrations account for the contribution of ambient (outdoor) pollutant concentration and influential factors, such as the penetration rate into indoor microenvironments, air exchange rates, decay/deposition rates, proximity to important outdoor sources, and indoor source emissions.

B.2.3 Risk Characterization

Two types of risk measures were generated for the risk assessment:

1) estimates of the number and percentage of all asthmatics (or asthmatic children) experiencing one or more occurrences of a defined lung function response associated with 5-minute exposures to SO_2 while engaged in moderate or greater exertion; and

[25] EPA, 2007. Ozone Population Exposure Analysis for Selected Urban Areas. EPA-452/R-07-010. Available at: http://epa.gov/ttn/naaqs/standards/ozone/s_o3_cr_td.html.

[26] Missouri Department of Health. (2002). Health Risk Behaviors of Adult Missourians, 2002 Annual Report, Behavioral Risk Factor Surveillance System. Division of Community and Public Health, Bureau of Health Informatics, Available at: http://www.dhss.mo.gov/BRFSS/2002AnnualReport.pdf.

2) the number of occurrences of the lung function response in asthmatics (or asthmatic children) in a year associated with 5-minute exposures at moderate or greater exertion.

In the final SO_2 rule,[27] the EPA Administrator concluded that the St. Louis exposure analysis indicated that a 1-hour standard at 100 ppb (25 ppb higher than the current NAAQS) would still be estimated to protect greater than 99% of asthmatic children at moderate or greater exertion from experiencing at least one 5-minute SO_2 exposure equal to or greater than 400 ppb per year, and about 97% of these children from exposures equal to or greater than 200 ppb. This conclusion is based on the discussion provided on page 393 of the REA and is illustrated in Table 8-19 of the REA, which is reproduced below.[28]

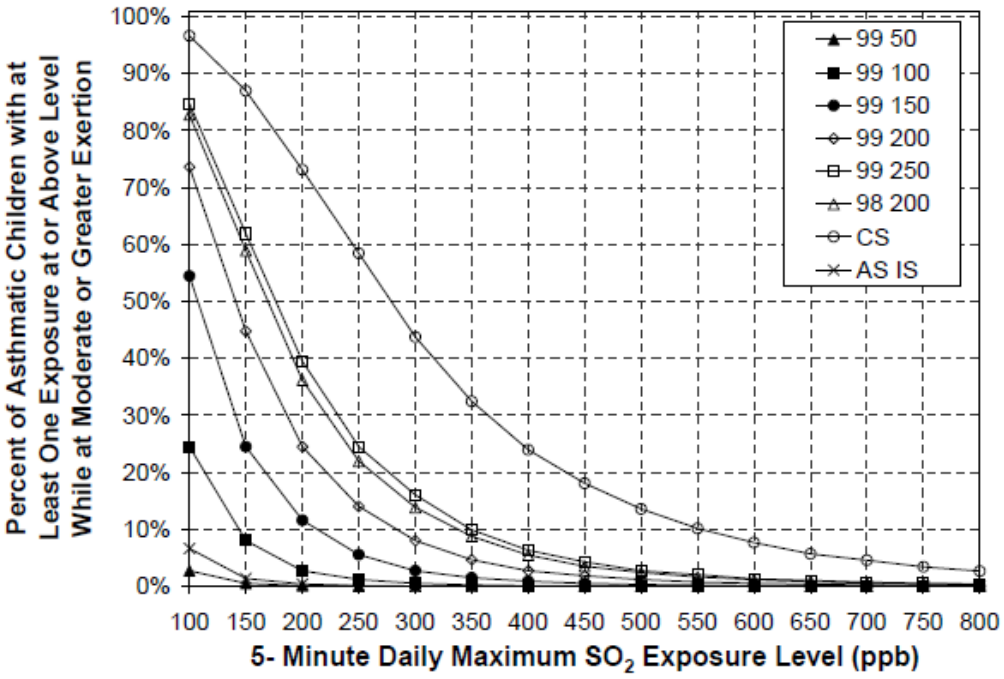

Figure 8-19. Percent of all asthmatics (top) and asthmatic children (bottom) experiencing at least one day with a 5-minute SO_2 exposure above selected benchmark levels in St. Louis, year 2002 air quality *as is* and adjusted to just meeting the current and potential alternative standards.

Source: EPA, 2009. Risk and Exposure Assessment to Support the Review of the SO_2 Primary National Ambient Air Quality Standards.

The line with the squares represents the percentage of exercising asthmatics with at least one 5-minute exposure at or above the levels indicated on the horizontal axis of the figure per year, if the NAAQS were set at an SO_2 level of 100 ppb (99th percentile). The line with the triangles represents the same, if the NAAQS were set a level of 50 ppb. As can be clearly seen by interpolating between those two lines (line with squares = 100 ppb and line with triangles = 50

[27] 75 FR 35520, Jun 22, 2010.

[28] EPA, 2009. U.S. Environmental Protection Agency. Risk and Exposure Assessment to Support the Review of the SO_2 Primary National Ambient Air Quality Standards. EPA-452/R-09-007.

ppb), at the current SO_2 NAAQS (75 ppb), the percentage of exercising asthmatics in St. Louis that are exposed to a 5-minute SO_2 concentration of 400 ppb or greater at least once is limited to much less than 1% (close to 0%).

There are several things to consider in determining the implications of the results from the St. Louis analysis. First, while aggregate data for Missouri (by county) was used in estimating the asthma prevalence in adults for St. Louis, EPA used national asthma prevalence rates in children, which resulted in higher asthma prevalence estimates than would have been estimated using Missouri data alone (i.e., 9.2% vs 8.8% based on 2002 data).[29] This approach overestimated the number of asthmatics in St. Louis and since the risk assessment is entirely focused on responses in asthmatics, it overestimated overall risk. In addition, the risk assessment emphasizes single exposures (i.e., at least one) to 5-minute concentrations above various benchmarks, when in fact, there is no evidence that a single exposure to 400 ppb SO_2 (or even 1,000 ppb) would have any lasting or detrimental effects on asthmatics or any other segment of the population. Finally, as discussed in the section on controlled human exposure studies, at 200 ppb SO_2, lung function changes are minimal and similar in magnitude to effects of exercise alone in clean air. As a result, many have argued that the 1-hour SO_2 standard should not have taken into account limiting 5-minute peaks as low as 200 ppb.

B.3 More Scientifically Supportable Short-Term Exposure Threshold

Primary NAAQS standards are established to provide public health protection, including protecting the health of "sensitive" populations such as asthmatics, children, and the elderly. "The legislative history of Section 109 indicates that a primary standard is to be set at ''the maximum permissible ambient air level * * *which will protect the health of any [sensitive] group of the population,'' and that for this purpose ''reference should be made to a representative sample of persons comprising the sensitive group rather than to a single person in such a group.''[30] Therefore, clinically relevant and statistically significant health effects observed in a representative sample of persons comprising the sensitive group should be used as the bases for primary standards, rather than those observed in a single individual in a sensitive subpopulation.

Many who commented on the rule, as well as state and non-state petitioners in litigation[31] over the rule, argued that the short-term exposure threshold for SO_2 should have been established at a concentration at least as high as 400 ppb, rather than 200 ppb. Even at 400 ppb, effects are transient, reversible, and of low severity.

B.4 Form and Level of the 1-Hour SO_2 NAAQS

NAAQS standards are established for use in evaluating area-wide concentrations of SO_2, not concentrations measured at discrete locations. Site-to-site correlations of monitored SO_2 concentrations can vary from very low to very high values, suggesting that the concentration of

[29] *Ibid*, p. 222.
[30] 75 CFR 35220 (Jun 22, 2010), footnote 1.
[31] USCA Case #10-1252, 2012.

SO_2 measured at any given monitoring site, may not be highly correlated with the average community concentration in some areas. There is always a random component to instrumental measurement error and the practice of averaging across multiple ambient monitors in a region helps to reduce the instrument measurement error. For this reason, compliance with NAAQS is often based on a statistical form.

B.4.1 Form

The form of the 1-hour Primary SO_2 NAAQS was established as the 3-year average of the 99th percentile (in most locations analyzed, the 99th percentile corresponds to the 4th highest daily maximum concentration in a year)[32] of the yearly distribution of 1-hour daily maximum concentrations. The form of the 1-hour SO_2 NAAQS was based on two studies[33] evaluated in the ISA, which reported an increase in SO_2-related respiratory health effects at the upper end (above 90th percentile values) of the distribution of ambient area-wide SO_2 concentrations (Section 5.3, p. 5–9 of the ISA) and the conclusion that a 99th percentile form for the 1-hour SO_2 NAAQS would be effective at limiting 5-minute peak SO_2 concentrations.

Using data from a small number of monitors that voluntarily report 5-minute data, EPA demonstrated that there is a high correlation between the 5-minute maximum level and the corresponding 1-hour average SO_2 concentration (section 2.5.2 of the REA) and used this information to support that a 1-hour standard, if set at the appropriate level and form, can limit peak (5-minute) exposures.

Therefore, even though the SO_2 NAAQS does not have a 5-minute averaging time, the form and level were established specifically to limit 5-minute peak concentrations (section 10.5.3 and Figures 7–27 and 7–28 of the REA). EPA concluded that a concentration-based NAAQS that is averaged over a three year period better reflects the continuum of health risks of increasing SO_2 concentrations by weighting years when 1-hour daily maximum SO_2 concentrations are well above the level of the standard more heavily than those when 1-hour daily maximum SO_2 concentrations are just above the level of the standard.

B.4.2 Level

The highest 98th and 99th percentile 1-hour daily maximum air quality levels were found in analyses conducted in the cities of Cincinnati (Figure 5-2 of the REA), Cleveland (Figures 5-2 and 5-4 of the REA) and New Haven (Figure 5-4 of the REA) and these studies were reported to have shown positive associations with respiratory related hospital admissions or ED visits during time periods when 98th and 99th percentile 1-hour daily maximum SO_2 concentrations ranged from 126 ppb to 457 ppb. On the bases of these epidemiological studies, and the clinical studies discussed previously, EPA analyzed SO_2 levels ranging from 50 ppb to 250 ppb as possible levels for an

[32] 75 CFR 35520, June 22, 2010.

[33] Schwartz, 1995. Short term fluctuations in air pollution and hospital admissions of the elderly for respiratory disease. *Thorax* 50:531-538 and Lin, Hwang, Pantea, Kielb, and Fitzgerald, 2004. Childhood asthma hospitalizations and ambient air sulfur dioxide concentrations in Bronx County, New York. *Arch Environ Health* 59:266-275.

hourly NAAQS that would protect against morbidity observed following peak (5-minute) exposures.

Because there were relatively few monitors that reported 5-minute SO_2 concentrations in the US (98 at the time of the SO_2 NAAQS review), a model was developed to estimate 5-minute maximum SO_2 concentrations from comprehensive 1-hour SO_2 ambient monitoring data (more than 800 monitors). The approach was based on monitored hourly SO_2 concentration levels and the variability observed at the monitors reporting both the 5-minute maximum and 1-hour average SO_2 concentrations.

EPA proposed to set the level of this new 1-hour standard within the range of 50 to 100 ppb and solicited comment on standard levels as high as 150 ppb. EPA's REA concluded that the level for the 1-hour NAAQS would need to be based on a weight-of-evidence approach, given the different types of information (controlled human exposures and epidemiological studies), with the following options:

1. Emphasize Epidemiological Study Results
    a. 99th percentile 1-hour daily maximum standard in the lower end of the proposed range for consideration (50 ppb – 100 ppb)
    b. In consideration of the fact that the strongest epidemiologic evidence of an association between ambient SO_2 and ED visits and hospitalizations is in cities where 99th percentile 1-hour daily maximum SO_2 concentrations ranged from about 75 to150 ppb, a 99th percentile 1-hour daily maximum standard in the upper end of the proposed range for consideration could be justified (100 ppb – 150 ppb)

2. Emphasize Controlled Human Study Results
    a. Based on the presumption that participants in human exposure studies do not represent the most SO_2 sensitive individuals, consideration could be given to a 99th percentile 1-hour daily maximum standard that provides increased protection against peak concentrations lower than 200 ppb to provide a margin of safety for these SO_2 sensitive individuals (50 – 100 ppb)
    b. Based on the fact that statistical significance is not seen and lung function decrements are not routinely accompanied by respiratory symptoms until SO_2 concentrations of at least 400 ppb are reached, it could be argued that the upper end of this range of alternative standard levels could be sufficient to protect public health (100 ppb – 150 ppb)

EPA rejected an alternate standard as high as 150 ppb based on the conclusion that it would not adequately limit 5-minute SO_2 exposures ≥ 200 ppb. This is based on findings from the St. Louis exposure analysis, for which EPA estimated that at a 1-hour daily maximum of 150 ppb would only protect 88% of asthmatic children at moderate or greater exertion from experiencing at least one SO_2 exposure ≥ 200 ppb per year (Figure 8-19 of the REA). If on the other hand, one accepts that 400 ppb represents a more appropriate exposure threshold, then based on the St. Louis exposure analysis, a 99th percentile 1-hour daily maximum of 150 ppb would protect > 99% of asthmatics, or asthmatic children at elevated ventilation rates from experiencing at least one 5-

minute SO_2 exposure ≥ 400 ppb. In addition, the estimated median percentage of exposed asthmatics at elevated ventilation rates expected to experience lung function decrement (≥ 100% increase in sRaw) at least once per year ranges from 2.9% to 3.6% for asthmatics (4.6% to 5.4% for asthmatic children), depending on the model used. By comparison, at a 1-hour daily maximum of 50 to 100 ppb, the estimated median percentage of exposed asthmatics at elevated ventilation rates expected to experience lung function decrement at least once per year ranges from 0.3% to 1.3% at 50 ppb and 0.7% 1.9% at 100 ppb.

Given a 1-hour daily maximum of 50 or 100 ppb, the 40 county air quality analysis estimated at most zero to 2 days per year when statistically estimated 5-minute daily maximum SO_2 concentrations would be ≥ 400 ppb. On the other hand, a 99th percentile 1-hour daily maximum standard of 150 ppb would be estimated to result in at most 7 days/year where 5-minute maximum SO_2concentrations were ≥ 400 ppb

Taken together, this information supports that limiting the 99th percentile 1-hour daily maximum to 150 ppb could similarly limit 5-minute SO_2 exposures ≥ 400 ppb when compared to standards in the range of 50-100 ppb and that in establishing the NAAQS at 75 ppb, EPA set an extremely stringent standard.

APPENDIX C

ISSUES ASSOCIATED WITH THE CLEAN AIR TASK FORCE (CATF) AND ENVIRONMENTAL INTEGRITY PROJECT (EIP) MODELS

ISSUES ASSOCIATED WITH THE CATF AND EIP MODELS

The underlying model used in the health impact analyses performed by the Clean Air Task Force (CATF) and the Environmental Integrity Project (EIP), the Climatological Regional Dispersion Model (CRDM), uses outdated algorithms and assumptions that are not in line with state-of-practice dispersion modeling approaches (both short-range and long-range transport and dispersion) that have been developed since the 1970's. The model does not fully account for the complexities of formation and deposition of secondary particulates during local and regional transport and dispersion within the atmosphere. Even the EPA[34] states that "Because of limited validation studies of the Source-Receptor (S-R) Matrix, it should be treated as a screening model that provides a crude estimate of the likely impact of a change in emissions on ambient $PM_{2.5}$ levels." EPA further states that "Relative to more sophisticated and resource-intensive three-dimensional modeling approaches, the CRDM does not fully account for all the complex chemical interactions that take place in the atmosphere in the secondary formation of $PM_{2.5}$. Instead it relies on more simplistic species dispersion-transport mechanisms supplemented with chemical conversion at the receptor location." Finally, EPA concludes that that "More sophisticated atmospheric dispersion models should be used to obtain detailed estimates of ambient air quality changes."

However, the issues associated with the health impact assessments conducted by groups like the CATF and EIP are not isolated to the air dispersion modeling. These groups are often not transparent with regard to the many inputs that are used in the analyses and on which the results are critically dependent. Because of the simplistic algorithms used, the $PM_{2.5}$ attributed to specific sources is likely overstated, perhaps dramatically. Moreover, the likelihood that distant populations are *actually* exposed is not even contemplated by the models. Instead, the standard presumption is that entire populations of counties are exposed to estimated outdoor concentrations of $PM_{2.5}$ that may or may not reach the hypothetical populations and may not be formed in the first place.

Details of the issues identified with the CATF and EIP models based on available information, many of which have been identified by others[35], include but are not limited to:

- Model Results are Highly Dependent on Emission Inventory Used
  - In calibration exercises, model estimates of annual concentrations at county centroids were compared to actual annual concentrations, from spatially-interpolated ambient monitor values, at county centroids. The county centroid values for model and monitor values, each, were averaged across the state to provide state values.
  - In a calibration exercise conducted by Abt Associates (2010) that used the 2001 emission inventory developed for the Clean Air Interstate Rule (CAIR) Rule and 2002 ambient monitoring data, the model was found to overestimate monitored concentrations for all states

[34] EPA, 2015. User's Manual for the Co-Benefits Risk Assessment (COBRA) Screening Model – Version 2.7.
[35] A Case Study: The Public Health Consequences of Air Emissions from Coal-Fired Power Plants in the St. Louis Area. http://www.americaspower.org/sites/default/files/FinalGradientReport_090115.pdf.

  - In a calibration exercise conducted by EPA (EPA, 2015), in which the 2005 emissions inventory developed for the Mercury Air Toxics Standard (MATS) Rule and 2005 ambient monitoring data, the model was found to underestimate monitored concentrations for 40 out of the 49 contiguous states
  - The results of the calibration exercises seem to critically depend on the emissions inventory used

- Simplistic Assumptions About Air Chemistry Results in Unrepresentative $PM_{2.5}$ Concentrations and Likely Overestimates Amount of $PM_{2.5}$ Attributable to Specific Sources
  - Ammonium nitrate and ammonium sulfate result from reactions among several precursor species
  - Ingredients for $PM_{2.5}$ (sulfate, nitrate, ammonium, VOC aerosols, and direct $PM_{2.5}$), which derive from many different source types (not just CFPPs) are summed to get a total for each county
  - Ammonium reacts preferentially with sulfates to form ammonium sulfates
    - For every mole of sulfate, two moles of ammonium are required
    - If there is little ammonium, sulfate is assumed to form ammonium bisulfate with leftover particulate sulfate
    - If there is an intermediate amount of ammonium, sulfate is assumed to form a combination of ammonium bisulfate and ammonium sulfate
    - If there is a lot of ammonium, sulfate is assumed to form ammonium sulfate
  - Ammonium nitrate formation is assumed only when there is excess ammonium present, and only under low temperature conditions
    - One mole of ammonium reacts with one mole of nitrate to produce ammonium nitrate
  - Organic VOCs and directly emitted $PM_{2.5}$ are added to the ammonium sulfate, ammonium bisulfate, sulfate, and ammonium nitrate to calculate overall concentration of $PM_{2.5}$
  - This approach likely overestimates the $PM_{2.5}$ attributed to specific sources because secondarily formed $PM_{2.5}$ would be completely attributable to a specific CFPP only if all the necessary precursor compounds are present in the county of interest and from the same CFPP to which impacts are attributed

- The Bases for the 2012 CATF Health Impact Estimates on the CATF Website Are Unclear
  - According to the Technical Support Document for the Power Plant Impact Estimate Tool (Abt, 2010) prepared for CATF, the EPA Multipollutant Analysis baseline scenario and the control scenario of the CAIR were used to forecast 2010, 2015 and 2020 emissions and impact levels (see Section A.2 of Appendix A)
  - However, section A.4 of Appendix A of the Technical Support Document (Abt, 2010) indicates that a 2001 emission inventory developed for the CAIR Rule was used to perform the model calibration comparing model levels to 2002 monitoring data
  - It can only be assumed that an approach similar to that discussed in the Technical Support Document (Abt, 2010) was used to estimate the 2012 health impact estimates currently provided on the CATF website (CATF, 2014), but the specifics are unclear, particularly regarding the emission inventory used

- The Majority of Impacts Are Estimated at Locations Distant from Modeled Plants

- This fact is rarely appreciated by citizens living near the modeled plants
- Likelihood that distant populations are *actually* exposed is not evaluated
  - Instead, the standard presumption is that entire populations of counties are exposed to estimated outdoor concentrations of $PM_{2.5}$ that may or may not reach the hypothetical populations and may not be formed in the first place
    - Majority of directly emitted $PM_{2.5}$ and $PM_{2.5}$ formed from other gaseous pollutants will not reach the populations assumed to be exposed because
      - they are modeled to a single hypothetical point at the center of each county, and
      - people spend approximately 90% of their time indoors, where concentrations of outdoor pollutants are generally much lower
  - Unconstrained $PM_{2.5}$ formation from emissions of gaseous species is assumed even though the conditions (temperature, humidity, season) for formation may not be favorable

- Transfer Coefficients Are Gross Oversimplifications
  - Transfer coefficients for each source modeled are used to calculate the proportion of directly emitted $PM_{2.5}$ emissions and $PM_{2.5}$ precursor species (e.g., SO_2, NOx, VOCs) emissions from each source
  - Both CATF and EIP transfer coefficients were derived from a single year (1990) of meteorological data for only 100 meteorological stations
    - 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)
    - 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 a significant source of uncertainty

- Hypothetical Impacts Are *Estimated* Under Assumption that Statistical Correlations from Epidemiology Studies Are *Causal* and Not Subject to Confounding, Bias, or Measurement Error
  - See discussion of substantial confounding/bias in epidemiology studies in **Section 3.2.1**
  - As previously discussed, it is widely acknowledged that causal inferences cannot be made from epidemiology studies (see discussion on limitations of epidemiology studies for making causal inferences in **Section 3.2.1**)
  - There are numerous issues associated with the epidemiology studies used by CATF and EIP, many of which likely lead to gross overestimates of health impacts, that should be considered when interpreting the hypothetical health impact assessments (see Gradient report (2015) for a discussion of those issues)

- Health Impacts from Labadie Energy Center Alleged by CATF (2014) and EIP (2012) Are Wildly Divergent
  - Details of the CATF 2014 (estimates for 2012 on CATF website) methodology are not publically available but the CATF 2010 report describes the epidemiology studies relied upon and it is assumed that those same studies were used in the CATF 2014 estimates
  - EIP (2012) calculated larger impacts, as compared to CATF (2014), using different epidemiology studies and a different emission inventory from the CATF studies

- The wildly divergent effect estimates attributed by the CATF (2014) and EIP (2012) to the Labadie plant highlight how dramatically different assumptions and the use of different epidemiology studies and emission inventories can affect the health impact estimates
- Hypothetical health impacts estimated using models such as those employed by CATF (2014) and EIP (2012) are highly uncertain and easily manipulated through choice of input parameters