



# **Policy Assessment for the Review of the Primary National Ambient Air Quality Standard for Sulfur Oxides, External Review Draft**

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# **Policy Assessment for the Review of the Primary National Ambient Air Quality Standard for Sulfur Oxides, External Review Draft**

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## LIST OF ACRONYMS AND ABBREVIATIONS

AHR	airway hyperresponsiveness
APEX	Air Pollutants Exposure model
AQCD	Air Quality Criteria Document
AQS	Air Quality System
CAA	Clean Air Act
CASAC	Clean Air Scientific Advisory Committee
CHAD	Consolidated Human Activity Database
DV	design value
ED	emergency department
EGU	Electricity generating unit
EPA	Environmental Protection Agency
FEM	federal equivalent method
FEV <sub>1</sub>	forced expiratory volume in one minute
FRM	federal reference method
IRP	Integrated Review Plan
ISA	Integrated Science Assessment
ME	microenvironment
NAAQS	National Ambient Air Quality Standard
NCEA	National Center for Environmental Assessment
NEI	National Emissions Inventory
NO <sub>2</sub>	nitrogen dioxide
O <sub>3</sub>	ozone
OAQPS	Office of Air Quality Planning and Standards
ppb	parts per billion
ppm	parts per million
PA	Policy Assessment
PM	particulate matter
REA	Risk and Exposure Assessment
SLAMS	State and Local Air Monitoring Stations
SO <sub>2</sub>	sulfur dioxide
SO <sub>3</sub>	sulfur trioxide
SO <sub>x</sub>	oxides of sulfur
sRaw	specific airway resistance
USB	United States background
UVF	ultraviolet fluorescence

# 1 INTRODUCTION

## 1.1 PURPOSE

This document, *Policy Assessment for the Review of the Primary National Ambient Air Quality Standard for Sulfur Oxides, External Review Draft* (hereafter referred to as *Draft PA*), presents the draft policy assessment for the U.S. Environmental Protection Agency's (EPA's) current review of the primary (health-based)<sup>1</sup> national ambient air quality standard (NAAQS) for sulfur oxides (SO<sub>x</sub>).<sup>2</sup> The overall plan and schedule for this review were presented in the *Integrated Review Plan for the Primary National Ambient Air Quality Standard for Sulfur Dioxide* (IRP; U.S. EPA, 2014). The IRP also identified key policy-relevant issues to be addressed in this review and discussed the key documents that generally inform NAAQS reviews, including an Integrated Science Assessment (ISA), a Risk and Exposure Assessment (REA), and a Policy Assessment (PA).

The PA presents a staff evaluation of the policy implications of the key scientific and technical information in the ISA and REA for consideration by the Administrator.<sup>3</sup> Ultimately, a final decision on the primary standard for SO<sub>x</sub> will reflect the judgments of the Administrator. The role of the PA is to help “bridge the gap” between the Agency's scientific assessments presented in the ISA and REA, and the judgments required of the EPA Administrator in determining whether it is appropriate to retain or revise the NAAQS.

In evaluating the adequacy of the current standard and whether it is appropriate to consider alternative standards, the PA focuses on information that is most pertinent to evaluating the basic elements of the NAAQS: indicator, averaging time, form, and level.<sup>4</sup> These elements,

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<sup>1</sup> The EPA is separately reviewing the welfare effects associated with sulfur oxides and the public welfare protection provided by the secondary SO<sub>2</sub> standard, in conjunction with a review of the secondary standards for nitrogen oxides and particulate matter with respect to their protection of the public welfare from adverse effects related to ecological effects (U.S. EPA, 2017a).

<sup>2</sup> This review focuses on the presence in ambient air of sulfur oxides, a group of closely related gaseous compounds that include sulfur dioxide and sulfur trioxide and of which sulfur dioxide is the most prevalent. Particulate atmospheric transformation products of SO<sub>x</sub>, such as sulfates, are addressed in the review of the NAAQS for particulate matter.

<sup>3</sup> The terms “staff,” “we,” and “our” throughout this document refer to the staff in the EPA's Office of Air Quality Planning and Standards (OAQPS).

<sup>4</sup> The basic elements of a standard include the indicator, averaging time, form, and level. The indicator defines the chemical species or mixture to be measured in the ambient air for the purpose of determining whether an area attains the standard. The averaging time defines the period over which air quality measurements are to be obtained and averaged or cumulated. The form of a standard defines the air quality statistic that is to be compared to the level of the standard in determining whether an area attains the standard. The level of the standard defines the air quality concentration used for that purpose (i.e., an ambient air concentration of the indicator).

1 which together serve to define each standard, must be considered collectively in evaluating the  
2 health protection afforded by the primary standard for SO<sub>x</sub>.

3 The development of the PA is also intended to facilitate advice to the Agency and  
4 recommendations to the Administrator from an independent scientific review committee, the  
5 Clean Air Scientific Advisory Committee (CASAC), as provided for in the Clean Air Act  
6 (CAA). As discussed below in section 1.2.1, the CASAC is to advise not only on the Agency's  
7 assessment of the relevant scientific information, but also on the adequacy of the current  
8 standards, and to make recommendations as to any revisions of the standards that may be  
9 appropriate. The EPA makes available to the CASAC and the public one or more drafts of the  
10 PA for CASAC review and public comment.

11 The decision whether to prepare one or more drafts of the PA is influenced by  
12 preliminary staff conclusions and associated CASAC advice and public comments, among other  
13 factors. Typically, a second draft PA has been prepared in cases where the available information  
14 calls into question the adequacy of the current standard and analyses of potential alternative  
15 standards are developed taking into consideration CASAC advice and public comment. In such  
16 cases, a second draft PA includes preliminary staff conclusions regarding potential alternative  
17 standards and undergoes CASAC review and public comment prior to preparation of the final  
18 PA. When such analyses are not undertaken, a second draft PA may not be warranted.

19 In this draft PA, we take into account the available scientific and technical information,  
20 as assessed in the second draft *Integrated Science Assessment for Sulfur Oxides – Health Criteria*  
21 (second draft ISA [U.S. EPA, 2016]) and the draft *Risk and Exposure Assessment for the Review*  
22 *of the Primary National Ambient Air Quality Standard for Sulfur Oxides, External Review Draft*  
23 (draft REA [U.S. EPA, 2017b]). The evaluation and preliminary staff conclusions presented in  
24 this draft PA for the primary NAAQS for SO<sub>x</sub> have been informed by comments and advice  
25 received from the CASAC in their reviews of the other draft Agency documents prepared thus  
26 far in this NAAQS review. Review and comments from the CASAC, and public comment, on  
27 this draft of the PA will inform the final evaluation and staff conclusions in the final PA. The  
28 final PA will inform the Administrator's decision in this review of the primary SO<sub>2</sub> NAAQS.

29 Beyond informing the EPA Administrator and facilitating the advice and  
30 recommendations of the CASAC, the PA is also intended to be a useful reference to all parties  
31 interested in the review of the primary NAAQS for SO<sub>x</sub>. In these roles, it is intended to serve as  
32 a source of policy-relevant information that is informing the Agency's review of the primary  
33 NAAQS for SO<sub>x</sub>, and it is written to be understandable to a broad audience.

## 1.2 BACKGROUND

### 1.2.1 Legislative Requirements

Two sections of the CAA govern the establishment and revision of the NAAQS. Section 108 [42 U.S.C. 7408] directs the Administrator to identify and list certain air pollutants and then to issue air quality criteria for those pollutants. The Administrator is to list those pollutants that in his “judgment, cause or contribute to air pollution which may reasonably be anticipated to endanger public health or welfare,” “the presence of which in the ambient air results from numerous or diverse mobile or stationary sources”; and for which he “plans to issue air quality criteria....” Air quality criteria are intended to “accurately reflect the latest scientific knowledge useful in indicating the kind and extent of all identifiable effects on public health or welfare which may be expected from the presence of [a] pollutant in the ambient air....” 42 U.S.C. § 7408(a)(2).

Section 109 [42 U.S.C. 7409] directs the Administrator to propose and promulgate “primary” and “secondary” NAAQS for pollutants for which air quality criteria are issued [42 U.S.C. § 7409(a)]. Section 109(b)(1) defines primary standards as ones “the attainment and maintenance of which in the judgment of the Administrator, based on such criteria and allowing an adequate margin of safety, are requisite to protect the public health.”<sup>5</sup> Under section 109(b)(2), a secondary standard must “specify a level of air quality the attainment and maintenance of which, in the judgment of the Administrator, based on such criteria, is requisite to protect the public welfare from any known or anticipated adverse effects associated with the presence of [the] pollutant in the ambient air.”<sup>6</sup>

The requirement that primary standards provide an adequate margin of safety was intended to address uncertainties associated with inconclusive scientific and technical information available at the time of standard setting. It was also intended to provide a reasonable degree of protection against hazards that research has not yet identified. See *Lead Industries Association v. EPA*, 647 F.2d 1130, 1154 (D.C. Cir. 1980), cert. denied, 449 U.S. 1042 (1980); *American Petroleum Institute v. Costle*, 665 F.2d 1176, 1186 (D.C. Cir. 1981), cert. denied, 455 U.S. 1034 (1982); *Coalition of Battery Recyclers Ass’n v. EPA*, 604 F.3d 613, 617-18 (D.C. Cir. 2010); *Mississippi v. EPA*, 744 F. 3d 1334, 1353 (D.C. Cir. 2013). Both kinds of uncertainties

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<sup>5</sup> 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.” S. Rep. No. 91-1196, 91st Cong., 2d Sess. 10 (1970).

<sup>6</sup> Under CAA section 302(h) (42 U.S.C. § 7602(h)), effects on welfare include, but are not limited to, “effects on soils, water, crops, vegetation, man-made materials, animals, wildlife, weather, visibility and climate, damage to and deterioration of property, and hazards to transportation, as well as effects on economic values and on personal comfort and well-being.”

1 are components of the risk associated with pollution at levels below those at which human health  
2 effects can be said to occur with reasonable scientific certainty. Thus, in selecting primary  
3 standards that include an adequate margin of safety, the Administrator is seeking not only to  
4 prevent pollution levels that have been demonstrated to be harmful but also to prevent lower  
5 pollutant levels that may pose an unacceptable risk of harm, even if the risk is not precisely  
6 identified as to nature or degree. The CAA does not require the Administrator to establish a  
7 primary NAAQS at a zero-risk level or at background concentration levels, see *Lead Industries*  
8 *v. EPA*, 647 F.2d at 1156 n.51, *Mississippi v. EPA*, 744 F. 3d at 1351, but rather at a level that  
9 reduces risk sufficiently so as to protect public health with an adequate margin of safety.

10 In addressing the requirement for an adequate margin of safety, the EPA considers such  
11 factors as the nature and severity of the health effects involved, the size of the sensitive  
12 population(s), and the kind and degree of uncertainties. The selection of any particular approach  
13 to providing an adequate margin of safety is a policy choice left specifically to the  
14 Administrator’s judgment. See *Lead Industries Association v. EPA*, 647 F.2d at 1161-62;  
15 *Mississippi v. EPA*, 744 F. 3d at 1353.

16 In setting primary and secondary standards that are “requisite” to protect public health  
17 and welfare, respectively, as provided in section 109(b), the EPA’s task is to establish standards  
18 that are neither more nor less stringent than necessary. In so doing, the EPA may not consider the  
19 costs of implementing the standards. See generally, *Whitman v. American Trucking Associations*,  
20 531 U.S. 457, 465-472, 475-76 (2001). Likewise, “[a]ttainability and technological feasibility are  
21 not relevant considerations in the promulgation of national ambient air quality standards.”  
22 *American Petroleum Institute v. Costle*, 665 F. 2d at 1185.

23 Section 109(d)(1) requires that “[n]ot later than December 31, 1980, and at five-year  
24 intervals thereafter, the Administrator shall complete a thorough review of the criteria published  
25 under section [108] and the national ambient air quality standards...and shall make such  
26 revisions in such criteria and standards and promulgate such new standards as may be  
27 appropriate....” Section 109(d)(2) requires that an independent scientific review committee  
28 “shall complete a review of the criteria...and the national primary and secondary ambient air  
29 quality standards...and shall recommend to the Administrator any new...standards and revisions  
30 of existing criteria and standards as may be appropriate....” Since the early 1980s, this  
31 independent review function has been performed by the CASAC of the EPA’s Science Advisory  
32 Board.<sup>7</sup>

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<sup>7</sup> Lists of the CASAC members and of members of the CASAC Sulfur Oxides Panel are available at:  
<https://yosemite.epa.gov/sab/sabpeople.nsf/WebCommitteesSubcommittees/CASAC%20Sulfur%20Oxides%20Panel>

## 1.2.2 History of the Reviews of the Primary NAAQS for SO<sub>x</sub>

The initial air quality criteria for SO<sub>x</sub> were issued in 1969 (34 FR 1988, February 11, 1969). Based on these criteria, the EPA, in initially promulgating NAAQS for SO<sub>x</sub> in 1971, established the indicator as SO<sub>2</sub>. The two primary standards set in 1971 were 0.14 parts per million (ppm) averaged over a 24-hour period, not to be exceeded more than once per year, and 0.03 ppm, as an annual arithmetic mean.

The first review of the air quality criteria and standards for SO<sub>x</sub> was completed in several stages. In the first stage, the EPA released the Air Quality Criteria Document (AQCD) for PM and SO<sub>x</sub> in December 1981, and an addendum presenting information from subsequently available controlled human exposure studies in 1982 (U.S. EPA, 1982a, 1982b). The policy aspects of the air quality criteria, and preliminary exposure analyses were evaluated by OAQPS staff in the 1982 Staff Paper (U.S. EPA, 1982c).

In 1986, the EPA published a second addendum to the 1982 AQCD, presenting newly available evidence from epidemiologic and controlled human exposure studies (U.S. EPA, 1986a). Policy-relevant aspects of the new evidence and staff findings from a companion population exposure assessment were evaluated in a 1986 Addendum to the 1982 Staff Paper (U.S. EPA, 1986b, 1986c). The CASAC reviewed all of these documents and provided advice and recommendations with regard to decisions for the review of the standards. Based on the evidence in the 1982 and 1986 documents, staff evaluations and CASAC recommendations, in 1988, the EPA proposed to retain the existing standards and solicited comment on the alternative of retaining the existing standards while additionally establishing a 1-hour standard of 0.4 ppm to protect against short-term exposures (53 FR 14926, April 26, 1988). In 1992, the American Lung association brought a lawsuit to compel the EPA to review and, if appropriate, revise the primary standards for SO<sub>x</sub>, and the remainder of the review was then completed under court order (59 FR 58962, November 15, 1994; 61 FR 25566, May 22, 1996).

In response to publication of additional relevant controlled human studies on health effects of short-term SO<sub>2</sub> concentrations, the EPA prepared a supplement to the second addendum to the 1982 AQCD (1994 AQCD supplement [U.S. EPA, 1994a]). Policy-relevant aspects of the full body of evidence, including that newly available, along with the 1986 exposure analysis were evaluated in the 1994 Supplement to the 1982 Staff Paper (U.S. EPA, 1994b). In 1994, based on the available evidence, staff evaluations, CASAC advice, and public comment on the 1988 proposal, the EPA re-proposed to retain the existing standards and also solicited comment on retaining the existing standards in combination with one of three policy options to further reduce the health risk posed by exposure to high 5-minute peaks of SO<sub>2</sub> if additional protection were judged to be necessary (59 FR 58958, November 15, 1994). The three alternatives were: (1) Revising the existing primary SO<sub>2</sub> NAAQS by adding a new 5-minute

1 standard of 0.60 ppm SO<sub>2</sub>, not to be exceeded more than once per calendar year; (2) establishing  
2 a new regulatory program under section 303 of the CAA to supplement protection provided by  
3 the existing NAAQS, with a trigger level of 0.60 ppm SO<sub>2</sub>, not to be exceeded more than once  
4 per calendar year; and (3) augmenting implementation of existing standards by focusing on those  
5 sources or source types likely to produce high 5-minute peak concentrations of SO<sub>2</sub>.

6 This review was completed in 1996 with the EPA's decision to retain without revision the  
7 existing standards (61 FR 25566, May 22, 1996). In reaching this decision, the Administrator  
8 concluded, based on the staff exposure analysis, that exposure of individuals with asthma to SO<sub>2</sub>  
9 levels that can reliably elicit adverse health effects was likely a rare event when viewed in the  
10 context of the entire population of people with asthma. As a result, the Administrator judged that  
11 5-minute peaks of SO<sub>2</sub> did not pose a broad public health problem when viewed from a national  
12 perspective, and a 5-minute standard was not promulgated (61 FR 25566, May 22, 1996).

13 In 1996, the American Lung Association and the Environmental Defense Fund  
14 challenged the EPA's decision not to establish a 5-minute standard. On January 30, 1998, the  
15 Court of Appeals for the District of Columbia ("D.C. Circuit") found that the EPA had failed to  
16 adequately explain its determination that no revision to the SO<sub>2</sub> NAAQS was appropriate and  
17 remanded the decision back to EPA for further explanation. Specifically, the court determined  
18 that the EPA had not provided adequate rationale to support the judgment that 5-minute peaks of  
19 SO<sub>2</sub> do not pose a public health problem from a national perspective even though these peaks  
20 will likely cause adverse health impacts in a subset of individuals with asthma. *American Lung*  
21 *Ass'n v. EPA*, 134 F. 3d 388, 392-393 (D.C. Cir. 1998). Following the remand, the EPA  
22 requested that states voluntarily submit 5-minute SO<sub>2</sub> monitoring data for the EPA to use to gain  
23 a better understanding of the magnitude and frequency of high, 5-minute peak SO<sub>2</sub>  
24 concentrations.

25 The next and most recent review of the air quality criteria and primary standards for SO<sub>x</sub>  
26 was completed in 2010 (75 FR 35520, June 22, 2010). As a result of this review, the EPA  
27 promulgated a new 1-hour standard to provide the requisite protection for at-risk populations  
28 such as people with asthma against an array of adverse respiratory health effects related to short-  
29 term SO<sub>2</sub> exposures. The 1-hour standard was set with SO<sub>2</sub> as the indicator based on its common  
30 occurrence in the atmosphere and the predominance of SO<sub>2</sub> studies in the health effects  
31 information for SO<sub>x</sub>. The standard was set at a level of 75 parts per billion (ppb), based on the 3-  
32 year average of the annual 99<sup>th</sup> percentile of 1-hour daily maximum SO<sub>2</sub> concentrations. The  
33 EPA also revoked the then-existing 24-hour and annual primary standards based largely on the  
34 conclusion that the 1-hour standard would also control longer-term average concentrations,  
35 maintaining 24-hour and annual concentrations generally well below the levels of those  
36 standards, and on the lack of evidence indicating the need for such longer-term standards. The

2010 action also addressed the remand by the D.C. Circuit in 1998. The 2010 and prior standards are summarized in Table 1-1.

**Table 1-1. History of the primary national ambient air quality standard(s) for sulfur oxides since 1971.**

Final Rule/Decision	Indicator	Averaging Time	Level	Form
April 30, 1971 (36 FR 8186)	SO <sub>2</sub>	24 hours	140 ppb <sup>a</sup>	one allowable exceedance
		1 year	30 ppb <sup>a</sup>	arithmetic average
May 22, 1996 (61 FR 25566)	Both the 24-hour and annual average standards retained without revision			
June 22, 2010 (75 FR 35520)	SO <sub>2</sub>	1 hour	75 ppb	99 <sup>th</sup> percentile of yearly distribution of 1-hour daily maximums, averaged over 3 years
		24-hour and annual standards revoked		
<sup>a</sup> Although the levels were set in terms of ppm (0.14 ppm for the 24-hour standard and 0.03 ppm for the annual standard), they are shown here in ppb for consistency with units of current standard.				

In conjunction with the 2010 revisions to the standards, the EPA revised the SO<sub>2</sub> ambient air monitoring regulations to require that monitoring agencies using continuous SO<sub>2</sub> methods report the highest 5-minute concentration for each hour of the day; many agencies additionally report all twelve 5-minute concentrations for each hour of the day (75 FR 35554, June 22, 2010; 40 CFR 58.16). The rationale for this requirement was to provide additional monitoring data for use in subsequent reviews of the primary standard, particularly in considering the extent of protection provided by the 1-hour standard against 5-minute peak SO<sub>2</sub> concentrations of concern (75 FR 35554, June 22, 2010).

After publication of the final rule, a number of industry groups and states filed petitions for review arguing (1) that the EPA failed to follow notice-and-comment rulemaking procedures because the proposal did not indicate that EPA was considering changing its method of determining attainment from an air-monitoring approach to a hybrid approach using computer modeling in combination with air monitoring, and (2) that the decision to establish a 1-hour SO<sub>2</sub> NAAQS at 75 ppb was arbitrary and capricious because it was lower than statutorily authorized. The D.C. Circuit rejected these challenges, dismissing the first argument for lack of jurisdiction and denying the petitions with respect to the second argument, explaining that the EPA did not act arbitrarily in setting the 2010 standard. *National Environmental Developmental Association's Clean Air Project v. EPA*, 686 F. 3d 803 (D.C. Cir. 2012). Accordingly, the 2010 standard was upheld. *Id.*

### 1.2.3 Current SO<sub>2</sub> NAAQS Review

In May 2013, the EPA announced the initiation of the current periodic review of the air quality criteria for sulfur oxides and the primary NAAQS for sulfur oxides and issued a call for information in the *Federal Register* (78 FR 27387, May 10, 2013). A wide range of external experts as well as EPA staff representing a variety of areas of expertise (e.g., epidemiology, human and animal toxicology, statistics, risk/exposure analysis, atmospheric science) participated in a workshop, held by the EPA on June 12-13, 2013 in Research Triangle Park, NC. The workshop provided for a public discussion of the key policy-relevant issues around which the EPA has structured the review and of the most meaningful new scientific information that would be available in this review to inform our understanding of these issues.

Building from the workshop discussions, the EPA developed the draft *Integrated Review Plan for the Primary National Ambient Air Quality Standards for Sulfur Dioxide* (IRP, U.S. EPA, 2014) outlining the schedule, process, and key policy-relevant questions that would guide the evaluation of the air quality criteria for SO<sub>2</sub> and the review of the primary NAAQS for SO<sub>x</sub>. The draft IRP was released in March 2014 (79 FR 14035, March 12, 2014) and was the subject of a consultation with the CASAC on April 22, 2014 (79 FR 16325, March 25, 2014). Comments received from the CASAC and the public were considered in the preparation of the final IRP, which was released in October 2014 (U.S. EPA, 2014).

The process for development of the first draft ISA included review of preliminary drafts of key ISA chapters by subject matter experts at a public workshop hosted by the EPA's National Center for Environmental Assessment (NCEA) on June 23-24, 2014 (79 FR 33750, June 12, 2014). Comments received from this review as well as comments from the public and the CASAC on the draft IRP were considered in preparation of the *Integrated Science Assessment for Sulfur Oxides – Health Criteria (External Review Draft – November 2015)*, released in November 2015 (80 FR 73183, November 24, 2015). The first draft ISA was reviewed by the CASAC at a public meeting in January 2016 and a public teleconference in April 2016 (80 FR 79330, December 21, 2015; 80 FR 79330, December 21, 2015; Diez Roux, 2016).

The EPA released the *Integrated Assessment for Sulfur Oxides – Health Criteria (Second External Review Draft – December 2016)* in December 2016 (81 FR 89097), which was reviewed by the CASAC at a public meeting in March 2017 and a public teleconference in June 2017 (82 FR 11449, February 23, 2017; 82 FR 23563, May 23, 2017). Completion of the final ISA is expected in December 2017.

As part of the planning process for development of the REA, the EPA completed the *Review of the Primary National Ambient Air Quality Standard for Sulfur Oxides: Risk and Exposure Assessment Planning Document* (REA Planning Document, U.S. EPA, 2017c) in February 2017 (82 FR 11356), and held a consultation with the CASAC at a public meeting in

1 March 2017 (82 FR 11449). In consideration of CASAC comments at that consultation and  
2 public comments, the EPA developed the draft REA (U.S. EPA, 2017b). The draft REA and this  
3 draft PA are being provided to the CASAC for their review and released to the public for  
4 comment. The CASAC advice and public comments will be considered in completing these  
5 documents.

6 The schedule for completion of this review is governed by a consent decree entered by  
7 the court, which, in relevant part, specifies that the appropriate EPA official issue a final  
8 Integrated Science Assessment addressing human health effects of SO<sub>x</sub> no later than December  
9 14, 2017; sign a notice setting forth its proposed decision concerning its review of the primary  
10 NAAQS for SO<sub>x</sub> no later than May 25, 2018; and sign a notice setting forth its final decision  
11 concerning its review of the primary NAAQS for SO<sub>x</sub> no later than January 28, 2019 (Consent  
12 Decree at 4, *Center for Biological Diversity et al. v. Pruitt*, Case No. 3:16-cv-03796-VC (N.D.  
13 Cal. April 28, 2017), Document No. 37).

### 14 **1.3 GENERAL APPROACH AND ORGANIZATION OF THIS** 15 **DOCUMENT**

16 This draft PA draws on the policy-relevant aspects of the scientific evidence and  
17 quantitative air quality, exposure and risk analyses. With regard to the health effects evidence,  
18 we consider the nature of the key effects associated with SO<sub>2</sub> in ambient air, the types and  
19 magnitudes of exposures associated with effects, and populations at greatest risk, as well as the  
20 uncertainties. Based on this information, we summarize associated potential public health  
21 impacts of SO<sub>2</sub> in ambient air. We additionally consider the magnitude of exposures and risks  
22 estimated in the REA, along with the associated uncertainties. This evaluation supports  
23 preliminary staff conclusions with regard to the key policy-relevant questions for the review,  
24 including whether the currently available information appears to call into question the adequacy  
25 of public health protection afforded by the current standard.

26 Following this introductory chapter, chapter 2 focuses on current air quality, including  
27 sources of SO<sub>2</sub> to ambient air, the ambient monitoring network for SO<sub>2</sub>, and trends and current  
28 conditions. Chapter 3 focuses on the review of the primary NAAQS for SO<sub>x</sub> presenting  
29 background information on the rationale for previous reviews and the approach followed in the  
30 current review. Chapter 3 further discusses the adequacy of the current standards, taking into  
31 account evidence- and exposure-/risk-based considerations, and includes preliminary staff  
32 conclusions. Chapter 3 also identifies key uncertainties and areas for future research.

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## 2 CURRENT AIR QUALITY

This chapter presents a summary of our current understanding of SO<sub>x</sub> in ambient air largely drawn from the more detailed discussion provided in the second draft ISA (second draft ISA, chapter 2). Section 2.1 summarizes the current information on sources and emissions and section 2.2 summarizes current ambient air monitoring methods and networks. Recent concentrations of SO<sub>2</sub> in ambient air are summarized in section 2.3.

### 2.1 SOURCES TO AMBIENT AIR

In this section, we describe the most recently available information on sources and emissions of SO<sub>x</sub> into the ambient air. The section does not provide a comprehensive list of all sources, nor does it provide estimates of emission rates or emission factors for all source categories. Rather, the discussion here is intended to identify the larger source categories, either on a national or local scale, and generally describe their emissions and distribution within the U.S. based on the U.S. EPA 2014 National Emissions Inventory (NEI).

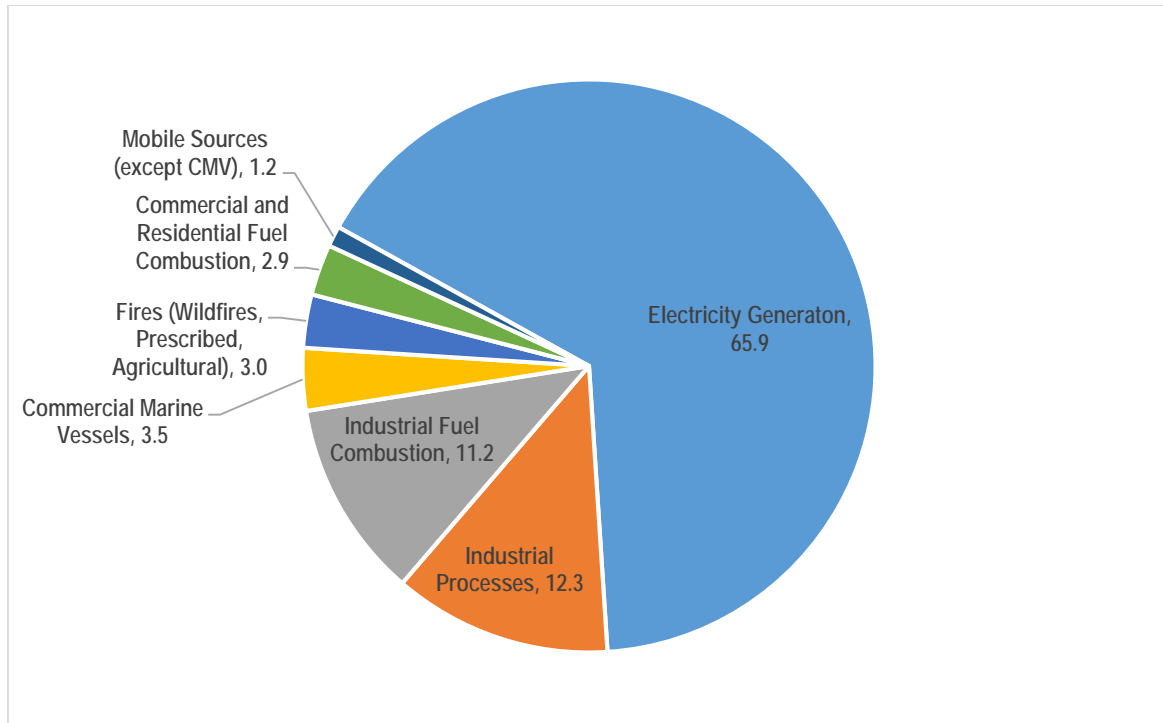
Sulfur oxides are emitted into air from specific sources (e.g., fuel combustion processes) and also formed in the atmosphere from other atmospheric compounds (e.g., as an oxidation product of reduced sulfur compounds, such as sulfides). Sulfur oxides are also transformed in the atmosphere to particulate sulfur compounds, such as sulfates. Sulfur oxides known to occur in the troposphere include SO<sub>2</sub> and sulfur trioxide (SO<sub>3</sub>) (second draft ISA, section 2.3). As a result of rapid atmospheric chemical reactions involving SO<sub>3</sub>, the most prevalent sulfur oxide in the atmosphere is SO<sub>2</sub> (second draft ISA, section 2.3).

Fossil fuel combustion is the main anthropogenic source of SO<sub>2</sub> emissions, while volcanos and landscape fires (wildfires as well as controlled burns) are the main natural sources (second draft ISA, section 2.1).<sup>1</sup> Industrial chemical production, pulp and paper production, natural biological activity (plants, fungi, and prokaryotes), and volcanoes are among many sources of reduced sulfur compounds that contribute, through various oxidation reactions in the atmosphere, to the formation of SO<sub>2</sub> in the atmosphere (second draft ISA, section 2.1). Anthropogenic SO<sub>2</sub> emissions originate primarily from point sources, including coal-fired electricity generating units (EGUs) and other industrial facilities (second draft ISA, section

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<sup>1</sup> The 2008 ISA (U.S. EPA, 2008) described a modeling analysis that estimated SO<sub>2</sub> concentrations for 2001 in the absence of any U.S. anthropogenic emissions of SO<sub>2</sub> (2008 ISA, section 2.5.3). Such concentrations are referred to as United States background or USB. The 2008 ISA analysis estimated USB concentrations of SO<sub>2</sub> to be below 0.01 ppb over much of the U.S., ranging up to a maximum of 0.03 ppb. In the U.S. Northwest, geothermal sources (e.g., volcanoes) were estimated to be responsible for up to 80% of the ambient air concentrations resulting solely from natural sources and sources outside of the U.S. (second draft ISA, section 2.5.5).

2.2.1). The largest SO<sub>2</sub>-emitting sector within the U.S. is electricity generation, of which 97% of SO<sub>2</sub> from electricity generation is from coal combustion, as shown in Figure 2-1. Other anthropogenic sources of SO<sub>2</sub> emissions include industrial fuel combustion and process emissions, industrial processing, commercial marine activity, and fire used in landscape management and agriculture (second draft ISA, section 2.2.1).

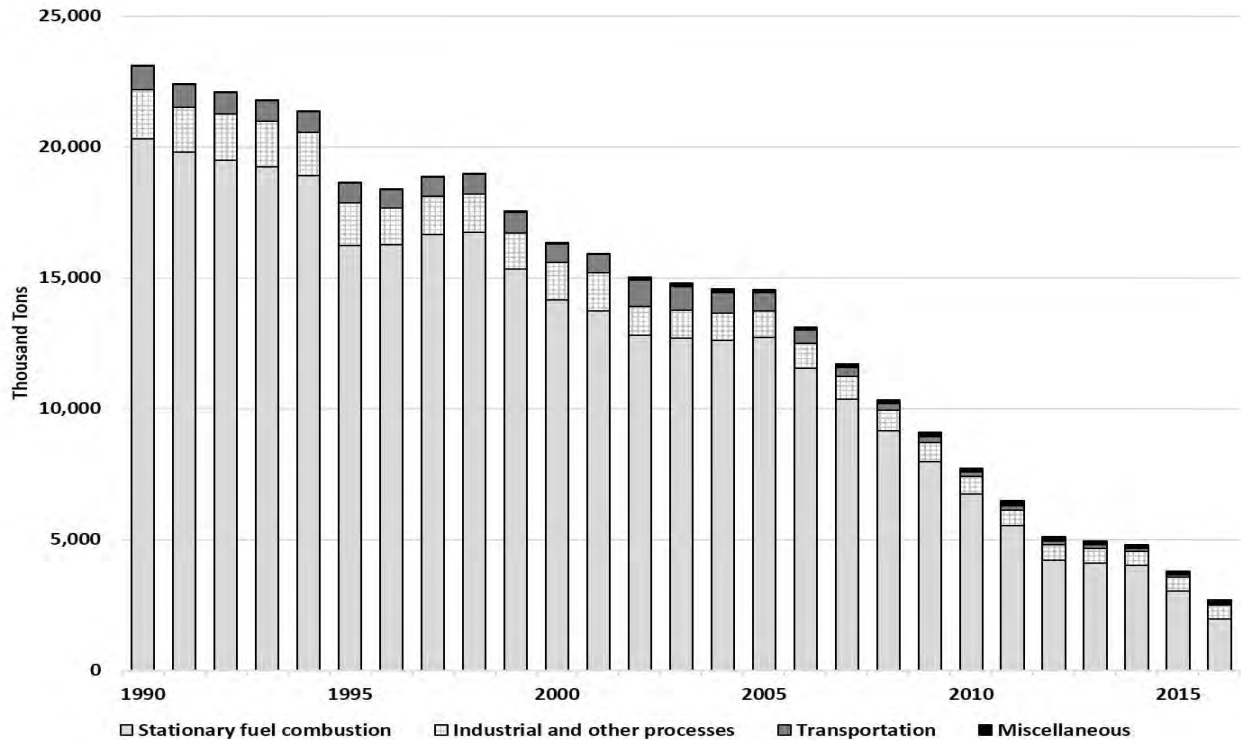


**Figure 2-1. Percent contribution of SO<sub>2</sub> emissions by sector (Source: 2014 NEI).<sup>2</sup>**

Figure 2-2 illustrates the emissions trends from 1990 to 2015. Declines in SO<sub>2</sub> emissions are likely related to the implementation of Clean Air Act national control programs including Phase I and II of the Acid Rain Program, the Clean Air Interstate Rule, and the Cross-State Air Pollution Rule. An additional factor is changes in market conditions, e.g., reduction in energy generation by coal (U.S. EIA, 2017).<sup>3</sup>

<sup>2</sup> Total SO<sub>2</sub> emissions from the 2014 NEI were 4,942,063 tons.

<sup>3</sup> The reduction in energy generation by coal resulted in the use of fuels that emit less SO<sub>2</sub> in energy generation (U.S. EIA, 2016).



**Figure 2-2. National SO<sub>2</sub> emission trends by sector.**

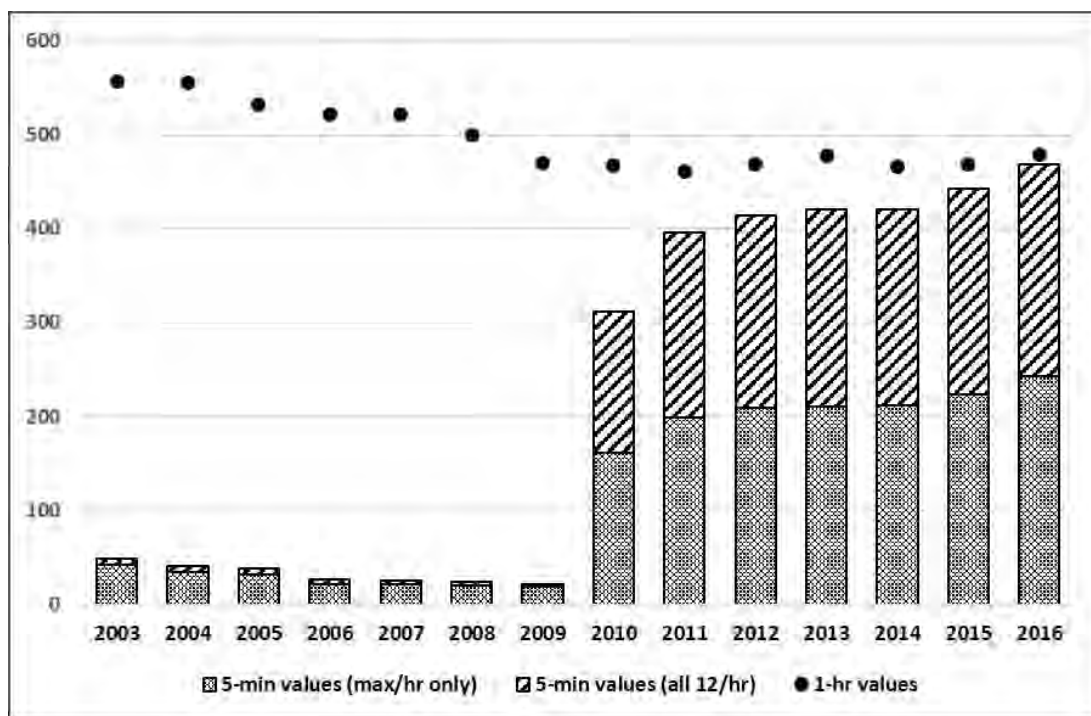
## 2.2 AMBIENT AIR MONITORING METHODS AND NETWORK

To promote uniform enforcement of the air quality standards set forth under the CAA, the EPA has established federal reference methods (FRMs) and federal equivalent methods (FEMs) for ambient air sample collection and analysis. Measurements for determinations of NAAQS compliance must be made with FRMs or FEMs. The current SO<sub>2</sub> monitoring network relies on the automated pulsed ultraviolet fluorescence (UVF) FRM (40 CFR Appendix A-1 to Part 50; 40 CFR Appendix A-2 to Part 50). The UVF method is a continuous automated method that quantifies SO<sub>2</sub> concentrations, providing averages across desired time periods, such as 5-minute and 1-hour averages.

Measurements of SO<sub>2</sub> concentrations in ambient air are collected by networks of FRM monitors, primarily operated by state and local monitoring agencies in the U.S. The main network providing ambient data for NAAQS surveillance monitoring purposes is the State and Local Air Monitoring Stations (SLAMS) network. In 2016, there were 363 SLAMS sites reporting SO<sub>2</sub> concentrations to the Air Quality System (AQS), the EPA's repository for detailed air pollution data. For each SO<sub>2</sub> monitoring site, the SLAMS monitoring agencies report hourly concentrations and either the maximum 5-minute concentration in the hour (one of twelve 5-

minute periods within an hour) or all twelve 5-minute average SO<sub>2</sub> concentrations within the hour.

Five minute SO<sub>2</sub> data have become much more widely available due to regulatory requirements promulgated in 2010 (Figure 2-3).<sup>4</sup> Although 5-minute data were available for fewer than 10% of monitoring sites at the time of the last review, such data (either all 12 values in each hour or just the maximum 5-minute concentrations) are currently available for nearly all of the operating SO<sub>2</sub> sites nationwide, providing a more robust foundation for characterization of 5-minute ambient air concentrations in this review. Further, the newly available monitoring data also include more monitors reporting the 12 consecutive 5-minute concentrations for each hour than were available in the last review (Figure 2-3). Of the monitors reporting 5-minute data in 2016, almost 40% are reporting all twelve 5-minute SO<sub>2</sub> measurements in each hour while about 60% are reporting the maximum 5-minute SO<sub>2</sub> concentration in each hour.<sup>5</sup>



**Figure 2-3. Temporal trend in number of monitors with 5-minute data.**

<sup>4</sup> At SO<sub>2</sub> NAAQS compliance monitoring sites, air monitoring agencies are now required to report, for every hour of the day, the hourly average and either the maximum 5-minute value (one of twelve 5-minute periods) in the hour or all twelve 5-minute averages within the hour (75 FR 35554, June 22, 2010).

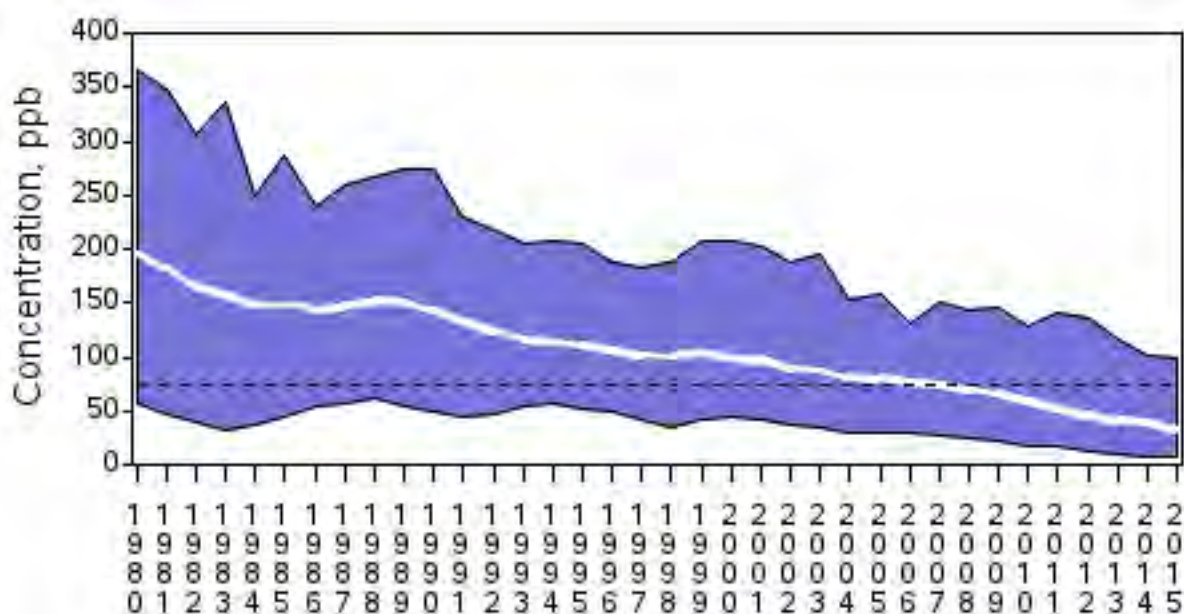
<sup>5</sup> In 2016, three of the sites reported both the continuous 5-minute data and the maximum 5-minute data separately. Therefore, these monitors are included in the count for each of the types of 5-minute monitors.

## 2.3 AMBIENT AIR MONITORING CONCENTRATIONS

This section briefly summarizes trends and current conditions based on recent ambient air monitoring data.

### 2.3.1 Trends

Ambient air concentrations of SO<sub>2</sub> in the U.S. have declined substantially from 1980 to 2015. Figure 2-4 illustrates this decline in terms of the distribution of 3-year average of annual 99<sup>th</sup> percentile daily maximum 1-hour concentrations<sup>6</sup> at 45 monitoring sites that have been operating across this period. The average of this dataset has declined by more than 83% over the 35-year period. Over the past 15 years, a larger dataset of 227 sites operating from 2000-2015 also indicates a decline, which is on the order of 67% for the average of that dataset (Figure 2-5).<sup>7</sup> Daily maximum 5-minute SO<sub>2</sub> concentrations have also consistently declined over time from 2011 to 2015 (Figure 2-6).<sup>8</sup>

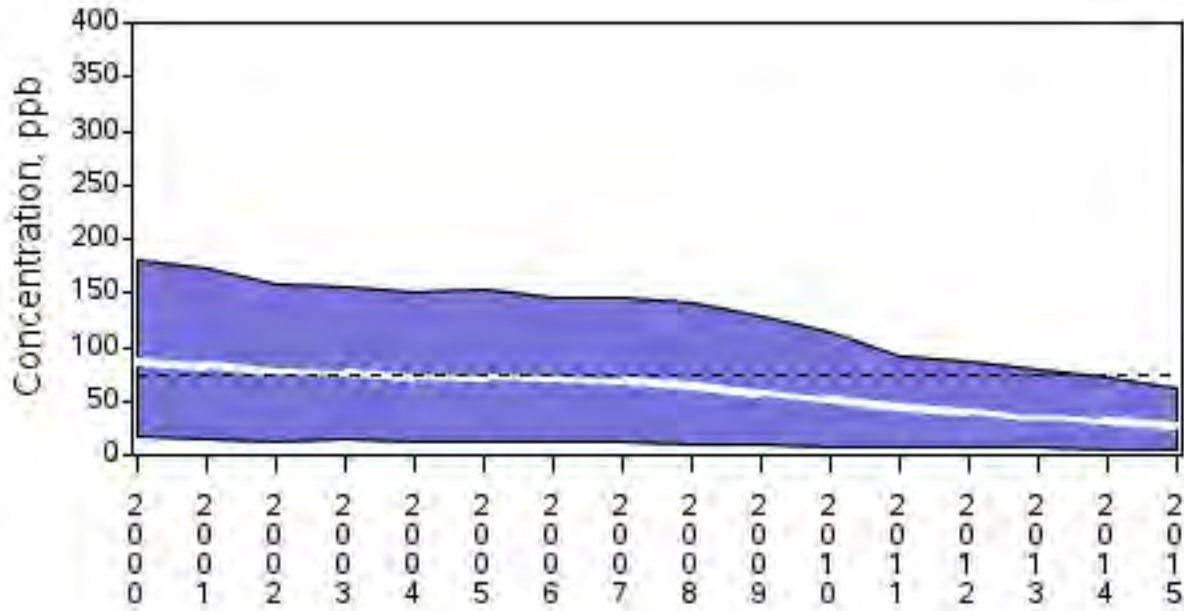


**Figure 2-4. National temporal trend in SO<sub>2</sub> concentrations: 1980-2015 (45 sites).** Three-year average of annual 99<sup>th</sup> percentile of daily maximum 1-hour concentrations. (Note: Dashed line indicates the current standard [75 ppb].)

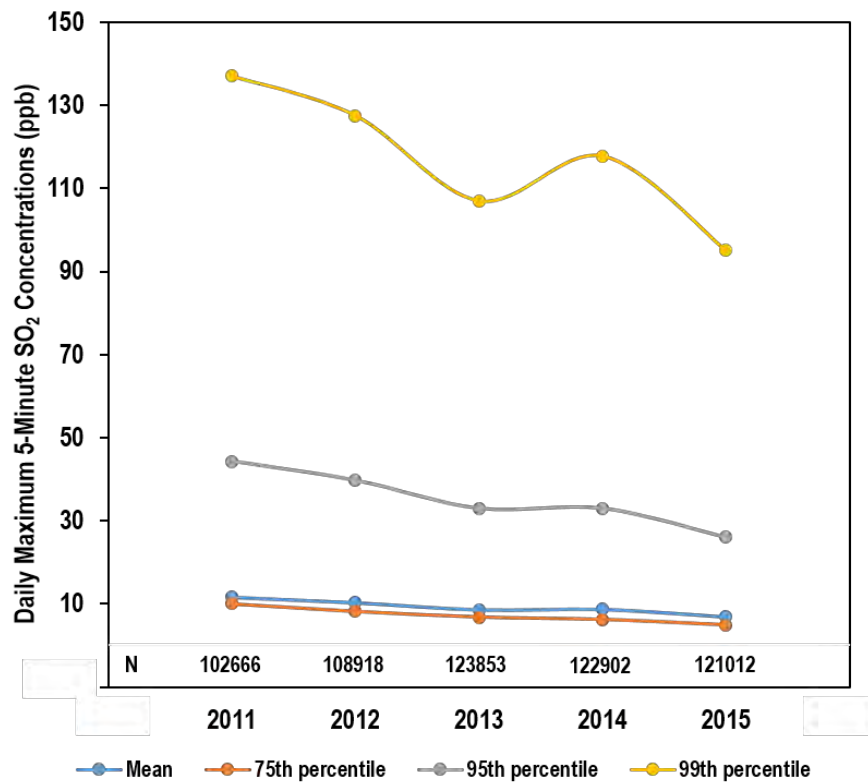
<sup>6</sup> The form of the current 1-hour SO<sub>2</sub> NAAQS is the 99<sup>th</sup> percentile of yearly distribution of 1-hour daily maximums, averaged over 3 years.

<sup>7</sup> In Figures 2-4 and 2-5, the year on the x-axis represents the last year of the 3 year average (e.g., 2015 represents the average of 2013-2015).

<sup>8</sup> In Figure 2-6, the number of sites with monitors for 2011, 2012, 2013, 2014, and 2015 were 301, 321, 366, 359, and 352, respectively.



**Figure 2-5. Temporal trend in SO<sub>2</sub> concentrations: 2000-2015 (227 sites).** Three-year average of annual 99th percentile of daily maximum 1-hour concentrations. (Note: Dashed line indicates the current standard [75 ppb]).

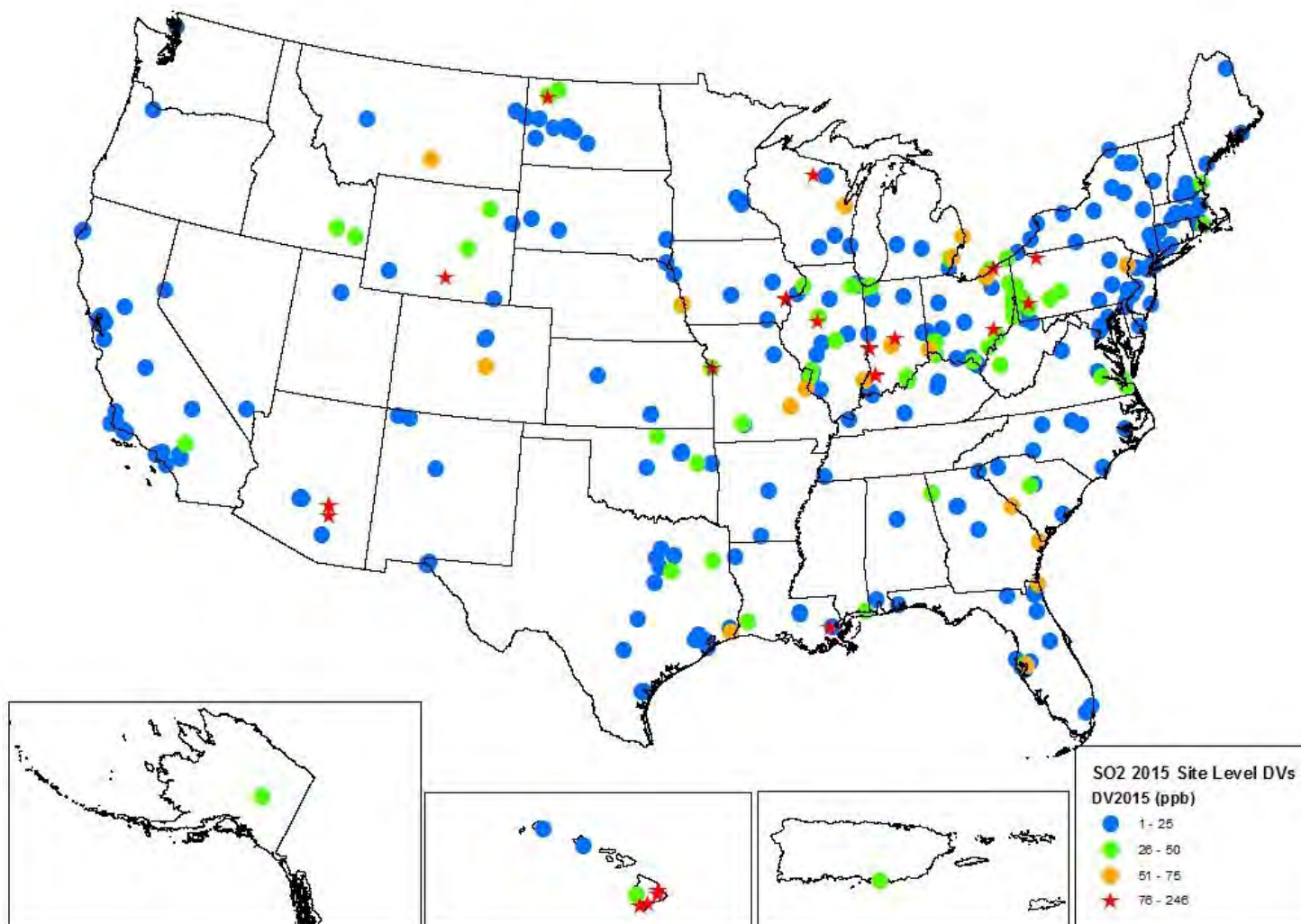


**Figure 2-6. Temporal trend in daily maximum 5-minute SO<sub>2</sub> concentrations: 2011-2015.** (N = number of measurements)

## **2.3.2 Current Concentrations**

### **2.3.2.1 Geographic Variation in Concentrations**

Concentrations of SO<sub>2</sub> vary across the U.S. and tend to be higher in areas with sources having relatively higher SO<sub>2</sub> emissions (e.g., EGUs). Consistent with the locations of larger SO<sub>2</sub> sources, higher concentrations are primarily located in the eastern half of the continental U.S., especially in the Ohio River valley, upper Midwest, and along the Atlantic coast (Figure 2-7). The point source nature of SO<sub>2</sub> emissions contribute to the relatively high spatial variability of SO<sub>2</sub> concentrations compared with pollutants such as ozone (O<sub>3</sub>) (second draft ISA, section 3.2.3). Another contributing factor to the spatial variability is the dispersion and oxidation of SO<sub>2</sub> in the atmosphere, resulting in decreasing SO<sub>2</sub> concentrations with increasing distance from the source. Sulfur oxides emitted from point sources tends to travel away from the emissions source as a plume, which may or may not impact large portions of surrounding populated areas depending on meteorological conditions and terrain.



**Figure 2-7. Concentrations of SO<sub>2</sub> in terms of the current standard (3-year average of annual 99<sup>th</sup> percentile daily maximum 1-hour concentrations) at sites with datasets meeting completeness requirements for 2013-2015.**

### 2.3.2.2 Seasonal and Diel Variability in Concentrations

Recent (2013-2015) data indicate that 1-hour daily maximum SO<sub>2</sub> concentrations vary across seasons, with the greatest variations seen in the upper percentile concentrations (versus average or lower percentiles) for each season (second draft ISA, section 2.5.3.2). This is seen in the data presented for six urban areas in the draft ISA<sup>9</sup> (second draft ISA, section 2.5.3.2). This variation, along with month-to-month variations in 1-hour daily maximum SO<sub>2</sub> concentrations also presented in the ISA, were generally consistent with month-to-month emissions patterns and the expected atmospheric chemistry of SO<sub>2</sub> for a given season. For example, “summertime minima, observed in the New York City, NY and Houston, TX, sites may correspond to enhanced oxidation of SO<sub>2</sub> to SO<sub>4</sub><sup>2-</sup> by photochemically derived atmospheric oxidants that are more prevalent during the humid summer (Khoder, 2002)” (second draft ISA, p. 2-55). The differences in seasonal pattern (as well as magnitude) of concentrations among areas studied indicate the variability of SO<sub>2</sub> concentrations across local and regional scales (second draft ISA, section 2.5.3.2).

Consistent with the nationwide diel patterns reported in the last review, 1-hour average and 5-minute hourly maximum SO<sub>2</sub> concentrations for 2013-2015 in all six urban areas evaluated were generally low during nighttime and approached maxima values during daytime hours (second draft ISA, section 2.5.3.3, Figures 2-23 and 2-24). The timing and duration of daytime maxima in the six sites evaluated were likely related to a combination of source emissions and meteorological parameters (second draft ISA, section 2.5.3.3; U.S. EPA 2008, section 2.5.1). For example, SO<sub>2</sub> emitted from elevated point sources (e.g., power plants and industrial sources) may be entrained into the mixed boundary layer, which expands during the day with rising surface temperatures (U.S. EPA 2008, section 2.5.1, Figures 2-23 and 2-24).

### 2.3.2.3 Relationship Between 1-hour and 5-minute Concentrations

Peak concentrations within a plume of SO<sub>2</sub> downwind from, but relatively nearby to, a source can greatly exceed mean concentrations across the plume (second draft ISA, section 2.5.4). Further, measured 5-minute concentrations at a particular location can be much higher than the average concentration at the same location for the associated hour. However, as emissions travel further downwind and experience ever increasing dispersion, these differences lessen both spatially and temporally. This can contribute to greater spatial and temporal

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<sup>9</sup> The six locations evaluated are: Cleveland, OH, Pittsburgh, PA, New York City, NY, St. Louis, MO-IL, Houston, TX, and Gila County, AZ (second draft ISA, section 2.5.2.2). These six locations were based on (1) their relevance to current health studies (i.e., areas with peer-reviewed, epidemiologic analysis), (2) the existence of four or more monitoring sites located within the area boundaries, and (3) the presence of several diverse SO<sub>2</sub> sources within a given focus area boundary.

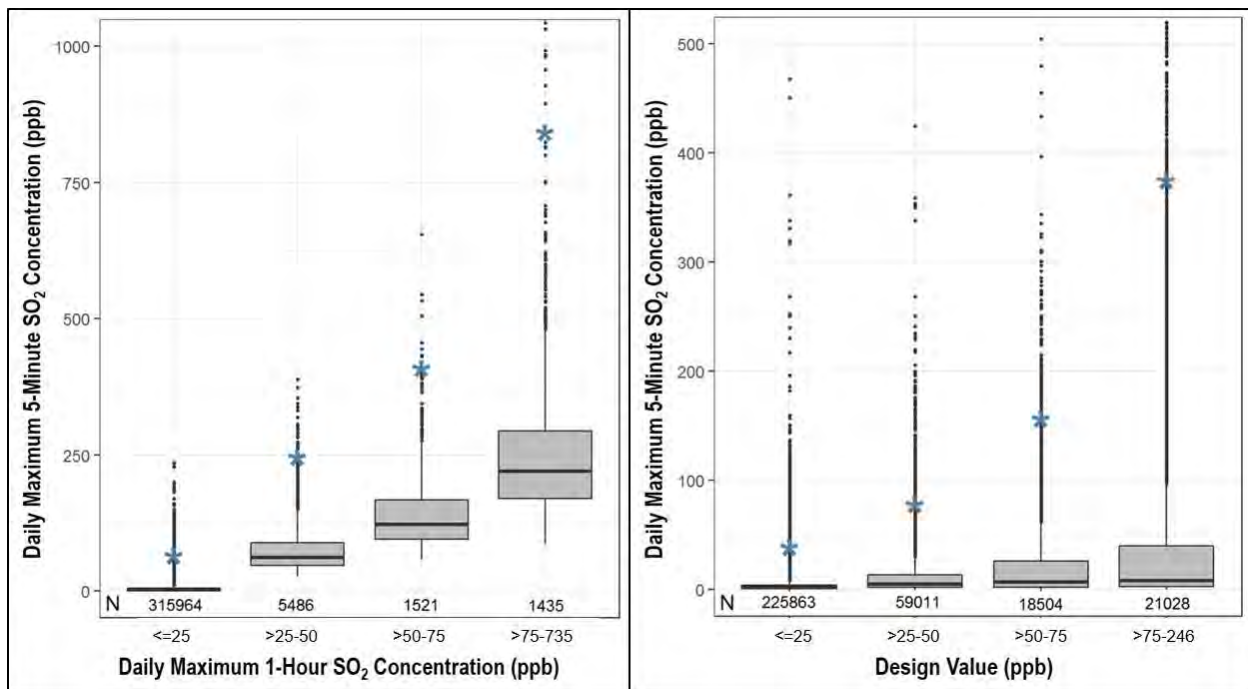
1 variability in 5-minute than in 1-hour concentrations, as is seen in the six urban locations  
2 evaluated in the ISA (second draft ISA, p. 2-52).

3       Using monitoring data from 2013-2015, Figure 2-8 illustrates the general pattern of lower  
4 5-minute concentrations with lower 1-hour concentrations. The left panel of Figure 2-8 shows  
5 that across the monitors meeting data completeness criteria, on days when the maximum 1-hour  
6 concentrations are relatively low, the maximum 5-minute concentrations are also relatively low.  
7 Similarly, as shown in the right panel of Figure 2-8, at sites with relatively lower design values,<sup>10</sup>  
8 the distribution of maximum 5-minute concentrations is also lower. This is documented by the  
9 distinct reduction in 99th percentile daily maximum 5-minute concentrations at lower design  
10 values. For example, in areas with design values at or below the current standard (75 ppb), 99.9  
11 percent of daily maximum 5-minute concentrations are at or below approximately 150 ppb.<sup>11</sup>  
12 This contrasts with the much higher distribution of daily maximum 5-minute concentrations at  
13 sites with design values exceeding the current standard. The 99th percentile of these daily  
14 maximum 5-minute concentrations is 365 ppb, meaning that one percent of the days at these sites  
15 has a maximum 5-minute concentration above 365 ppb (i.e., 210 occurrences).

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<sup>10</sup> The design value (DV) for the standard is the metric used to determine whether areas meet or exceed the NAAQS. A design value is a statistic that describes the air quality status of a given area relative to the NAAQS. Design values are considered to be valid if the monitoring data used to calculate them meet the regulatory completeness criteria which for SO<sub>2</sub> require four quarters of all three years of the period to have data for at least 75 percent of the sampling days (40 CFR 50.17 and appendix T to Part 50).

<sup>11</sup> Additional information related to data in Figure 2-8 is presented in Appendix B.



**Figure 2-8. Distributions of daily maximum 5-minute concentrations during 2013-2015.** Left panel presents varying distributions with varying daily maximum 1-hour concentrations. Right panel presents varying distributions with varying design values; the last bin (>75 ppb) presents data for sites not meeting the current standard. (Note: The values represented in the boxplots are the 25<sup>th</sup> percentile, the median, and the 75<sup>th</sup> percentile. The asterisk represents the 99<sup>th</sup> percentile.)

Analyses of the current monitoring dataset, expanded since the last review, provide information on the occurrence of daily maximum 5-minute concentrations of interest at monitors having differing design values. For example, analysis of these data for the years 2013 to 2015 indicates that among monitors with design values meeting the current standard (i.e., at or below 75 ppb), the vast majority have zero days with a daily maximum 5-minute concentration above 100 ppb or even 400 ppb. Among the few monitors with any days recording a 5-minute concentrations above 400 ppb, the maximum number of such days in a year was five; for monitors with any days recording 5-minute concentrations above 200 ppb, the maximum number of such days/year was 22 (Appendix C, Figures C-2 and C-4, lower panel).

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21 [secondarystandards-planning-documents-current](https://www.epa.gov/naaqs/nitrogen-dioxide-no2-and-sulfur-dioxide-so2-secondarystandards-planning-documents-current)

# 3 REVIEW OF THE PRIMARY STANDARD FOR SULFUR OXIDES

This chapter evaluates the policy implications of the key scientific and technical information in the second draft ISA and draft REA. This evaluation is based on consideration of the available body of evidence assessed in the second draft ISA and of quantitative analyses of SO<sub>2</sub> air quality, exposures and risks presented in the draft REA and in this document. Based on this information, the staff offer preliminary conclusions regarding each of the critical elements of the standard, including indicator, averaging time, form, and level. The final PA will also be informed by the advice and recommendations received from the CASAC during its review of the draft PA, and by public comments received on the draft document. The final PA is designed to help the Administrator in considering the currently available scientific and risk information and formulating judgments regarding the adequacy of the current primary standard.<sup>1</sup>

## 3.1 APPROACH

Staff's approach in this review of the primary standard for SO<sub>x</sub> takes into consideration the approaches used in the previous review. The past and current approaches described below are both based, most fundamentally, on using the EPA's assessment of the current scientific evidence and associated quantitative analyses to inform the Administrator's judgment regarding a primary standard for SO<sub>x</sub> that is requisite to protect public health with an adequate margin of safety. In reaching conclusions on options for the Administrator's consideration, we note that the final decision to retain or revise the current SO<sub>2</sub> primary standard is a public health policy judgment to be made by the Administrator.

The final decision by the Administrator will draw upon the available scientific evidence for SO<sub>2</sub>-attributable health effects, and on quantitative analyses of population exposures and health risks, including judgments about the appropriate weight to assign the range of uncertainties inherent in the evidence and analyses. Therefore, in developing conclusions in this draft PA, we are mindful that the Administrator's judgments on the standard will reflect an interpretation of the available scientific evidence and exposure/risk information in consideration of the strengths and limitations of that evidence and information. Our general approach to

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<sup>1</sup> The basic elements of a standard include the indicator, averaging time, form, and level. The indicator defines the chemical species or mixture to be measured in the ambient air for the purpose of determining whether an area attains the standard. The averaging time defines the period over which air quality measurements are to be obtained and averaged or cumulated. The form of a standard defines the air quality statistic that is to be compared to the level of the standard in determining whether an area attains the standard. The level of the standard defines the air quality concentration used for that purpose (i.e., an ambient air concentration of the indicator).

1 informing these judgments, discussed more fully below, recognizes that the available health  
2 effects evidence reflects a continuum from relatively higher SO<sub>2</sub> concentrations, at which  
3 scientists generally agree that health effects are likely to occur, through lower concentrations at  
4 which the likelihood and magnitude of a response become increasingly uncertain. This approach  
5 is consistent with the requirements of sections 108 and 109 of the CAA, as well as with how the  
6 EPA and the courts have historically interpreted the Act. These provisions require the  
7 Administrator to establish primary standards that in the Administrator's judgment are requisite to  
8 protect public health with an adequate margin of safety. In so doing, the Administrator seeks to  
9 establish standards that are neither more nor less stringent than necessary for this purpose. The  
10 Act does not require that primary standards be set at a zero-risk level, but rather at a level that  
11 reduces risk sufficiently so as to protect public health with an adequate margin of safety.<sup>2</sup>

12 Section 3.1.1 below summarizes the approach used in the last review of the primary  
13 NAAQS for SO<sub>x</sub> and section 3.1.2 summarizes the general approach for the current review.

### 14 **3.1.1 Approach in the Previous Review**

15 The last review of the primary NAAQS for SO<sub>x</sub> was completed in 2010 and resulted in  
16 substantial revisions to the standards (75 FR 35520, June 22, 2010). In consideration of the  
17 evidence of respiratory effects in people with asthma in response to exposures as short as five  
18 minutes, the EPA established a new short-term standard to provide increased protection for this  
19 at-risk group and other potentially at-risk populations<sup>3</sup> against an array of adverse respiratory  
20 effects that have been linked to short-term SO<sub>2</sub> exposures in both controlled human exposure and  
21 epidemiologic studies (75 FR 35525, June 22, 2010; 2008 ISA, section 5.5). Specifically, the  
22 EPA replaced the then-existing 24-hour standard with a short-term standard defined by the 3-  
23 year average of the 99<sup>th</sup> percentile of the yearly distribution of 1-hour daily maximum SO<sub>2</sub>  
24 concentrations, with a level of 75 ppb. In addition to replacing the 24-hour standard with a new  
25 1-hour standard, the EPA revoked the then-existing annual standard, based largely on the lack of  
26 sufficient health evidence to support a long-term standard and a recognition that a 1-hour  
27 standard set at 75 ppb would have the effect of generally maintaining annual SO<sub>2</sub> concentrations  
28 well below the level of the revoked annual standard (75 FR 35550, June 22, 2010).

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<sup>2</sup> The four basic elements of the NAAQS (indicator, averaging time, level and form) are considered collectively in evaluating the health protection afforded by the current standard.

<sup>3</sup> As used here and similarly throughout the document, the term *population* refers to persons having a quality or characteristic in common, such as a specific pre-existing illness or a specific age or lifestage. A lifestage refers to a distinguishable time frame in an individual's life characterized by unique and relatively stable behavioral and/or physiological characteristics that are associated with development and growth. Identifying at-risk populations includes consideration of intrinsic (e.g., genetic or developmental aspects) or acquired (e.g., disease or smoking status) factors that increase the risk of health effects occurring with exposure to sulfur oxides as well as extrinsic, nonbiological factors, such as those related to socioeconomic status, reduced access to health care, or exposure.

1 The emphasis in the 2010 review on short-term exposures of people with asthma  
2 reflected the health effects evidence that has expanded in this area over the four decades since  
3 the then-existing 24-hour and annual standards were set in 1971. A key element of the expanded  
4 evidence base was a series of controlled human exposure studies which documented effects on  
5 lung function in people with asthma exposed, while at moderate or greater levels of exertion, for  
6 periods as short as five minutes. In the 2010 review, the EPA also conducted quantitative  
7 analyses of air quality data, including 5-minute ambient air measurements, and of potential  
8 exposures for people with asthma. Consideration of these key aspects of the evidence and  
9 quantitative analyses informed the decision in the 2010 review, which additionally addressed the  
10 court remand<sup>4</sup> to the EPA of the EPA's 1996 decision to retain the 1971 standards without  
11 revision.

12 The evidence-based and quantitative assessments performed for the 2010 review focused  
13 particularly on the issue of exposures to SO<sub>2</sub> in ambient air of a duration as short as five minutes  
14 (2008 ISA; 2009 REA). The quantitative analyses documented in the REA included  
15 characterizations of the likelihood of people with asthma being exposed (while they were at  
16 elevated exertion<sup>5</sup>) to concentrations of SO<sub>2</sub> from ambient air of a magnitude documented to  
17 elicit decrements in lung function (2009 REA). These analyses were performed both for air  
18 quality conditions associated with just meeting the then-existing standards and for conditions  
19 associated with just meeting potential alternative standards. The REA additionally included air  
20 quality analyses that explored the extent to which potential alternative standards with 1-hour, 24-  
21 hour, and annual averaging times might be expected to control 5-minute ambient air  
22 concentrations (2009 REA, section 7.3). Together the evidence-based and quantitative  
23 assessments informed the policy options considered by the Administrator in that review.  
24 Considerations, conclusions and judgments by the Administrator that provided the basis for her  
25 decisions in the 2010 review are summarized below.

### 26 **3.1.1.1 Considering the Need for Revision**

27 The conclusions reached by the Administrator in the last review were based on the  
28 extensive body of scientific evidence on SO<sub>2</sub>-related health effects and quantitative analyses of  
29 air quality, exposure and risk. In her conclusion on the adequacy of the then-existing standards,

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<sup>4</sup> See *Am. Lung Ass'n v. EPA*, 134 F.3d 388 (D.C. Cir. 1998) (remanding the 1996 decision to EPA).

<sup>5</sup> The phrase “elevated ventilation” (or “moderate or greater exertion”) was used in the 2009 REA and Federal Register notices in the last review to refer to activity levels that in adults would be associated with ventilation rates at or above 40 liters per minute; an equivalent ventilation rate was derived in order to identify corresponding rate for the range of ages and sizes of the simulated populations (2009 REA, section 4.1.4.4). Accordingly, this phrase is used in this draft PA when referring to the REA from the last review. Otherwise, however, the draft REA and draft PA for this review generally uses the phrase “elevated ventilation” to refer to the same occurrence.

1 which were set in 1971, the Administrator emphasized the evidence and quantitative analyses  
2 concerning 5-minute exposures. The Administrator gave particular attention to the robust  
3 evidence base, comprised of findings from controlled human exposure, epidemiologic, and  
4 animal toxicological studies that collectively were judged “sufficient to infer a causal  
5 relationship” between short-term SO<sub>2</sub> exposures ranging from 5 minutes to 24 hours and  
6 respiratory morbidity (75 FR 35535, June 22, 2010). The “definitive evidence” for this  
7 conclusion came from studies of 5- to 10-minute controlled exposures that reported respiratory  
8 symptoms and decreased lung function in exercising individuals with asthma (2008 ISA, section  
9 5.2). Supporting evidence was provided by epidemiologic studies of a broader range of  
10 respiratory outcomes, with uncertainty noted about the magnitude of the study effect estimates,  
11 quantification of the exposure concentration-response relationship, potential confounding by co-  
12 pollutants, and other areas (75 FR 35535-35536, June 22, 2010; 2008 ISA, section 5.3).

13 In the controlled human exposure studies of exercising individuals with asthma, a dose-  
14 response relationship was documented, with both the percentage of individuals affected and the  
15 severity of the response increasing with increasing SO<sub>2</sub> concentrations (75 FR 35525, June 22,  
16 2010). The evidence from these studies documents the occurrence of SO<sub>2</sub>-related decrements in  
17 lung function based on reductions in forced expiratory volume (FEV<sub>1</sub>) and increases in specific  
18 resistance of the airways (sRaw). Moderate<sup>6</sup> or greater decrements in lung function were reported  
19 in response to short (5- to 10-minute) exposures to concentrations as low as 200 to 300 ppb in  
20 approximately 5-30% of exercising individuals with asthma. In response to exposures at or above  
21 400 ppb, approximately 20-60% experienced such decrements, frequently accompanied by  
22 respiratory symptoms; in many studies, responses at these concentrations were often statistically  
23 significant at the group mean level<sup>7</sup> (75 FR 35525-35526, June 22, 2010).

24 In reaching conclusions regarding the significance of the reported responses to the 5- to  
25 10-minute controlled exposures, the Administrator considered guidelines from the American  
26 Thoracic Society (ATS), the CASAC’s written advice and recommendations, and judgments  
27 made by the EPA in considering similar effects in previous NAAQS reviews (75 FR 35526 and

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<sup>6</sup> In assessments for NAAQS reviews, the lung function responses described as indicative of a moderate functional response include increases in sRaw of at least 100% (e.g., 2008 ISA; U.S. EPA, 1994, Table 8; U.S. EPA, 1996, Table 8-3). The moderate category has also generally included reductions in FEV<sub>1</sub> of 10 to 20% (e.g., U.S. EPA, 1996, Table 8). For the 2008 ISA, the midpoint of that range (15%) was used to indicate a moderate response. A focus on 15% reduction in FEV<sub>1</sub> is also consistent with the relationship observed between sRaw and FEV<sub>1</sub> responses in the Linn et al. studies for which “a 100% increase in sRaw roughly corresponds to a 12 to 15% decrease in FEV<sub>1</sub>” (U.S. EPA, 1994, p. 20). Thus, in the 2008 review, moderate or greater SO<sub>2</sub>-related bronchoconstriction or decrements in lung function referred to the occurrence of at least a doubling in sRaw or at least a 15% reduction in FEV<sub>1</sub> (2008 ISA, p. 3-5).

<sup>7</sup> At concentrations of 400 to 500 ppb, the 2008 ISA notes that the evidence shows “stronger evidence with some statistically significant increases in respiratory symptoms,” and at 600 ppb to 1 ppm, the 2008 ISA notes the evidence to show “clear and consistent increases in SO<sub>2</sub> induced respiratory symptoms” (2008 ISA, Table 3-1).

1 35536, June 22, 2010). Based on these considerations, the Administrator judged that the effects  
2 reported in exercising people with asthma following 5- to 10-minute SO<sub>2</sub> exposures at or above  
3 200 ppb, especially at or above 400 ppb, can result in adverse health effects (75 FR 35536, June  
4 22, 2010). In so doing, she recognized that effects reported for exposures below 400 ppb are  
5 appreciably less severe than those at and above 400 ppb (75 FR 35547, June 22, 2010).

6 In applying the health effects evidence to her consideration of the adequacy of the then-  
7 existing standards, the Administrator gave particular attention to the quantitative analyses that  
8 evaluated the potential for exercising individuals with asthma to experience exposures of a  
9 magnitude associated with adverse effects under air quality conditions that just met the then-  
10 existing standards. In addition to comparison of 5-minute air concentrations in 40 U.S. counties  
11 to 5-minute concentrations of potential concern (benchmark concentrations ranging from 100-  
12 400 ppb), the analyses included a population exposure-based assessment in two study areas, St.  
13 Louis, MO and Greene County, MO. Five-minute exposure concentrations were estimated for  
14 people with asthma while at elevated exertion levels. The 5-minute exposure concentrations were  
15 compared to benchmark concentrations, and also used to estimate the risk of lung function  
16 decrements in simulated at-risk populations. Among these analyses, the Administrator  
17 emphasized those that utilized the 5-minute benchmark concentrations that were derived from  
18 the controlled human exposure evidence and ranged from 100 ppb to 400 ppb. Based on her  
19 judgments regarding the significance of effects associated with 5-minute concentrations at or  
20 above 200 ppb and 400 ppb, the Administrator considered results of comparisons of exposure  
21 estimates to those benchmark concentrations to be particularly important, giving greater  
22 emphasis to those at or above 400 ppb (75 FR 35547, June 22, 2010).

23 The exposure-based assessment estimated the portion of the population with asthma in  
24 these two areas that would be expected to experience 5-minute exposures at or above 400 ppb  
25 and 200 ppb while engaged in activities causing them to be at elevated exertion levels. The  
26 Administrator particularly noted the exposure analysis results for the St. Louis case study. This  
27 analysis estimated that for air quality simulated to just meet the then-existing standards,  
28 substantial percentages of children with asthma at moderate or greater exertion<sup>8</sup> would be  
29 exposed, at least once annually, to air quality exceeding the 200 ppb and 400 ppb 5-minute  
30 benchmarks (75 FR 35536, June 22, 2010). The Administrator judged these 5-minute exposures  
31 to be significant from a public health perspective due to their estimated frequency:  
32 approximately 24% of children with asthma in St. Louis were estimated to be exposed while at  
33 moderate or greater exertion at least once per year to air quality exceeding the 5-minute 400 ppb

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<sup>8</sup> In the 2009 REA, an equivalent ventilation rate of 22 L/min-m<sup>2</sup> was identified as the minimum value to reflect “moderate” or greater exertion that would correspond to the elevated ventilation rate for the exercising subjects in the controlled human exposure studies, which was 40-50 L/min (2009 REA, p. 236).

1 benchmark. Additionally, approximately 73% of children with asthma in St. Louis at moderate or  
2 greater exertion were estimated to be exposed at least once per year to air quality exceeding the  
3 5-minute 200 ppb benchmark (75 FR 35536, June 22, 2010).

4 The Administrator also took note of the CASAC conclusion that the then-existing  
5 standards did not adequately protect public health. Specifically, the CASAC advised that: “the  
6 current 24-hour and annual standards are not adequate to protect public health, especially in  
7 relation to short-term exposures to SO<sub>2</sub> (5-10 minutes) by exercising asthmatics” (Samet, 2009,  
8 p. 15). Based on all of the considerations summarized above, the Administrator concluded that  
9 the then-existing 24-hour and annual primary standards were not providing the requisite  
10 protection of public health with an adequate margin of safety. In considering approaches to  
11 revising the standards, the Administrator concluded it to be appropriate to set a new standard that  
12 would provide requisite protection with an adequate margin of safety to people with asthma at  
13 elevated ventilation and that would afford protection from the adverse health effects of 5-minute  
14 to 24-hour SO<sub>2</sub> exposures (75 FR 35536, June 22, 2010).

### 15 **3.1.1.2 Approach for Considering Revisions to the Standards**

16 With regard to revisions to provide requisite public health protection, the Administrator  
17 concluded it was appropriate to set a 1-hour SO<sub>2</sub> standard at a level of 75 ppb based on the 3-  
18 year average of the 99<sup>th</sup> percentile of the yearly distribution of 1-hour daily maximum  
19 concentrations. The rationale and approach for selecting the 1-hour standard is presented below  
20 in terms of the individual elements of a NAAQS: indicator, averaging time, form, and level.

#### 21 **3.1.1.2.1 Indicator**

22 In reaching her decision on the indicator for the new standard, the Administrator  
23 considered the conclusions of the ISA and REA, as well as advice from the CASAC and public  
24 comments (75 FR 35536, June 22, 2010). The EPA continued to focus on SO<sub>2</sub> as the most  
25 appropriate indicator for gaseous sulfur oxides because the available scientific information  
26 regarding health effects was overwhelmingly indexed by SO<sub>2</sub>. Although the presence of gaseous  
27 SO<sub>x</sub> species other than SO<sub>2</sub> in ambient air had been recognized, no alternative to SO<sub>2</sub> had been  
28 advanced as a more appropriate surrogate for ambient gaseous SO<sub>x</sub> (75 FR 35536, June 22,  
29 2010). Controlled human exposure studies and animal toxicological studies provided specific  
30 evidence for health effects following exposures to SO<sub>2</sub>, and epidemiologic studies typically  
31 reported effects associated with SO<sub>2</sub> concentrations. Based on the information available in the  
32 last review and consistent with the views of the CASAC that “for indicator, SO<sub>2</sub> is clearly the  
33 preferred choice” (Samet, 2009, p. 14), the Administrator concluded it was appropriate to  
34 continue to use SO<sub>2</sub> as the indicator for a standard that was intended to address effects associated  
35 with exposure to SO<sub>2</sub>, alone or in combination with other gaseous sulfur oxides (75 FR 35536,

June 22, 2010). In so doing, the EPA recognized that measures leading to reductions in population exposures to SO<sub>2</sub> will also likely reduce exposures to other sulfur oxides (75 FR 35536, June 22, 2010).

#### **3.1.1.2.2 Averaging Time**

With regard to the setting of the new standard, the Administrator agreed with the staff conclusion, based on conclusions in the ISA, advice from the CASAC, and air quality analyses, that the standard should be set to provide protection from short-term exposures of 5 minutes to 24 hours (75 FR 35539, June 22, 2010). Based on air quality analyses presented in the REA, the Administrator judged that the requisite protection from 5- to 10-minute exposure events could be provided without having a standard with a 5-minute averaging time (75 FR 35539, June 22, 2010). She judged that a standard with a 5-minute averaging time would result in significant and unnecessary instability in public health protection (75 FR 35539, June 22, 2010).<sup>9</sup> Accordingly, she considered other averaging times.

Results of air quality analyses in the REA suggested that a standard based on 24-hour average SO<sub>2</sub> concentrations would not likely be an effective or efficient approach for addressing 5-minute peak SO<sub>2</sub> concentrations, likely over-controlling in some areas, while under-controlling in others (2009 REA, section 10.5.2.2). In contrast, these analyses suggested that a 1-hour averaging time would be more efficient and effective at limiting 5-minute peaks of SO<sub>2</sub> (2009 REA, section 10.5.2.2.). Drawing on this information, the Administrator concluded that a 1-hour standard, with the appropriate form and level, would be likely to substantially reduce 5- to 10-minute peaks of SO<sub>2</sub> that had been shown in controlled human exposure studies to result in increased prevalence of respiratory symptoms and/or decrements in lung function in exercising people with asthma (75 FR 35539, June 22, 2010). Further she found that a 1-hour standard could substantially reduce the upper end of the distribution of SO<sub>2</sub> concentrations in ambient air that were more likely to be associated with respiratory outcomes (75 FR 35539, June 22, 2010).

The Administrator additionally took note of advice from the CASAC. The CASAC stated that the REA had presented a “convincing rationale” for a 1-hour standard, and that “a one-hour standard is the preferred averaging time” (Samet, 2009, pp. 15, 16). The CASAC further stated that it was “in agreement with having a short-term standard and finds that the REA supports a one-hour standard as protective of public health” (Samet, 2009, p. 1). Thus, in consideration of the available information summarized here and the CASAC’s advice, the Administrator concluded that a 1-hour standard (given the appropriate level and form) was an appropriate

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<sup>9</sup> Such instability could reduce public health protection by disrupting an area’s ongoing implementation plans and associated control programs (75 FR 35537, June 22, 2010).

means of controlling short-term exposures to SO<sub>2</sub> ranging from 5 minutes to 24 hours (75 FR 35539, June 22, 2010).

#### **3.1.1.2.3 Form**

In considering the statistical form for the new short-term standard, the Administrator judged that the form of the standard should reflect the health effects evidence presented in the ISA that indicated that the percentage of people with asthma affected and the severity of the response increased with increasing SO<sub>2</sub> concentrations (75 FR 35541, June 22, 2010). She additionally found it reasonable to consider stability (e.g., to avoid disruption of programs implementing the standard and the related public health protections from those programs) as part of her consideration of the form for the standard (75 FR 35541, June 22, 2010). In so doing, she noted that a concentration-based form averaged over three years would likely be appreciably more stable than a no-exceedance based form, which had been the form of the then-existing 24-hour standard (75 FR 35541, June 22, 2010). The CASAC additionally stated that “[t]here is adequate information to justify the use of a concentration-based form averaged over 3 years” (Samet, 2009, p. 16). In consideration of this information, the Administrator judged a concentration-based form averaged over three years to be most appropriate (75 FR 35541, June 22, 2010).

In selecting a specific concentration-based form, the Administrator considered health evidence from the ISA as well as air quality and exposure information from the REA. In so doing, the Administrator concluded that the form of a new 1-hour standard should be especially focused on limiting the upper end of the distribution of ambient SO<sub>2</sub> concentrations (i.e., above 90<sup>th</sup> percentile SO<sub>2</sub> concentrations) in order to provide protection with an adequate margin of safety against effects reported in epidemiologic and controlled human exposure studies (75 FR 35541, June 22, 2010). The Administrator further noted, based on results of air quality and exposure analyses in the REA, that a 99<sup>th</sup> percentile form was likely to be appreciably more effective at limiting 5-minute peak exposures of concern than a 98<sup>th</sup> percentile form (75 FR 35541, June 22, 2010). Thus, the Administrator selected a 99<sup>th</sup> percentile form averaged over three years (75 FR 35541, June 22, 2010).

#### **3.1.1.2.4 Level**

In selecting the level of a new 1-hour standard, the Administrator gave primary emphasis to the body of health effects evidence assessed in the ISA. In so doing, she noted that the controlled human exposure studies provided the most direct evidence of respiratory effects from exposure to SO<sub>2</sub>. The Administrator drew on evidence from these studies in reaching judgments on the magnitude of adverse respiratory effects and associated potential public health

1 significance for the air quality exposure and risk analysis results of air quality scenarios  
2 representing just meeting alternative levels for a new 1-hour standard.

3 In particular, the Administrator considered effects in exercising people with asthma after  
4 5- to 10-minute exposures as low as 200 ppb to be adverse in light of the CASAC advice on  
5 relevance of these effects, conclusions on similar effects in prior NAAQS reviews, and ATS  
6 guidelines (ATS, 1985, 2000). This judgment was based on several findings from the controlled  
7 human exposures studies. Five-to 10-minute exposures to 400 ppb or greater resulted in  
8 moderate or greater decrements in lung function in 20-60% of exercising individuals with  
9 asthma. These decrements are often statistically significant at the group mean level and  
10 frequently accompanied by respiratory symptoms. Thus, exposures to SO<sub>2</sub> concentrations at or  
11 above 400 ppb were concluded to clearly result in adverse respiratory effects based on the ATS  
12 guidelines (ATS, 1985). Further, 5- to 10-minute exposures to 200 to 300 ppb resulted in  
13 moderate or greater decrements in lung function in 5-30% of exercising individuals with asthma  
14 (75 FR 35546, June 22, 2010). Although such effects have not been shown to be statistically  
15 significant at the full study group mean level,<sup>10</sup> or to be frequently accompanied by respiratory  
16 symptoms, the Administrator considered effects associated with exposures as low as 200 ppb to  
17 be adverse in light of the CASAC's advice<sup>11</sup> and similar conclusions in prior reviews as well as  
18 the ATS guidelines (ATS, 1985, 2000).

19 The Administrator then considered what the findings of the REA exposure analyses  
20 indicated with regard to varying degrees of protection that different 1-hour standard levels might  
21 be expected to provide against 5-minute benchmark concentrations of 200 ppb and 400 ppb.<sup>12</sup>  
22 For example, the exposure assessment for St. Louis<sup>13</sup> estimated that a 1-hour standard at 100 ppb  
23 would likely protect more than 99% of children with asthma in that city from experiencing any  
24 days in a year with at least one 5-minute exposure at or above 400 ppb while at moderate or  
25 greater exertion, and approximately 97% of those children with asthma from experiencing any  
26 days in a year with at least one exposure at or above 200 ppb while at moderate or greater  
27 exertion (75 FR 35547, June 22, 2010). Results for the air quality scenario for a 1-hour standard

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<sup>10</sup> As summarized in section 3.2.1.1 below and described more fully in the second draft ISA for this review, study subjects have since been characterized as falling into two subpopulations that differ in susceptibility to SO<sub>2</sub>.

<sup>11</sup> The CASAC letter on the first draft SO<sub>2</sub> REA to the Administrator stated: "CASAC believes strongly that the weight of clinical and epidemiology evidence indicates there are detectable clinically relevant health effects in sensitive subpopulations down to a level at least as low as 0.2 ppm SO<sub>2</sub>" (Henderson, 2008).

<sup>12</sup> The Administrator additionally noted the results of the 40-county analysis of limited available 5-minute concentration data that indicated for a 1-hour standard level of 100 ppb a maximum annual average of two days per year with 5-minute concentrations above 400 ppb and 13 days with 5-minute concentrations above 200 ppb (76 FR 35546, June 22, 2010).

<sup>13</sup> St. Louis was one of two study areas assessed in the REA (2009 REA).

1 level of 50 ppb suggested that such a standard would somewhat further limit exposures, such that  
2 more than 99% of children at moderate or greater exertion would likely be protected from  
3 experiencing any days in a year with a 5-minute exposure at or above the 200 ppb benchmark  
4 concentration (75 FR 35542-47, June 22, 2010).

5 In considering the implications of the exposure assessment results the Administrator  
6 noted that although she considered the health effects resulting from 5-minute SO<sub>2</sub> exposures as  
7 low as 200 ppb to be adverse, she also recognized that such effects are appreciably less severe  
8 than those at SO<sub>2</sub> concentrations at or above 400 ppb and found little difference between the  
9 results for standard levels of 50 and 100 ppb with regard to 5-minute exposures at or above 400  
10 ppb. She recognized that a standard level below 100 ppb may somewhat further limit 5-minute  
11 SO<sub>2</sub> ambient air concentrations and exposures above 200 ppb, although she did not judge that a  
12 standard level of 50 ppb was warranted.

13 Before reaching her conclusion with regard to level for the 1-hour standard, the  
14 Administrator additionally considered the epidemiological evidence among the U.S.  
15 epidemiologic studies (some conducted in multiple locations) reporting mostly positive and  
16 sometimes statistically significant associations between ambient SO<sub>2</sub> concentrations and  
17 emergency department visits and hospital admissions. She noted there was a cluster of three  
18 studies for which 99<sup>th</sup> percentile 1-hour daily maximum concentrations were estimated to be  
19 between 78-150 ppb and for which the SO<sub>2</sub> effect estimate remained positive and statistically  
20 significant in copollutant models with particulate matter (PM) (75 FR 35547, June 22, 2010).<sup>14</sup>

21 Given the above considerations and the comments received on the proposal, the  
22 Administrator determined that the appropriate judgment, based on the entire body of evidence  
23 and information available in this review, and the related uncertainties,<sup>15</sup> was a standard level of  
24 75 ppb. She concluded that such a standard, with a 1-hour averaging time and 99<sup>th</sup> percentile  
25 form, would provide a significant increase in public health protection compared to the current  
26 standards and would be expected to provide protection, with an adequate margin of safety,  
27 against the respiratory effects that have been linked with SO<sub>2</sub> exposures in both controlled  
28 human exposure and epidemiologic studies. Specifically, she concluded that such a standard  
29 would limit 1-hour exposures at and above 75 ppb. (75 FR 35548, June 22, 2010). Such a  
30 standard was also considered likely “to maintain SO<sub>2</sub> concentrations below those in locations

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<sup>14</sup> Regarding the monitor concentrations in these studies, the EPA noted that although they may be a reasonable approximation of concentrations occurring in the areas, the monitored concentrations were likely somewhat lower than the absolute highest 99<sup>th</sup> percentile 1-hour daily maximum SO<sub>2</sub> concentrations occurring across these areas (75 FR 35547, June 22, 2010).

<sup>15</sup> Such uncertainties included both those with regard to the epidemiologic evidence and also those with regard to the information from controlled human exposure studies for at-risk groups, including representation of individuals with more severe asthma than that in study subjects (75 FR 35546, June 22, 2010).

1 where key U.S. epidemiologic studies have reported that ambient SO<sub>2</sub> is associated with clearly  
2 adverse respiratory health effects, as indicated by increased hospital admissions and emergency  
3 department visits.” The Administrator also found that “a 1-hour standard at a level of 75 ppb is  
4 expected to substantially limit asthmatics’ exposure to 5–10 minute SO<sub>2</sub> concentrations ≥ 200  
5 ppb, thereby substantially limiting the adverse health effects associated with such exposures.”  
6 Lastly, the Administrator noted “that a standard level of 75 ppb is consistent with the consensus  
7 recommendation of CASAC”. The Administrator also considered the likelihood of public health  
8 benefits at lower standard levels, and judged a 1-hour standard at 75 ppb to be sufficient to  
9 protect public health with an adequate margin of safety (75 FR 35547-35548, June 22, 2010).

10 This judgment included consideration of the appropriate degree of protection with an  
11 adequate margin of safety for populations at increased risk for adverse respiratory effects from  
12 short-term exposures to SO<sub>2</sub> for which the evidence supports a causal relationship with SO<sub>2</sub>  
13 exposures. In reaching these conclusions, the Administrator considered the requirement for a  
14 standard that is neither more nor less stringent than necessary for this purpose and recognized  
15 that the CAA does not require that primary NAAQS be set at a zero-risk level or to protect the  
16 most susceptible individual, but rather at a level that reduces risk sufficiently so as to protect  
17 public health with an adequate margin of safety (75 FR 35548, June 22, 2010).

#### 18 **3.1.1.2.5 Revoking the Then-Existing 24-Hour and Annual Standards**

19 In addition to setting a new 1-hour standard at 75 ppb, the then-current 24-hour and  
20 annual standards were revoked in the last review based largely on the recognition that a 1-hour  
21 standard set at 75 ppb would have the effect of generally maintaining 24-hour and annual SO<sub>2</sub>  
22 concentrations well below the levels of those standards (75 FR 35550, June 22, 2010). In  
23 addition, the annual standard was also revoked because of the lack of evidence supporting a  
24 relationship between long-term SO<sub>2</sub> exposures and adverse health effects. That is, the 2008 ISA  
25 judged the health evidence linking long-term SO<sub>2</sub> exposure to adverse health effects to be  
26 “inadequate” to infer the presence or absence of a causal relationship (75 FR 35550, June 22,  
27 2010; 2008 ISA, section 5.5).

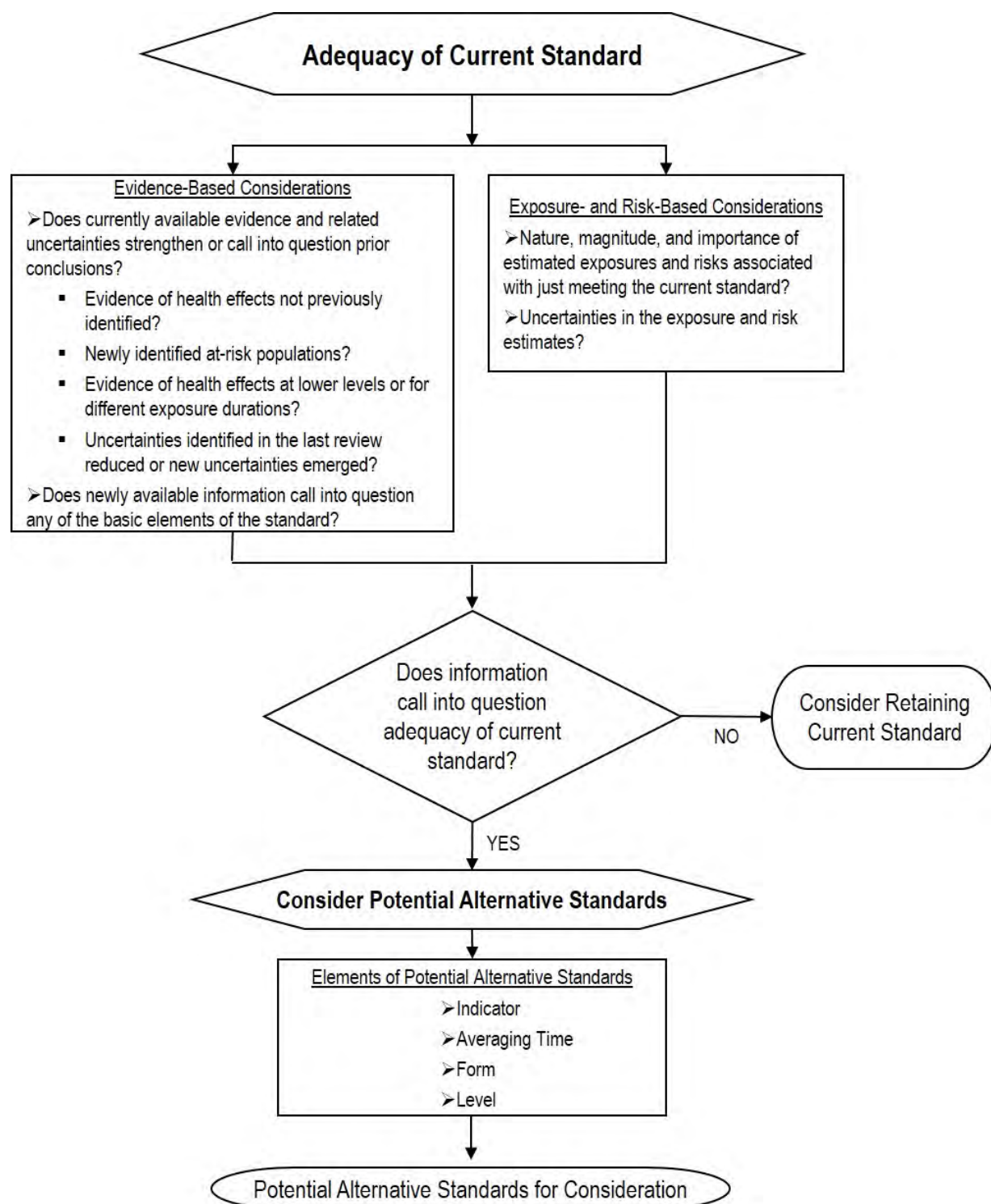
#### 28 **3.1.2 Approach for the Current Review**

29 To evaluate whether it is appropriate to consider retaining the current SO<sub>2</sub> primary  
30 standard, or whether consideration of revision is appropriate, we have adopted an approach in  
31 this review that builds on the general approach used in the last review and reflects the body of  
32 evidence and information now available. As summarized above, the Administrator’s decisions in  
33 the prior review were based on an integration of information on health effects associated with  
34 exposure to SO<sub>2</sub>, expert judgments on the adversity and public health significance of key health  
35 effects, air quality and related analyses and quantitative exposure and risk assessments, and

1 policy judgments as to when the standard is requisite to protect public health with an adequate  
2 margin of safety.

3 In conducting this assessment, we draw on the current evidence and quantitative  
4 assessments of exposure pertaining to the public health risk of SO<sub>2</sub> in ambient air. In considering  
5 the scientific and technical information, we consider both the information available at the time of  
6 the last review and information newly available since the last review, including the second draft  
7 ISA and draft REA for this review. Figure 3-1 below illustrates the basic construct of our two-  
8 part approach in developing preliminary conclusions regarding options to consider with regard to  
9 the adequacy of the current primary standard and, as appropriate, potential alternative standards.  
10 In the boxes of Figure 3-1, the range of questions that we consider in sections 3.2.1 and 3.2.2  
11 below are represented by a summary of policy-relevant questions that frame our consideration of  
12 the scientific evidence and quantitative analyses.

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4 **Figure 3-1. Overview of the approach for review of the current primary standard.**

## 3.2 ADEQUACY OF THE CURRENT STANDARD

In considering the adequacy of the current SO<sub>2</sub> primary standard, the overarching question we consider is:

- **Does the currently available scientific evidence- and exposure/risk-based information, as reflected in the ISA and REA, support or call into question the adequacy of the protection afforded by the current SO<sub>2</sub> primary standard?**

To assist us in interpreting the currently available scientific evidence and the results of recent quantitative exposure/risk analyses to address this question, we have focused on a series of more specific questions, as detailed in sections 3.2.1 and 3.2.2 below. In considering the scientific and technical information, we consider both the information available at the time of the last review and information newly available since the last review which have been critically analyzed and characterized in the 2008 ISA for the last review and the second draft ISA for the current review.

### 3.2.1 Evidence-based Considerations

In considering the evidence with regard to the overarching question posed above regarding the adequacy of the current standard, we address a series of more specific questions that focus on policy-relevant aspects of the evidence. These questions begin with consideration of the available evidence regarding the health effects associated with exposure to SO<sub>x</sub>, and particularly SO<sub>2</sub> (section 3.2.1.1). The subsequent questions consider identification of populations at-risk of SO<sub>2</sub>-related health effects (section 3.2.1.2), and the exposure durations and levels of SO<sub>2</sub> associated with health effects (section 3.2.1.3). Important uncertainties associated with the evidence are considered in section 3.2.1.4.

#### 3.2.1.1 Health Effects Associated with Exposure to SO<sub>x</sub>

Among the species of SO<sub>x</sub> (a group of closely related gaseous compounds including SO<sub>2</sub> and SO<sub>3</sub>), SO<sub>2</sub> is the most commonly occurring in the atmosphere. Accordingly, the large body of scientific evidence has over the past reviews been predominantly focused on exposures to SO<sub>2</sub>.

- **Is there newly available evidence that indicates the importance of SO<sub>x</sub> other than SO<sub>2</sub> with regard to abundance in ambient air, and potential for human exposures and health effects?**

As in the last review, the health effects evidence evaluated in the second draft ISA for SO<sub>x</sub> is focused on SO<sub>2</sub> (second draft ISA, p. 5-1). This is consistent with the conclusion that “[o]f the sulfur oxides, SO<sub>2</sub> is the most abundant in the atmosphere, the most important in atmospheric chemistry, and the one most clearly linked to human health effects” (second draft ISA, p. 2-1). While “SO<sub>3</sub> can be emitted by some sources, it reacts within seconds with water in

the stacks or immediately after release into the atmosphere to form H<sub>2</sub>SO<sub>4</sub> and gas-phase sulfuric acid quickly condenses or contributes to particle formation” (second draft ISA, section 2.3). Thus, the second draft ISA states that “only SO<sub>2</sub> is present at concentrations relevant for chemistry in the troposphere, boundary layer, and for human exposures” (second draft ISA, p. 2-17), and also that the available health evidence for SO<sub>x</sub> is focused on SO<sub>2</sub> (second draft ISA, p. 5-1). Thus, we conclude that the current evidence, including that newly available in this review, continues to support a focus on SO<sub>2</sub> in considering the adequacy of public health protection provided by the primary NAAQS for SO<sub>x</sub>.

- **Does the current evidence alter our conclusions from the previous review regarding the health effects associated with exposure to SO<sub>2</sub>?**

Rather than altering our conclusions from the last review, the current evidence continues to support our prior conclusions regarding the key health effects associated with SO<sub>2</sub> exposure. Specifically, the full body of evidence continues to support the conclusion that short-term SO<sub>2</sub> exposures of durations as short as a few minutes are causally related to respiratory effects in at-risk individuals (second draft ISA, section 5.2.1.9). With regard to respiratory effects and long-term exposures, as well as total mortality and short-term exposures, the evidence available in this review is “suggestive of, but not sufficient to infer,” a causal relationship (second draft ISA, sections 5.2.2.7 and 5.5.1.6). The evidence is inadequate for reaching conclusions regarding causality for other categories of effects (second draft ISA, section 1.6.2).<sup>16</sup>

### Respiratory Effects

As in the last review, the currently available evidence in this review supports the conclusion that there is a causal relationship between short-term SO<sub>2</sub> exposure and respiratory effects, particularly in individuals with asthma (second draft ISA, p. 1-16).<sup>17</sup> The clearest evidence for this conclusion comes from controlled human exposure studies available at the time of the previous review and included in the 2008 ISA. These studies demonstrate lung function

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<sup>16</sup> Based on the currently available evidence, the ISA concluded that the evidence was inadequate to infer the presence or absence of a causal relationship between SO<sub>2</sub> exposures and reproductive and developmental effects; between long-term SO<sub>2</sub> exposures and mortality or cancer; and, between short- or long-term SO<sub>2</sub> exposures and cardiovascular effects (second draft ISA, section 1.6.2).

<sup>17</sup> While effects have been documented for short (5 to 10 minutes) exposures lower than 1.0 ppm in controlled exposure studies of individuals with asthma, the exposure concentrations consistently eliciting effects in study subjects without asthma are higher. Such exposures are generally above 1.0 or 5.0 ppm, with most studies reporting no respiratory symptoms at concentrations up to 2.0 ppm (second draft ISA, section 5.2.1.7).

1 decrements<sup>18</sup> and respiratory symptoms in people with asthma exposed to SO<sub>2</sub> for 5 to 10  
2 minutes at elevated breathing rates (second draft ISA, section 5.2.1). The epidemiologic  
3 evidence, including recent evidence not available at the time of the previous review, includes  
4 studies reporting positive associations for asthma-related hospital admissions and emergency  
5 department visits with short-term SO<sub>2</sub> exposures (second draft ISA, section 5.2.1). These  
6 findings are generally supportive of the causal relationship conclusion for which the controlled  
7 exposure studies are the primary basis (second draft ISA, section 5.2.1.9).

8 Sulfur dioxide is a highly reactive and water-soluble gas that once inhaled is absorbed  
9 almost entirely in the upper respiratory tract<sup>19</sup> (second draft ISA, sections 4.2 and 4.3). Under  
10 conditions of elevated ventilation (e.g., while exercising), SO<sub>2</sub> penetrates into the  
11 tracheobronchial region,<sup>20</sup> where it may contribute to responses linked to asthma exacerbation in  
12 individuals with asthma (second draft ISA, sections 4.2, 4.3 and 5.2). More specifically,  
13 bronchoconstriction, which is characteristic of an asthma attack, is the most sensitive indicator of  
14 SO<sub>2</sub>-induced lung function effects. Associated with this bronchoconstriction response is an  
15 increase in airway resistance which is an index of airway hyperresponsiveness (AHR),<sup>21</sup>  
16 Exercising individuals without asthma have also been found to exhibit such responses, but at  
17 much higher SO<sub>2</sub> exposure concentrations, above 1000 ppb (second draft ISA, section 1.5.2).

18 Bronchoconstriction, evidenced by decrements in lung function, is observed in controlled  
19 human exposure studies after approximately 5- to 10-minute exposures and can occur at SO<sub>2</sub>  
20 concentrations as low as 200 ppb in some people with asthma exposed while breathing at  
21 elevated ventilation, such as during exercise (second draft ISA, section 5.2.1.2).<sup>22</sup> More  
22 consistent decrements in lung function are seen in such individuals with asthma following  
23 exposures to 400 ppb and greater (second draft ISA, section 5.2.1.2). In contrast, respiratory  
24 effects are not observed in other people with asthma (nonresponders) and healthy adults exposed

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<sup>18</sup> The specific responses reported in the evidence base that are described in the ISA as lung function decrements are increased specific airway resistance (sRaw) and reduced forced expiratory volume in 1 second (FEV<sub>1</sub>) (second draft ISA, section 5.2.1.2).

<sup>19</sup> The term “upper respiratory tract” refers to the portion of the respiratory tract, including the nose, mouth and larynx, that precedes the tracheobronchial region (second draft ISA, sections 4.2 and 4.3).

<sup>20</sup> The term “tracheobronchial region” refers to the region of the respiratory tract subsequent to the larynx and preceding the deep lung (or alveoli). This region includes the trachea and bronchii.

<sup>21</sup> Airway hyperresponsiveness, which is an increased propensity of the airways to narrow in response to bronchoconstrictive stimuli, is a characteristic feature of people with asthma (second draft ISA, section 5.2.1.2).

<sup>22</sup> The data from controlled human exposure studies of people with asthma indicate there to be two subpopulations that differ in their airway responsiveness to SO<sub>2</sub>, with the second subpopulation being insensitive to SO<sub>2</sub> bronchoconstrictive effects at concentrations as high as 1.0 ppm (second draft ISA, pp. 5-14 to 5-20; Johns et al., 2010).

1 while exercising to SO<sub>2</sub> concentrations below 1000 ppb (second draft ISA, sections 5.2.1.2 and  
2 5.2.1.7). Across studies, bronchoconstriction in response to SO<sub>2</sub> exposure is mainly seen during  
3 conditions of increased ventilation rates, such as exercise or laboratory-facilitated rapid, deep  
4 breathing.<sup>23</sup> These conditions lead to a shift from nasal breathing to oral/nasal breathing, which  
5 increases the concentration of SO<sub>2</sub> reaching the tracheobronchial region of lower airways, where  
6 depending on dose and the exposed individual's susceptibility, it may cause bronchoconstriction  
7 (second draft ISA, sections 4.1.2.2, 4.2.2 and 5.2.1.2).

8 The evidence base of controlled human exposure studies for people with asthma is the  
9 same in this review as in the last review. Such studies reporting asthma exacerbation-related  
10 effects for individuals with asthma are summarized in Tables 5-1 and 5-2, and section 5.2.1.2 of  
11 the second draft ISA. The main responses observed include increases in specific airway  
12 resistance (sRaw) and reductions in forced expiratory volume in one second (FEV<sub>1</sub>) after 5- to  
13 10-minute exposures. As in the last review, the second draft ISA in this review quantifies the  
14 percentage of exposed study subjects with at least 100%, 200% or 300% increases in sRaw (i.e.,  
15 a doubling, tripling or greater increase) and also those with at least 15%, 20% or 30% reduction  
16 in FEV<sub>1</sub>. As recognized in the last review, the results of these studies indicate that among  
17 individuals with similar disease status, some individuals have a greater response to SO<sub>2</sub> than  
18 others (second draft ISA, p. 5-14). The SO<sub>2</sub>-induced bronchoconstriction in these studies occurs  
19 rapidly, in as little as two minutes from exposure start, and is transient, with recovery following  
20 cessation of exposure (second draft ISA, p. 5-41).

21 The studies of subjects with asthma breathing at elevated ventilation have found effects  
22 to become more pronounced with increased exposure concentrations. Among individuals with  
23 asthma, both the percentage of individuals affected and the severity of the response increases  
24 with increasing SO<sub>2</sub> concentrations. For example, at concentrations ranging from 200 to 300 ppb,  
25 as many as 5 to 30% of exercising study subjects with asthma experienced moderate<sup>24</sup> or greater  
26 decrements in lung function (second draft ISA, Table 5-2). At concentrations at or above 400  
27 ppb, moderate or greater decrements in lung function occurred in 20 to 60% of exercising study  
28 subjects with asthma, and compared to exposures at 200 to 300 ppb, a larger percentage of  
29 subjects experienced severe decrements in lung function (i.e., an increase in sRaw of at least  
30 200%, and/or a reduction in FEV<sub>1</sub> of at least 20%) (second draft ISA, Table 5-2). Moreover, at

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<sup>23</sup> In the laboratory, study subjects perform this rapid, deep breathing through a mouthpiece that provides a mixture of oxygen with enough carbon dioxide to prevent the imbalance of gases in the blood usually resulting from hyperventilation. Breathing in the laboratory with this technique is referred to as eucapnic hyperpnea.

<sup>24</sup> As in the last review (described in section 3.1.1.1 above), the second draft ISA describes moderate or greater lung function decrements as the occurrence of at least a doubling in sRaw or at least a 15% reduction in FEV<sub>1</sub> (second draft ISA, section 1.6.1.1).

1 the higher SO<sub>2</sub> concentrations, moderate or greater decrements in lung function were frequently  
2 accompanied by respiratory symptoms, such as cough, wheeze, chest tightness, or shortness of  
3 breath (second draft ISA, Table 5-2).

4 With regard to newly available epidemiological studies, there are a limited number of  
5 such studies that have investigated SO<sub>2</sub> effects related to asthma exacerbation, with the most  
6 cohesive evidence coming from studies on asthma-related emergency department (ED) visits  
7 (second draft ISA, section 5.2.1.2). As in the last review, areas of uncertainty in the  
8 epidemiologic evidence relate to the characterization of exposure through the use of fixed site  
9 monitor concentrations as surrogates for population exposure (often over a substantially sized  
10 area and for durations greater than an hour) and the potential for confounding by PM or other  
11 copollutants (second draft ISA, section 5.2.1). In general, the pattern of associations across the  
12 newly available studies is consistent with the studies available in the last review (second draft  
13 ISA, p. 5-70).

14 As in the last review, the evidence base for short-term SO<sub>2</sub> exposures and respiratory  
15 effects other than asthma exacerbation is limited and inconsistent. The second draft ISA finds the  
16 evidence for an effect of SO<sub>2</sub> exposure on allergy exacerbation, COPD exacerbation, respiratory  
17 infection, respiratory effects in healthy populations, and respiratory mortality to be inconsistent  
18 within and across disciplines and outcomes, and finds there to be uncertainty associated with the  
19 epidemiological evidence for these endpoints that is related to potential confounding by  
20 copollutants (second draft ISA, section 5.2.1.9).

21 The evidence base for long-term SO<sub>2</sub> exposure and respiratory effects is somewhat  
22 augmented since the last review such that the second draft ISA in the current review concludes it  
23 to be suggestive of, but not sufficient to infer, a causal relationship (second draft ISA, section  
24 5.2.2). The support for this conclusion comes mainly from the limited epidemiological study  
25 findings of associations between long-term SO<sub>2</sub> concentrations and increases in asthma incidence  
26 combined with findings of laboratory animal studies involving newborn rodents that indicate a  
27 potential for SO<sub>2</sub> exposure to contribute to the development of asthma, especially allergic  
28 asthma, in children (second draft ISA, section 1.6.1.2). The evidence showing increases in  
29 asthma incidence is coherent with results of animal toxicological studies that provide a  
30 pathophysiologic basis for the development of asthma. The overall body of evidence, however,  
31 lacks consistency (second draft ISA, section 1.6.1.2). Further there are uncertainties, discussed in  
32 section 3.2.1.4 below, that apply to the epidemiologic evidence, including that newly available,  
33 across the respiratory effects examined for long-term SO<sub>2</sub> exposure (second draft ISA, section  
34 5.2.2.7).

## Other Health Effects

For effects other than respiratory effects, the current evidence is generally similar to the evidence available in the last review, and is supportive of similar conclusions. With regard to a relationship between short-term SO<sub>2</sub> exposure and total mortality, the second draft ISA reaches the same conclusion as in the previous review that the evidence is suggestive of, but not sufficient to infer, a causal relationship (second draft ISA, section 5.5.1). This conclusion is based on previous and recent multicity epidemiologic studies providing consistent evidence of positive associations, although there is uncertainty regarding the potential for SO<sub>2</sub> to have an independent effect on mortality. While recent studies have analyzed some key uncertainties and data gaps from the previous review, uncertainties still exist, given the limited number of studies that examined copollutant confounding, the evidence for a decrease in the size of SO<sub>2</sub>-mortality associations in copollutant models with NO<sub>2</sub> and PM<sub>10</sub>, and the lack of a potential biological mechanism for mortality following short-term SO<sub>2</sub> exposures (second draft ISA, section 1.6.2.4).

For other categories of health effects,<sup>25</sup> the evidence is inadequate to infer the presence or absence of a causal relationship, mainly due to inconsistent evidence across specific outcomes and uncertainties regarding exposure measurement error, copollutant confounding, and potential modes of action (second draft ISA, sections 5.3.1, 5.3.2, 5.4, 5.5.1, 5.5.2, 5.6). These conclusions are consistent with those made in the previous review.

In summary, rather than altering our conclusions from the previous review, the current evidence provides continued support for our previous conclusions regarding the health effects associated with exposure to SO<sub>2</sub> and most particularly respiratory effects following short-term SO<sub>2</sub> exposure, particularly in individuals with asthma. Accordingly, as in prior reviews, this review gives primary focus to those effects most pertinent to exposures related to current concentrations in ambient air, in particular, asthma exacerbation in individuals with asthma.

### **3.2.1.2 Populations At-Risk of SO<sub>2</sub>-Related Health Effects**

Populations or lifestages can be at increased risk of an air pollutant-related health effect due to one or more of a number of factors. These factors can be intrinsic, such as physiological factors that may influence the internal dose or toxicity of a pollutant, or extrinsic, such as sociodemographic, or behavioral factors. The questions considered in this section address what the currently available evidence indicates regarding which populations are particularly at risk of health effects related to exposure to SO<sub>2</sub> in ambient air.

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<sup>25</sup> The other categories evaluated in the ISA include cardiovascular effects with short or long term exposures; reproductive and developmental effects; and cancer and total mortality with long-term exposure (second draft ISA, Table 1-1).

- **Does the current evidence alter our understanding of populations that are particularly at-risk from SO<sub>2</sub> exposures? Is there new evidence that suggests additional at-risk populations that should be given increased focus in this review?**

The currently available evidence continues to support our primary conclusions from the previous review that people with asthma are at increased risk for SO<sub>2</sub>-related health effects, specifically for respiratory effects, and specifically asthma exacerbation, associated with short-term exposures while at elevated ventilation (second draft ISA, sections 5.2.1.2 and 6.3.1). This conclusion of the at-risk status of people with asthma is based on the well-established, and well-characterized, evidence from controlled human exposure studies, supported by the evidence on mode of action for SO<sub>2</sub> and with limited additional support from epidemiologic studies (second draft ISA, sections 5.2.1.2 and 6.3.1). Somewhat similar to the conclusion in the last review that children and older adults are potentially susceptible populations, the second draft ISA (relying on a more systematic approach for evaluating the evidence than in the last review) indicates the evidence to be suggestive of increased risk for these groups, with some limitations and inconsistencies (second draft ISA, sections 6.5.1.1 and 6.5.1.2).<sup>26</sup>

Further, the second draft ISA finds that children with asthma may be particularly at risk compared to adults with asthma (second draft ISA, section 6.3.1). This conclusion reflects several characteristics of children as compared to adults, which include their greater responsiveness to methacholine,<sup>27</sup> a chemical that can elicit bronchoconstriction in people with asthma, as well as their greater use of oral breathing, particularly by boys (second draft ISA, sections 5.2.1.2 and 4.1.2). Oral breathing (vs nasal breathing) and increased ventilation are factors that allow for greater SO<sub>2</sub> penetration into the tracheobronchial region of the lower airways, and reflect conditions of individuals with asthma in which bronchoconstriction-related responses have been observed in the controlled exposure studies (second draft ISA, sections 4.2.2, 5.2.1.2 and 6.3.1).

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<sup>26</sup> The current evidence for risk to older adults relative to other lifestages comes from epidemiological studies, for which the findings are somewhat inconsistent, and studies with which there are uncertainties in the association with the health outcome (second draft ISA, section 6.5.1.2).

<sup>27</sup> The second draft ISA concluded that potential differences in airway responsiveness of children to SO<sub>2</sub> relative to adolescents and adults may be inferred by the responses to methacholine (second draft ISA, section 5.2.1.2). Methacholine is a chemical that can elicit bronchoconstriction through its action on airway smooth muscle receptors. It is commonly used to identify people with asthma and accordingly has been used to screen subjects for studies of SO<sub>2</sub> effects. An analysis of the extent to which airway responsiveness to methacholine, a history of respiratory symptoms, and atopy were significant predictors of airway responsiveness to SO<sub>2</sub>, found that about 20 to 25% of subjects ranging in age from 20 to 44 years were hyperresponsive to methacholine (second draft ISA, section 5.2.1.2; Nowak et al., 1997). Another study focused on individuals with airway responsiveness to methacholine found only a weak correlation between airway responsiveness to SO<sub>2</sub> and methacholine (second draft ISA, section 5.2.1.2; Horstman et al., 1986).

We additionally recognize the well-documented finding that some individuals with asthma have a greater response to SO<sub>2</sub> than others with similar disease status (second draft ISA, section 5.2.1.2; Horstman et al., 1986; Johns et al., 2010). This occurrence is quantitatively analyzed in a study newly available in this review. This study uses the available individual subject data from five studies involving exposure of individuals with asthma to multiple concentrations of SO<sub>2</sub> for 5 to 10 minutes while at elevated ventilation to examine the differences in lung function response (Johns et al., 2010). As noted in the second draft ISA, “these data demonstrate a bimodal distribution of airway responsiveness to SO<sub>2</sub> in individuals with asthma, with one subpopulation that is insensitive to the bronchoconstrictive effects of SO<sub>2</sub> even at concentrations as high as 1.0 ppm, and another subpopulation that has an increased risk for bronchoconstriction at low concentrations of SO<sub>2</sub>” (second draft ISA, p. 5-17). To date, the characteristics that may define the subpopulation of responders have not been identified. The current evidence for factors other than those discussed above (asthma status and lifestage) is inadequate to determine whether they might contribute to an increased risk of SO<sub>2</sub>-related effects (second draft ISA, section 6.6).

### **3.2.1.3 Exposure Concentrations Associated with Health Effects**

At the time of the last review, the EPA’s conclusions regarding concentrations of SO<sub>2</sub> associated with respiratory effects were based primarily on the strong evidence base of controlled human exposure studies of individuals with asthma. These studies have documented bronchoconstriction-related moderate or greater decrements in lung function following 5- to 10-minute exposures during exercise. The severity of observed responses, the percentage of individuals responding, statistical significance at the study group level and the accompanying occurrence of respiratory symptoms have been found to increase with increasing exposure concentration (75 FR 35526, June 22, 2010). This information was critical in the REA analyses in the last review, the results of which were a primary consideration in reaching a conclusion on the level for the 2010 standard.

- Does the current evidence alter our conclusions from the previous review regarding the exposure duration and concentrations associated with health effects?**

The current evidence, including that newly available in this review, supports conclusions from the last review on exposure duration and concentrations associated with SO<sub>2</sub>-related health effects. These conclusions were largely based on the longstanding evidence base of controlled human exposure studies that demonstrates a relationship between 5- and 10-minute SO<sub>2</sub> exposure concentrations and decrements in lung function (e.g., increased sRaw and reduced FEV<sub>1</sub>) in individuals with asthma exposed while at elevated ventilation rate (second draft ISA, section

1 1.6.1.1). At the higher concentrations, there are clear and consistent increases in SO<sub>2</sub>-induced  
2 respiratory symptoms (second draft ISA, Table 5-2).

3 The available and well characterized evidence documents an effect of short-term  
4 exposures on the respiratory system. As summarized in section 3.2.1.1, SO<sub>2</sub>-induced  
5 bronchoconstriction occurs rapidly in responding study subjects with asthma exposed for just a few  
6 minutes while breathing at elevated ventilation rates (second draft ISA, section 5.2.1.2).  
7 Additionally, exposures as short as 5 minutes have been found to elicit a similar  
8 bronchoconstrictive response as somewhat longer exposures. For example, during exposure to  
9 SO<sub>2</sub> over a 30-minute period with continuous exercise, the response to SO<sub>2</sub> has been found to  
10 develop rapidly and is maintained throughout the 30-minute exposure (second draft ISA, p. 5-  
11 14). In a study involving short exercise periods within a 6-hour exposure period, the effects  
12 observed following exercise were documented to return to baseline levels within one hour after  
13 the cessation of exercise, even with continued exposure (Linn et al., 1984). In considering the  
14 epidemiological evidence with regard to the question of exposure duration, while we note the  
15 associations of asthma-related emergency room visits and hospital admissions with 1-hour to 24-  
16 hour ambient air concentration metrics, we recognize that current methods are not able to address  
17 whether these associations are due to exposure on the order of hours or much shorter-term  
18 exposure to peaks in SO<sub>2</sub> concentration. As noted in the second draft ISA, the air quality metrics  
19 in the epidemiological studies are for time periods longer than the 5- to 10-minute exposures  
20 eliciting effects in the controlled human exposure studies and may not adequately capture the  
21 spatial and temporal variation in SO<sub>2</sub> concentrations (second draft ISA, pp. 5-47, 5-55).

22 With regard to the evidence for exposure concentrations eliciting effects, we focus  
23 primarily on the controlled human exposure study findings for which data are available to the  
24 EPA for individual subjects with asthma that were exposed during elevated ventilation,  
25 summarized in Table 3-1 (second draft ISA, Table 5-2).<sup>28</sup> These data demonstrate that SO<sub>2</sub>  
26 concentrations as low as 200 to 300 ppb for 5 to 10 minutes elicited moderate or greater  
27 bronchoconstriction, measured as a decrease in FEV<sub>1</sub> of at least 15% or an increase in sRaw of at  
28 least 100% in a subset of the subjects (second draft ISA, section 5.2.1). Both the percent of  
29 individuals affected and the severity of response increased with increasing SO<sub>2</sub> concentrations.  
30 At concentrations ranging from 200 to 300 ppb, the lowest levels tested in free breathing  
31 chamber studies, 5 to 30% of exercising individuals with asthma experienced moderate or greater  
32 decrements in lung function (second draft ISA, section 5.2.1). At concentrations at or above 400  
33 ppb, moderate or greater decrements in lung function occurred in 20 to 60% of exercising

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<sup>28</sup> The findings summarized in Table 5-2 of the second draft ISA and in Table 3-1 of this draft PA are based on results that have been adjusted for effects at exercise in clean air so that they have separated out any effect of exercise in causing bronchoconstriction and reflect the SO<sub>2</sub>-specific effect.

1 individuals with asthma and a larger percentage of individuals with asthma experienced more  
2 severe decrements in lung function (i.e., an increase in sRaw of at least 200%, and/or a 20% or  
3 more decrease in FEV<sub>1</sub>), compared to exposures at 200 to 300 ppb (second draft ISA, section  
4 5.2.1). Additionally, at concentrations at or above 400 ppb, moderate or greater decrements in  
5 lung function were frequently accompanied by respiratory symptoms, with some of these  
6 findings reaching statistical significance (second draft ISA, section 5.2.1).

1 **Table 3-1. Percentage of adults with asthma in controlled human exposure studies experiencing sulfur dioxide-induced**  
2 **decrements in lung function and respiratory symptoms (adapted from Table 5-2 in the second draft ISA).**

SO <sub>2</sub> Conc (ppm)	Exposure Duration (min)	N	Ventil- ation (L/min)	Cumulative Percentage of Responders (Number of Subjects) <sup>a</sup>				Study	Respiratory Symptoms: Supporting Studies
				sRaw	≥100% ↑	≥200% ↑	≥300% ↑		
				FEV <sub>1</sub>	≥15% ↓	≥20% ↓	≥30% ↓		
0.2	5	23	~48	sRaw	9% (2) <sup>b</sup>	0	0	Linn et al. (1983b)	Limited evidence of SO <sub>2</sub> -induced increases in respiratory symptoms in some people with asthma: Bethel et al., 1985; Horstman et al., 1986; Linn et al., 1983b; Linn et al., 1987; Linn et al., 1988; Linn et al. 1990; Schachter et al., 1984
	10	40	~40	sRaw	7.5% (3) <sup>c</sup>	2.5% (1) <sup>c</sup>	0 <sup>c</sup>	Linn et al. (1987) <sup>c</sup>	
	10	40	~40	FEV <sub>1</sub>	9% (3.5) <sup>c</sup>	2.5% (1) <sup>c</sup>	1% (0.5) <sup>c</sup>	Linn et al. (1987) <sup>c</sup>	
0.25	5	19	~50–60	sRaw	32% (6)	16% (3)	0	Bethel et al. (1985)	
	5	9	~80–90	sRaw	22% (2)	0	0	Bethel et al. (1985)	
	10	27	~42	sRaw	0	0	0	Horstman et al. (1986)	
	10	28	~40	sRaw	4% (1)	0	0	Roger et al. (1985)	
0.3	10	20	~50	sRaw	10% (2)	5% (1)	5% (1)	Linn et al. (1988) <sup>d</sup>	Stronger evidence with some statistically significant increases in respiratory symptoms: Balmes et al., 1987; <sup>f</sup> Gong et al., 1995 ; Linn et al., 1983b; Linn et al., 1987 ; Roger et al., 1985
	10	21	~50	sRaw	33% (7)	10% (2)	0	Linn et al. (1990) <sup>d</sup>	
	10	20	~50	FEV <sub>1</sub>	15% (3)	0	0	Linn et al. (1988)	
	10	21	~50	FEV <sub>1</sub>	24% (5)	14% (3)	10% (2)	Linn et al. (1990)	
0.4	5	23	~48	sRaw	13% (3)	4% (1)	0	Linn et al. (1983b)	
	10	40	~40	sRaw	24% (9.5) <sup>c</sup>	9% (3.5) <sup>c</sup>	4% (1.5) <sup>c</sup>	Linn et al. (1987) <sup>c</sup>	
	10	40	~40	FEV <sub>1</sub>	27.5% (11) <sup>c</sup>	17.5% (7) <sup>c</sup>	10% (4) <sup>c</sup>	Linn et al. (1987) <sup>c</sup>	
0.5	5	10	~50–60	sRaw	60% (6)	40% (4)	20% (2)	Bethel et al. (1983)	
	10	27	~42	sRaw	22.2% (6)	7.4% (1)	3.7% (1)	Horstman et al. (1986)	
	10	28	~40	sRaw	18% (5)	4% (1)	4% (1)	Roger et al. (1985)	

SO <sub>2</sub> Conc (ppm)	Exposure Duration (min)	N	Ventil- ation (L/min)	Cumulative Percentage of Responders (Number of Subjects) <sup>a</sup>				Study	Respiratory Symptoms: Supporting Studies
				sRaw	≥100% ↑	≥200% ↑	≥300% ↑		
				FEV <sub>1</sub>	≥15% ↓	≥20% ↓	≥30% ↓		
	10	45	~30	sRaw	36% (16)	16% (7)	13% (6)	Magnussen et al. (1990) <sup>f</sup>	
0.6	5	23	~48	sRaw	39% (9)	26% (6)	17% (4)	Linn et al. (1983b)	Clear and consistent increases in SO <sub>2</sub> -induced respiratory symptoms: Gong et al., 1995; Horstman et al., 1988; Linn et al., 1983b; Linn et al., 1987; Linn et al., 1990
	10	40	~40	sRaw	34% (13.5) <sup>c</sup>	24% (9.5) <sup>c</sup>	19% (7.5) <sup>c</sup>	Linn et al. (1987) <sup>c</sup>	
	10	20	~50	sRaw	60% (12)	35% (7)	10% (2)	Linn et al. (1988)	
	10	21	~50	sRaw	62% (13)	29% (6)	14% (3)	Linn et al. (1990)	
	10	40	~40	FEV <sub>1</sub>	47.5% (19) <sup>c</sup>	39% (15.5) <sup>c</sup>	17.5% (7) <sup>c</sup>	Linn et al. (1987) <sup>c</sup>	
	10	20	~50	FEV <sub>1</sub>	55% (11)	55% (11)	5% (1)	Linn et al. (1988)	
	10	21	~50	FEV <sub>1</sub>	43% (9)	38% (8)	14% (3)	Linn et al. (1990)	
1.0	10	28	~40	sRaw	50% (14)	25% (7)	14% (4)	Roger et al. (1985) <sup>e</sup>	
	10	10	~40	sRaw	60% (6)	20% (2)	0	Kehrl et al. (1987)	
	10	27	~42	sRaw	55.6% (15)	25.9% (7)	11% (1)	Horstman et al. (1986)	

Conc = concentration; FEV<sub>1</sub> = forced expiratory volume in 1 sec; sRaw = specific airway resistance; SO<sub>2</sub> = sulfur dioxide.

<sup>a</sup>Data presented from all references from which individual data were available in the published paper or were provided to EPA (Johns, 2009; Johns and Simmons, 2009; Smith, 1993). Percentage of individuals who experienced greater than or equal to a 100, 200, or 300% increase in specific airway resistance, or a 15, 20, or 30% decrease in FEV<sub>1</sub>. Lung function decrements are adjusted for the effects of exercise in clean air (calculated as the difference between the percent change relative to baseline with exercise/SO<sub>2</sub> and the percent change relative to baseline with exercise/clean air).

<sup>b</sup>Numbers in parenthesis represent the number of subjects experiencing the indicated effect.

<sup>c</sup>Responses of people with mild and moderate asthma reported in Linn et al. (1987) have been combined. Data are the average of the first and second round exposure responses following the first 10 min period of exercise.

<sup>d</sup>Analysis includes data from only people with mild Linn et al. (1988) and moderate Linn et al. (1990) asthma who were not receiving supplemental medication.

<sup>e</sup>One subject was not exposed to 1 ppm due to excessive wheezing and chest tightness experienced at 0.5 ppm. For this subject, the values used for 0.5 ppm were also used for 1.0 ppm under the assumption that the response at 1.0 ppm would be equal to or greater than the response at 0.5 ppm.

<sup>f</sup>Indicates studies in which exposures were conducted using a mouthpiece rather than a chamber.

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1 The lowest exposure concentration in Table 3-1 is 200 ppb. This is the lowest exposure  
2 concentration tested by studies in which study subjects breathed freely (e.g., without using a  
3 mouthpiece).<sup>29</sup> In such studies that tested 200 ppb, a portion of the exercising study subjects with  
4 asthma (approximately 8 to 9%) responded with at least a doubling in sRaw or an increase in  
5 FEV1 of at least 15% (Table 3-1; Linn et al., 1983b; Linn et al., 1987).

6 With regard to exposure concentrations below 200 ppb, the available evidence is very  
7 limited. This information is derived from studies in which the subjects were exposed by  
8 mouthpiece rather than freely breathing in an exposure chamber (Sheppard et al., 1981; Sheppard  
9 et al., 1984; Koenig et al., 1989; Koenig et al., 1990; Trenga et al., 2001).<sup>30</sup> Additionally, some  
10 of these studies did not include an exposure to clean air while exercising that would have  
11 allowed for determining the effect of SO<sub>2</sub> *versus* that of exercise in causing bronchoconstriction.  
12 In those cases, the lung function measurements (e.g., sRaw, FEV<sub>1</sub>) following SO<sub>2</sub> exposure are  
13 assessed relative to measurements taken prior to exposure (baseline), rather than being assessed  
14 relative to measurements for a control exposure to clean air while exercising. The studies cited  
15 here, of a limited number of adults and adolescents, reported small changes in FEV<sub>1</sub> or sRaw in  
16 the individual study subjects, with magnitudes of change appearing to be smaller than responses  
17 reported from studies at exposure concentrations of 200 ppb or more. For example, the increase  
18 in sRaw reported for two young adult subjects exposed to 100 ppb in the study by Sheppard et al.  
19 (1981) was approximately half the response of these subjects at 250 ppb. In the two studies of  
20 adolescents (aged 12 to 18 years), among the individual study subjects for which respiratory  
21 resistance increased with SO<sub>2</sub> exposure, the magnitude of increase was less than 100% in each  
22 subject (Koenig et al., 1990; Koenig et al., 1989).

23 In considering what can be gleaned from these mouthpiece studies of 100 ppb, we note  
24 that the results of studies that utilize a mouthpiece exposure system cannot be directly compared  
25 to results from studies involving freely breathing subjects because when a mouthpiece is used,  
26 the inhaled breath completely bypasses the nasal passages where SO<sub>2</sub> is efficiently removed, thus  
27 allowing more of the inhaled SO<sub>2</sub> to penetrate into the tracheobronchial airways (2008 ISA, p. 3-  
28 4; second draft ISA, section 4.1.2.2). This occurrence as well as limited evidence comparing

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<sup>29</sup> Studies of free-breathing subjects generally make use of small rooms in which the atmosphere is experimentally controlled such that study subjects are exposed by freely breathing the surrounding air (e.g., Linn et al., 1987).

<sup>30</sup> A subset of these studies are cited in the second draft ISA; additionally, three of them (Sheppard et al., 1981; Koenig et al., 1990; Trenga et al., 2001) are cited in the 2008 ISA and a fourth (Sheppard et al., 1984) is cited in the 1986 Addendum and 1994 Supplement to the 1982 AQCD. The fifth study (Koenig et al., 1989) is not cited in the prior AQCDs, the 2008 ISA, or the second draft ISA. This study is an investigation involving nine adolescent subjects with allergic asthma (positive response to a methacholine challenge test at or below 20 mg/mL) exposed by mouthpiece to 0.1 ppm during exercise. Measurements of FEV<sub>1</sub> and R<sub>T</sub> were taken at baseline and subsequent to SO<sub>2</sub> and air only exposures during exercise (Koenig et al., 1989).

1 responses by mouthpiece and chamber exposures leads to the expectation that SO<sub>2</sub>-responsive  
2 people with asthma breathing SO<sub>2</sub> using a mouthpiece, particularly while at elevated ventilation,  
3 would experience greater lung function responses than if exposed to the same test concentration  
4 while freely breathing in an exposure chamber (second draft ISA, p. 5-22; Linn et al., 1983a).

5 We have also considered what can be gleaned from the epidemiological studies regarding  
6 exposure concentrations associated with health effects. Although exposure concentrations  
7 eliciting respiratory responses are not available from such studies, the ambient air concentrations  
8 occurring in studies that find associations with outcomes such as asthma-related ED visits and  
9 hospitalizations have the potential to indicate ambient air concentrations that may contribute to  
10 exposures that may be eliciting effects. For example, in recognizing the general coherence of  
11 epidemiological study findings for 24-hour ambient air concentrations with the findings of the  
12 controlled human exposure studies for exercising study subjects with asthma exposed for 5 to 10  
13 minutes, the 2008 ISA recognized that “it is possible that these epidemiologic associations are  
14 determined in large part by peak exposures within a 24-h period” (2008 ISA, p. 5-5). In  
15 considering the epidemiological studies in this light, however, we note that given the important  
16 role of SO<sub>2</sub> as a precursor to PM in ambient air, a key uncertainty in the epidemiological  
17 evidence available in the last review was potential confounding by PM (second draft ISA, p. 5-  
18 5). Among the U.S. epidemiologic studies (some conducted in multiple locations) reporting  
19 mostly positive and sometimes statistically significant associations between ambient SO<sub>2</sub>  
20 concentrations and emergency department visits and hospital admissions, few studies have  
21 attempted to address this uncertainty, e.g., through the use of copollutant models. For example,  
22 as in the last review, there are three U.S. studies for which the SO<sub>2</sub> effect estimate remained  
23 positive and statistically significant in copollutant models with PM (Appendix D).<sup>31</sup> No  
24 additional such studies have been newly identified in this review. Further, the second draft ISA  
25 states that uncertainty with regard to potential confounding by PM remains in the currently  
26 available epidemiologic evidence base (second draft ISA, p. 5-144).

#### 27 **3.2.1.4 Uncertainties in the Health Effects Evidence**

28 A number of key uncertainties and limitations were identified in the previous review with  
29 respect to the health effects evidence, as described in the 2009 REA. This section considers the  
30 currently available information, including that newly available in this review, with regard to such  
31 areas of uncertainty.

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<sup>31</sup> Based on data available for specific time periods at some monitors in the areas of these studies, the 99<sup>th</sup> percentile 1-hour daily maximum concentrations were estimated in the last review to be between 78-150 ppb (Thompson and Stewart, 2009).

- **To what extent have important uncertainties identified in the last review been reduced and/or have new uncertainties emerged?**

We have not identified any new uncertainties since the last review. However, we continue to recognize important uncertainties that also existed in the last review. This array of important areas of uncertainty related to the current health evidence, including that newly available in this review, is summarized below.

Although the evidence clearly demonstrates that short-term SO<sub>2</sub> exposures cause respiratory effects, particularly asthma exacerbation in exercising individuals with asthma, as in the previous review, we continue to recognize uncertainties that remain in several aspects of our understanding of these effects. Such uncertainties include those associated with severity and prevalence of responses to very short (5- to 10-minute) SO<sub>2</sub> exposures below 200 ppb and responses of some population groups not included in the controlled exposure studies (e.g., those with more severe asthma and children). There are also uncertainties concerning the potential influence of exposure history and co-exposure to other pollutants on the relationship between short-term SO<sub>2</sub> exposures and respiratory effects. With regard to the evidence base, we also recognize a complication associated with interpreting the epidemiologic evidence related to uncertainty in the exposure estimates. The following discussion touches on each of these types of uncertainty.

With regard to the potential for and magnitude of these effects in at-risk populations exposed to 5- to 10-minute concentrations below 200 ppb, there is very limited evidence from small mouthpiece studies of exposure concentrations as low as 100 ppb, as discussed in section 3.2.1.3 above. These studies indicate the likelihood of an appreciable reduction in SO<sub>2</sub>-induced response in exercising people with asthma from that observed from exposures at 200 ppb. Given the limited size of these studies and their differences from free breathing chamber studies, however, uncertainties remain with regard to a complete characterization of the extent of response in exercising individuals with asthma exposed through natural or free breathing to exposure concentrations below 200 ppb. The extent to which the epidemiological evidence, including that newly available, can inform this area of uncertainty also may be limited.<sup>32</sup> Accordingly, this remains an area of uncertainty in this review.

Some uncertainty also remains with regard to the extent to which the controlled human exposure study evidence describes the responses of the populations most at risk of SO<sub>2</sub>-related

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<sup>32</sup> As associations reported in the epidemiologic analyses are associated with air quality concentration metrics as surrogates for the actual pattern of exposures experienced by study population individuals over the period of a particular study, the studies are limited in what they can convey regarding the specific patterns of exposure circumstances (e.g., magnitude of concentrations over specific durations and frequency) that might be eliciting reported health outcomes.

1 respiratory effects (e.g., those with the most severe responses, or greatest likelihood of response).  
2 For example, the available studies have generally involved subjects with mild or moderate  
3 asthma, such that the response of individuals with more severe asthma is unknown.<sup>33</sup> Further,  
4 while it is well documented that some individuals have a greater response to SO<sub>2</sub> than others  
5 with the same disease status, the factors contributing to this greater susceptibility are not yet  
6 known (second draft ISA, pp. 5-14 to 5-20).

7       Uncertainty also remains related to the responses for children with asthma. Although the  
8 epidemiological evidence includes a number of studies focused on health outcomes in children  
9 that are supportive of the qualitative conclusions of causality (second draft ISA, section 5.2.1.2),  
10 there are few controlled human exposure studies to inform our understanding of concentrations  
11 associated with effects. Those studies have not included subjects younger than 12 years (second  
12 draft ISA, p. 5-21). Some characteristics particular to school age children younger than 12 years,  
13 such as increased propensity for mouth breathing (second draft ISA, section 4.1.2.2), however,  
14 suggest that this age group of children with asthma might be expected to experience larger lung  
15 function decrements than adults with asthma (second draft ISA, p. 5-24).

16       Other areas of uncertainty concerning the potential influence of SO<sub>2</sub> exposure history and  
17 co-exposure to other pollutants on the relationship between short-term SO<sub>2</sub> exposures and  
18 respiratory effects also remain from the last review. There is some limited evidence regarding the  
19 potential for an increased response to SO<sub>2</sub> exposures occurring in the presence of other common  
20 pollutants (e.g., PM, nitrogen dioxide and ozone), although the studies are limited (e.g., with  
21 regard to their relevance to ambient exposures) and/or provide inconsistent results (second draft  
22 ISA, p. 5-24; 2008 ISA, section 3.1.4.7). There is also some evidence suggestive of a potential  
23 for SO<sub>2</sub> exposure to contribute to an increased sensitivity to allergens, however the studies are  
24 very few and are limited to experimental animal models (second draft ISA, section 5.2.1.9).

25       There are additional complications associated with interpretation of epidemiologic  
26 studies of SO<sub>2</sub> in ambient air that pertain to exposure measurement error and copollutant  
27 confounding (second draft ISA, sections 3.4, 5.2.1.1 and 5.2.1.2). With regard to the former, a  
28 key uncertainty in the epidemiologic evidence is whether study findings reflect an independent  
29 association for SO<sub>2</sub> given that the studies assigned exposure from fixed site monitors while SO<sub>2</sub>  
30 concentrations in ambient air tend to show high spatiotemporal variability within a city, and

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<sup>33</sup> The second draft ISA identifies two studies that have investigated the influence of asthma severity on responsiveness to SO<sub>2</sub>, with one finding that a larger change in lung function observed in the moderate/severe asthma group was attributable to the exercise component of the study protocol while the other did not assess the role of exercise in differences across individuals with asthma of differing severity (Linn et al., 1987; Trenga et al., 1999). Based on the criteria used in the study by Linn et al (1987) for placing individuals in the “moderate/severe” group, the ISA concluded that the asthma of these individuals “would likely be classified as moderate by today’s classification standards” (second draft ISA, p. 5-20; Johns et al., 2010; Reddel, 2009).

1 correlations with personal exposure are poorly characterized. Accordingly, there is uncertainty  
2 regarding the extent to which measurements at the study monitors, and the associated air quality  
3 concentration metric for the study, adequately represent the spatiotemporal variability in ambient  
4 SO<sub>2</sub> concentrations in the study area (second draft ISA, sections 5.2.1.2 and 3.4.1.3).

5 Further, not only is SO<sub>2</sub> but one component of a complex mixture of pollutants present in  
6 the ambient air, an issue not unique to SO<sub>2</sub> epidemiological studies, but SO<sub>2</sub> is also a precursor to  
7 sulfate, which can be a principal component of PM, an air pollutant commonly occurring across  
8 the U.S. This uncertainty affects the extent to which effect estimates from epidemiologic studies  
9 reflect the independent contribution of SO<sub>2</sub> to the adverse respiratory outcomes assessed in these  
10 studies. This area of uncertainty was recognized in the last review and remains in the current  
11 review. In first summarizing the epidemiological evidence from the last review, the second draft  
12 ISA indicated that it was strongest for increased respiratory symptoms and respiratory-related  
13 hospital admissions and ED visits, especially in children, while noting that “a key uncertainty  
14 was potential confounding by copollutants, particularly PM” (second draft ISA, p. 5-5). With  
15 regard to the newly available evidence, the second draft ISA states that “[t]he caution expressed  
16 in the 2008 SO<sub>x</sub> ISA (U.S. EPA, 2008d) related to the limitation of attributing an independent  
17 effect to SO<sub>2</sub> (due to the relationship of SO<sub>2</sub> levels to PM levels) is still a concern” (second draft  
18 ISA, p. 5-144).<sup>34</sup>

19 There remains uncertainty in the evidence with regard to the potential role of long-term  
20 exposure to SO<sub>2</sub> in eliciting SO<sub>2</sub>-related respiratory effects. As noted in section 3.2.1.1 above,  
21 the ISA has determined the evidence to be suggestive of this being a causal relationship. The  
22 strongest evidence supporting this conclusion is provided by epidemiological study findings of  
23 associations between long-term SO<sub>2</sub> concentrations and increases in asthma incidence combined  
24 with findings of laboratory animal studies involving newborn rodents that indicate a potential for  
25 SO<sub>2</sub> exposure to contribute to the development of asthma, especially allergic asthma, in children.  
26 However, uncertainties that relate to the limitations of the animal toxicological evidence,  
27 particularly for long-term exposure, and the potential for confounding by other pollutants is  
28 unexamined, and largely unavailable, for epidemiologic studies of asthma among children  
29 (second draft ISA, section 5.2.2.7).

30 Another area of uncertainty recognized by the ISA, is that contributing to conclusions  
31 regarding the potential for SO<sub>2</sub> in ambient air to contribute to health effects other than respiratory

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<sup>34</sup> A few recent epidemiologic studies add evidence for SO<sub>2</sub> in copollutant models with PM, NO<sub>2</sub>, or O<sub>3</sub>, although the pollutants are measured at central site monitors (second draft ISA, p. 5-8). Across the full epidemiologic evidence base, some associations were relatively unchanged in magnitude after adjustment for a copollutant, while others did not persist. However, the second draft concludes that “inference from copollutant models is limited given potential differences in exposure measurement error for SO<sub>2</sub> compared to NO<sub>2</sub>, CO, PM, and O<sub>3</sub> and in many cases, high copollutant correlations” (second draft ISA, p. 5-139).

1 effects. As noted in section 3.2.1.1 above, the ISA has determined the evidence to be suggestive  
2 of, but insufficient to infer, a causal relationship between short-term SO<sub>2</sub> exposure and mortality  
3 and to be inadequate to infer the presence or absence of a causal relationship for other types of  
4 exposures and health effects for which there are studies available.

5 In summary, a variety of uncertainties from the last review remain, including those  
6 related to the extent of effects at concentrations below those evaluated in controlled human  
7 exposure studies of exercising individuals with asthma, and the potential for greater impacts in  
8 individuals with more severe asthma and in children with asthma (second draft ISA, section  
9 5.2.1.9).

### 10 **3.2.1.5 Public Health Implications**

11 Implications and the magnitude of potential impacts on public health are dependent upon  
12 the type and severity of the effect, as well as the size of population affected. With regard to SO<sub>2</sub>  
13 concentrations in ambient air, the public health implications and potential public health impacts  
14 relate to the effects causally related to SO<sub>2</sub> exposures of interest in this review. These are  
15 respiratory effects of short-term exposures, and particularly those effects associated with asthma  
16 exacerbation in people with asthma. As summarized in section 3.2.1.1, the most strongly  
17 demonstrated effects are bronchoconstriction-related effects resulting in decrements in lung  
18 function elicited by short term exposures during periods of elevated ventilation, while asthma-  
19 related health outcomes such as ED visits and hospital admissions have also been statistically  
20 associated with ambient air SO<sub>2</sub> concentration metrics in epidemiological studies (second draft  
21 ISA, section 5.2.1.9).

22 In considering public health implications, in addition to the difference in severity of  
23 different effects, it is important to consider aspects of the same effect with regard to its impact on  
24 population groups of differing susceptibility. For example, with regard to bronchoconstriction-  
25 related effects, the same percentage increase in sRaw or reduction in FEV<sub>1</sub> for two groups of  
26 individuals that differ in their baseline sRaw or FEV<sub>1</sub> may result in the two groups being affected  
27 differently with regard to increased susceptibility to other physiological threats or challenges.  
28 Accordingly, consideration of such baseline differences and also the relative transience or  
29 persistence of such sRaw or FEV<sub>1</sub> changes, as well as other factors, is important to  
30 characterizing implications for public health, as recognized by the American Thoracic Society in  
31 their statements on evaluating adverse health effects of air pollution (ATS, 2000; Thurston et al.,  
32 2017).

33 The most recent policy statement by the ATS on what constitutes an adverse health effect  
34 of air pollution provides a general framework for interpreting evidence that proposes a “set of  
35 considerations that can be applied in forming judgments” for this context (Thurston et al., 2017).

1 The earlier ATS statement, in addition to emphasizing clinically relevant effects, also  
2 emphasized both the need to consider changes in “the risk profile of the exposed population,”  
3 and effects on the portion of the population that may have a diminished reserve that puts its  
4 members at potentially increased risk if affected by another agent (ATS, 2000). These concepts,  
5 including the consideration of the magnitude of effects occurring in just a subset of study  
6 subjects, continue to be recognized as important in the more recent ATS statement (Thurston et  
7 al., 2017) and continue to be relevant to the evidence base for SO<sub>2</sub>.<sup>35</sup>

8 As summarized in section 3.2.1.3 above, people with asthma are the key population at  
9 risk for SO<sub>2</sub>-related effects and children with asthma are considered to be at relatively greater  
10 risk than other age groups within this at-risk population (second draft ISA, section 6.3.1). In  
11 recognizing that asthma as a disease can vary in its severity, we take note of the lack of evidence  
12 for individuals with the most severe asthma. The evidence base of controlled exposure studies of  
13 exercising people with asthma provides limited information that indicates there to be similar  
14 relative responses of individuals with differences in severity of their asthma,<sup>36</sup> although the  
15 evidence indicates that the absolute changes in lung function are larger for individuals with more  
16 severe asthma compared to those characterized as having mild asthma. It is uncertain whether a  
17 greater response to the exercise itself (vs the SO<sub>2</sub> exposure) played a role in such findings,  
18 however the available studies “suggest that adults with moderate/severe asthma may have more  
19 limited reserve to deal with an insult compared with individuals with mild asthma” (second draft  
20 ISA, p. 5-20; Linn et al., 1987; Trenga et al., 1999).

21 The information below characterizes the size and other features of the populations in the  
22 U.S. concluded to be at risk of SO<sub>2</sub>-related effects, when under elevated ventilation conditions.  
23 As a whole, the discussion in this section indicates the potential for exposures to SO<sub>2</sub> in ambient  
24 air to be of appreciable public health importance. Such considerations contributed to the basis for  
25 the 2010 decision to appreciably strengthen the primary SO<sub>2</sub> NAAQS and to establish a 1-hour  
26 standard to protect the at-risk populations from short term exposures of concern. Such  
27 considerations remain relevant in the current review.

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<sup>35</sup> In the Administrator’s judgments on the then-existing standard in the last review, as well as on the appropriate level for the new 1-hour standard, the Administrator considered the 2000 ATS policy statement, as well as advice from CASAC and recommendations and judgments made by EPA in previous NAAQS reviews (section 3.1.1 above).

<sup>36</sup> These studies categorized with regard to asthma severity based mainly on the individual’s use of medication to control asthma, such that individuals not regularly using medication were classified as minimal/mild, and those regularly using medication as moderate/severe (Linn et al., 1987). The second draft ISA indicates that the moderate/severe grouping would likely be classified as moderate by today’s asthma classification standards due to the level to which their asthma was controlled and ability to engage in moderate to heavy levels of exercise (second draft ISA, p. 5-20 to 5-21; Johns et al., 2010; Reddel, 2009).

- **What does the information available in this review indicate with regard to the size of at-risk populations and their distribution in the U.S.?**

The magnitude and characterization of a public health impact is dependent upon the size and characterization of the populations affected, as well as the type or severity of the effect. As summarized above, the population group most at risk of health effects associated with exposure to SO<sub>2</sub> in ambient air is people with asthma. The National Center for Health Statistics data for 2015 indicate that approximately 8.0% of the U.S. population has asthma (Table 3-2; CDC, 2017). These data indicate the size of the key at-risk population for SO<sub>2</sub> in ambient air. It is this population that the primary NAAQS for SO<sub>2</sub> is intended to protect. Table 3-2 below considers the currently available information that helps to characterize key features of this population.

The age group for which the prevalence documented by these data is greatest is children aged five to 19, with 10.2% of children aged 15 to 19 years having asthma. In 2012 (the most recent year for which such an evaluation is available), it was the leading chronic illness affecting children (Bloom et al., 2013). The prevalence is greater for boys than girls. Among populations of different races or ethnicities, black non-Hispanic children have the highest prevalence, at 13.4%. Asthma prevalence is also increased among populations in poverty (e.g., 11.1% among people living in households below the poverty level compared to 7.2% of those living above it). Population groups with relatively greater asthma prevalence might be expected to have a relatively greater potential for SO<sub>2</sub> impacts.

1 **Table 3-2. 2015 National Asthma Prevalence.**

Characteristic <sup>1</sup>	Number with Current Asthma (in thousands) <sup>2</sup>	Percent with Current Asthma
<b>Total</b>	<b>24,633</b>	<b>7.8</b>
Child (Age <18)	6,188	8.4
Adult (Age 18+)	18,445	7.6
<b>All Age Groups</b>		
0-4 years	935	4.7
5-14 years	4,033	9.8
15-19 years	2,107	10.2
20-24 years	1,655	7.6
25-34 years	2,916	6.8
35-64 years	9,907	8.0
65+ years	3,079	6.6
<b>Child Age Group</b>		
0-4 years	935	4.7
5-11 years	2,761	9.6
12-17 years	2,492	10.3
<b>Sex</b>		
Males	9,998	6.5
Boys (Age <18)	3,705	9.9
Men (Age 18+)	6,293	5.4
Females	14,634	9.1
Girls (Age <18)	2,483	6.9
Women (Age 18+)	12,151	9.7
<b>Race/Ethnicity</b>		
White NH <sup>3</sup>	15,244	7.8
Child (Age <18)	2,810	7.4
Adult (Age 18+)	12,435	7.9
Black NH	3,931	10.3
Child (Age <18)	1,336	13.4
Adult (Age 18+)	2,595	9.1
Other NH	1,793	6.9
Child (Age <18)	605	8.4
Adult (Age 18+)	1,188	6.3
Hispanic	3,665	6.6
Child (Age <18)	1,438	8.0
Adult (Age 18+)	2,227	5.9
<b>Federal Poverty Threshold</b>		
Below 100% of poverty level	5,086	11.1
100% to less than 250% of poverty level	7,664	8.4
250% to less than 450% of poverty level	4,989	6.3
450% of poverty level or higher	6,894	6.9

<sup>1</sup> Numbers within selected characteristics may not sum to total due to rounding

<sup>2</sup> Includes persons who answered "yes" to the questions "Have you EVER been told by a doctor or other health professional that you had asthma" and "Do you still have asthma?"

<sup>3</sup> NH = non-Hispanic

Adapted from [https://www.cdc.gov/asthma/most\\_recent\\_data.htm](https://www.cdc.gov/asthma/most_recent_data.htm)

### 3.2.2 Exposure/Risk-based Considerations

Our consideration of the scientific evidence available in the current review, as at the time of the last review (summarized in section 3.1 above), is informed by results from a quantitative analysis of estimated population exposure and associated risk. The overarching consideration is whether the current exposure/risk information alters our overall conclusions from the previous review regarding health risk associated with exposure to SO<sub>2</sub> in ambient air. As in our consideration of the evidence in section 3.2.1 above, we have organized the discussion regarding the exposure/risk information around a set of key questions to assist us in considering the exposure/risk analyses of at-risk populations living in three urban areas under air quality conditions simulated to just meet the existing SO<sub>2</sub> primary standard.

Prior to addressing the individual exposure/risk questions, we provide a summary of key aspects of the assessment, including the study areas, populations simulated, modeling tools and exposure and risk metrics derived (section 3.2.2.1). We then consider aspects of the questions beginning with the magnitude of exposure and risk estimated for the simulated at-risk populations (section 3.2.2.2), followed by the key uncertainties associated with the quantitative analyses with regard to drawing conclusions as to the adequacy of protection afforded by the current SO<sub>2</sub> standard (section 3.2.2.3). Lastly, we consider the exposure and risk estimates from the quantitative assessment with regard to the extent to which such estimates may be judged to be important from a public health perspective (section 3.2.2.4).

#### 3.2.2.1 Exposure/risk analyses

In the assessment conducted for this review, described in detail in the draft REA, we have estimated SO<sub>2</sub> exposure and risk associated with air quality conditions that just meet the current standard. These analyses inform our understanding of the protection provided by the current SO<sub>2</sub> standard from effects that the health effects evidence indicates to be elicited in some portion of exercising people with asthma by short (e.g., 5 to 10 minutes) elevations in SO<sub>2</sub> exposure concentrations. The analyses estimate exposure and risk for at-risk populations in three urban study areas in: (1) Fall River, MA; (2) Indianapolis, IN; and, (3) Tulsa, OK.

The three study areas present a variety of circumstances with regard to population exposure to short-term peak concentrations of SO<sub>2</sub> in ambient air. This set of study areas and the associated exposed populations are intended to be informative to the EPA's consideration of potential exposures and risks that may be associated with the air quality conditions that meet the current SO<sub>2</sub> standard. As discussed further in sections 3.2.2.2 and 3.2.2.4 below, the Fall River study area is found to present particularly informative exposure circumstances given that it provides an example of an area in the U.S. in which there is substantial overlap between locations that are relatively more populated and where SO<sub>2</sub> concentrations are relatively higher

(draft REA, section 5.4). As such, this area represents places in the U.S. with the potential for exposures of greatest concern, making it important in considering the protection provided by the current standard.

The three study areas range in total population size from approximately 180,000 to 540,000 and reflect different mixtures of SO<sub>2</sub> emissions sources, including utilities using fossil fuel and non-utility sources, such as petroleum refineries and secondary lead smelting (draft REA, section 3.1). They include locations in New England, Ohio River Valley and the Midwest, the latter two regions comprising the part of the U.S. with generally the greatest prevalence of elevated SO<sub>2</sub> concentrations and large emissions sources (Figure 2-7, Appendix F). Additionally, continuous 5-minute ambient air monitoring data (i.e., all 12 5-minute values for each hour) are available in two of the three study areas, with hourly maximum 5-minute concentration data available in the third (draft REA, section 3.2).

Asthma prevalence estimates for the populations simulated in the three study areas ranges from 8.0 to 8.7% (draft REA, section 5.1). For children, the study area prevalence rates range from 9.7 to 11.2% (draft REA, section 5.1). Variation within each study related to age, sex and whether family income is above or below the poverty level was also accounted for (section 4.1.2 and Appendix E of draft REA).<sup>37</sup> This variation is greatest in the Fall River study area, with census block level, age-specific prevalence estimates ranging from 7.9 to 18.6% for girls and from 10.7 to 21.5% for boys (draft REA, Table 4-1).

In the draft REA, 1-hour SO<sub>2</sub> concentrations were estimated across a 3-year period (consistent with the period represented by the form of the standard) using air quality modeling of SO<sub>2</sub> emissions sources in each area, and were adjusted, as described in the draft REA, such that the air quality modeling receptor location with the highest concentrations just met the current standard.<sup>38</sup> In addition, sensitivity analyses were performed using an alternative adjustment approach and are summarized in section 3.2.2.2. Relationships between 1-hour and 5-minute concentrations at local monitors were then used to estimate 5-minute concentrations associated with the adjusted 1-hour concentrations across the 3-year period at all receptor locations in each area (draft REA, section 3.5).

The exposure modeling, presented in detail in the draft REA, relied on the EPA's Air Pollutant Exposure model (APEX), which estimates human exposure using a stochastic, event-based microenvironmental approach. This model has a history of application, evaluation, and

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<sup>37</sup> As described in section 4.1.2 and Appendix E of the draft REA, asthma prevalence in the exposure modeling domain is estimated based on national prevalence information and study area demographic information related to age, sex and poverty status.

<sup>38</sup> As described in more detail in section 3.4 of the draft REA, the adjustments were implemented with a focus on reducing emissions from the source contributing to the standard exceedances until the areas just met the standard.

1 progressive model development in estimating human exposure and dose for reviews of NAAQS  
2 for gaseous pollutants (U.S. EPA, 2008; 2010; 2014). This general exposure modeling approach  
3 was also used in the 2009 REA for the last review of the primary standard for SO<sub>x</sub>, although a  
4 number of updates have been made to the model and various datasets used with it (2009 REA;  
5 U.S. EPA, 2017b, section 3.4). For example, exposure modeling for the draft REA includes  
6 reliance on updates to several key inputs to the model including (1) a significantly expanded  
7 Consolidated Human Activity Database (CHAD), that now has over 55,000 diaries, with over  
8 25,000 for school-aged children; (2) the updated NHANES data (2009-2014), which are the basis  
9 for the age- and sex-specific body mass distributions from which APEX samples to specify the  
10 individuals in the modeled population; (3) the algorithms used to estimate age- and sex-specific  
11 resting metabolic rate, a key input to estimating a simulated individual's activity-specific  
12 ventilation rate; and (4) the ventilation rate algorithm itself. Further, the current model uses  
13 updated population demographic data based on the most recent Census.

14 The APEX model probabilistically generates a sample of hypothetical individuals from  
15 an actual population database and simulates each individual's movements through time and  
16 space (e.g., indoors at home, inside vehicles) to estimate his or her exposure to a pollutant.  
17 Population characteristics are taken into account to represent the demographic profile of the  
18 population in each study area. Age and gender demographics for the simulated at-risk population  
19 (adults and children with asthma) were drawn from the prevalence estimates provided by the  
20 2011-2015 National Health Interview Survey.<sup>39</sup> The APEX model generates each simulated  
21 person or profile by probabilistically selecting values for a set of profile variables, including  
22 demographic variables, status and physical attributes (e.g., residence with air conditioning,  
23 height, weight, body surface area) and ventilation rate.

24 Based on minute-by-minute activity levels, and physiological characteristics of the  
25 simulated person (see draft REA, section 4.1), APEX estimates an equivalent ventilation rate  
26 (EVR), based on normalizing the simulated individuals' activity-specific ventilation rate to their  
27 body surface area; the EVR is used to identify exposure periods during which an individual is at  
28 or above a specified ventilation level (draft REA, section 4.1.4.4). The level specified is based on  
29 the ventilation rates of subjects in the controlled human exposure studies of exercising people  
30 with asthma (Table 3-1). The APEX simulations performed for this review have focused on

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<sup>39</sup> Information about the National Health Interview Survey is available at <http://www.cdc.gov/nchs/nhis.htm>.

1 exposures to SO<sub>2</sub> emitted into ambient air that occurs in microenvironments,<sup>40</sup> without additional  
2 contribution from indoor SO<sub>2</sub> emissions sources.<sup>41</sup>

3 As in the last review, the draft REA for this review uses the APEX model estimates of 5-  
4 minute exposure concentrations for simulated individuals with asthma at elevated ventilation to  
5 characterize health risk in two ways based on information from the controlled human exposure  
6 studies on the occurrence of bronchoconstriction-related effects in some study subjects with  
7 asthma who are exposed during exercise (draft REA, section 4.5). In drawing on this evidence  
8 base for this purpose, the draft REA has given primary focus to the well-documented studies  
9 summarized in Table 5-2 and Figure 5-1 of the second draft ISA for 5- to 10-minute exposure  
10 concentrations ranging from 200 ppb to 600 ppb (Table 3-1 of this document). The first risk  
11 metric is based on comparison of the estimated 5-minute exposure concentrations individuals at  
12 elevated ventilation to 5-minute concentrations of potential concern (benchmark concentrations),  
13 and the second utilizes exposure-response information for study subjects experiencing  
14 bronchoconstriction-related effects on lung function (specifically a doubling or more in sRaw) to  
15 estimate the portion of the simulated at-risk population likely to experience one or more days  
16 with an SO<sub>2</sub>-related increase in sRaw of at least 100%. Both of these metrics are used in the draft  
17 REA to characterize health risk associated with 5-minute peak SO<sub>2</sub> exposures among the  
18 simulated at-risk population during periods of elevated ventilation. These risk metrics were also  
19 derived in the REA for the last review and the associated estimates informed the Administrator's  
20 2010 decision on the new standard (75 FR 35546-35547, June 22, 2010).

21 For the benchmarks metric, the draft REA for this review, like the 2009 REA in the last  
22 review, uses benchmark concentrations that range from 400 ppb down to 100 ppb (draft REA,  
23 section 4.5.1). At the upper end of this range, 400 ppb represents the lowest concentration in  
24 free-breathing controlled human exposure studies of exercising people with asthma where  
25 moderate or greater lung function decrements occurred that were often statistically significant at  
26 the group mean level and were frequently accompanied by respiratory symptoms. The 200 ppb  
27 benchmark concentration represents the lowest level tested in studies where subjects were freely  
28 breathing in exposure chambers (moderate or greater lung function decrements in some of these

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<sup>40</sup> Five microenvironments (MEs) are modeled in the draft REA as representative of a larger number of microenvironments. The 2009 REA results indicated that the majority of peak SO<sub>2</sub> exposures occurred while individuals were within outdoor microenvironments (2009 REA, Figure 8-21). Based on that finding and the objective (i.e., understanding how often and where short-term peak SO<sub>2</sub> exposures occur), the approach implemented in the draft REA recognizes the added efficiency of minimizing the number of MEs, particularly indoor MEs, that are parameterized and included in the modeling. Accordingly, the number of MEs was aggregated to address exposures of ambient origin that occur within a core group of indoor, outdoor, and vehicle MEs (draft REA, section 4.2).

<sup>41</sup> Indoor sources are generally minor in comparison to SO<sub>2</sub> from ambient air (draft REA, sections 2.1.1 and 2.1.2).

1 subjects) (75 FR 35527, June 22, 2010). The lowest benchmark concentration (100 ppb), which  
2 is one half the lowest exposure concentration tested in free breathing exposure studies, has been  
3 included in consideration of the nonzero percentage of subjects with asthma experiencing  
4 moderate transient decrements in lung function at the 200 ppb exposure concentration  
5 (approximately 8 to 9%) and the lack of specific study data for some groups of individuals with  
6 asthma, such as primary-school-age children and those with more severe asthma.<sup>42</sup>

7 The exposure-response (E-R) function for the risk of lung function decrements was  
8 developed from the individual subject results for sRaw from the controlled exposure studies of  
9 exercising freely breathing people with asthma exposed to SO<sub>2</sub> concentrations from 1000 ppb  
10 down as low as 200 ppb (draft REA, Table 4-9). Beyond the assessment of these studies and  
11 their results in past reviews, there has been extensive evaluation of the individual subject results,  
12 including a data quality review in the last SO<sub>2</sub> NAAQS review (Johns and Simmons, 2009), and  
13 detailed analysis in two subsequent publications (Johns et al., 2010; Johns and Linn, 2011). The  
14 sRaw responses reported in these studies have been summarized in the second draft ISA, as in  
15 the last review, in terms of percent of study subjects experiencing responses of a magnitude  
16 equal to a doubling or tripling or more. Across the exposure range from 200 to 1000 ppb, the  
17 percentage of exercising study subjects with asthma having at least a doubling of sRaw increases  
18 from about 8-9% (at exposures of 200 ppb) up to approximately 50-60% (at exposures of 1000  
19 ppb) (draft REA, Table 4-9). The E-R function used in the main analysis of the draft REA was  
20 derived from these data using a probit function (draft REA, section 4.5.2).

21 In summary, while the general approach and methodology for the exposure-based  
22 assessment in this review is similar to that in the last review, there are a number of ways in  
23 which these analyses differ (see 2009 REA and draft REA for this review). In addition to the  
24 expansion in the number and type of study areas assessed, we note the number of improvements  
25 to input data and modeling approaches, including the availability of continuous 5-minute air

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<sup>42</sup> Recognizing that even the study subjects described as “moderate/severe” group (had well-controlled asthma, were generally able to withhold medication, were not dependent on corticosteroids, and were able to engage in moderate to heavy levels of exercise) would likely be classified as moderate by today’s classification standards (second draft ISA, pp. 5-20 to 5-21; Johns et al., 2010; Reddel, 2009), we have considered the evidence with regard to the response of individuals with severe asthma that are not generally represented in the full set of controlled human exposure studies. There is no evidence to indicate such individuals would experience moderate or greater lung function decrements at lower SO<sub>2</sub> exposure concentrations than individuals with moderate asthma. With regard to the severity of the response, the limited data that are available indicate a similar magnitude SO<sub>2</sub>-specific response (in sRaw) as that for individuals with less severe asthma, although the individuals with more severe asthma are indicated to have a greater response to exercise prior to SO<sub>2</sub> exposure, indicating that those individuals “may have more limited reserve to deal with an insult compared with individuals with mild asthma” (second draft ISA, p. 5-21). As noted in sections 3.2.1.3 and 3.2.1.4 above, evidence from controlled human exposure studies are not available for children younger than 12 years old, and the second draft ISA indicates that the information regarding behavior and methacholine responsiveness for the subset of this age group that is of school age (e.g., 5-12 years) indicates a potential for greater response (second draft ISA, pp. 5-21 to 5-24).

monitoring data at monitors within two of the three study areas. The current draft REA extends the time period of simulation to a 3-year simulation period, consistent with the form established for the now-current standard. Further, the years simulated reflect more recent emissions and circumstances subsequent to the 2010 decision.

### **3.2.2.2 At-risk population exposures and risk**

In this section, we summarize the exposure and risk estimates from the draft REA and consider the following question.

- **What is the magnitude of population exposure and risk in at-risk populations in areas simulated to just meet the current SO<sub>2</sub> standard? What portion of the at-risk populations are estimated to experience exposures of concern or lung function decrements at levels of potential health concern?**

In addressing these questions, we consider the population estimates provided by the draft REA simulations of exposure to SO<sub>2</sub> emitted into ambient air (draft REA, Chapters 5 and 6). In considering these REA estimates for air quality conditions just meeting the current standard, we particularly focus on the extent of protection provided by the standard from SO<sub>2</sub> exposures of potential concern. As described in the prior section, the draft REA presents two sets of risk estimates for the 3-year simulation in each study area: (1) the number (and percent) of simulated persons experiencing exposures at or above the particular benchmark concentrations of interest, while at elevated ventilation; and (2) the number and percent of people estimated to experience at least one SO<sub>2</sub>-related lung function decrement in a year and the number and percent of people experiencing multiple lung function decrements associated with SO<sub>2</sub> exposures.

In presenting the exposure and risk estimates, the draft REA recognizes that the approach applied to adjust air quality to conditions just meeting the current standard can have important impacts on the risk and exposures estimates (draft REA, section 6.2.2). Because of this, the draft REA presents results for two different approaches to adjusting air quality. The first approach uses the highest design value across all modeled air quality receptors to adjust the air quality concentrations in each area to just meet the standard (draft REA, section 3.4). This is done by estimating the amount of SO<sub>2</sub> concentration reduction needed for this highest receptor to be adjusted to the current SO<sub>2</sub> standard, and based on this amount, all other receptors impacted by the highest source(s) are adjusted accordingly. The second approach is included as a sensitivity analysis that recognizes the potential uncertainty associated with the modeled concentrations, particularly the very highest modeled concentrations. Accordingly, the second approach uses the air quality receptor having the 99<sup>th</sup> percentile of the distribution of design values (instead of the receptor having the maximum design value) to estimate the SO<sub>2</sub> concentration reductions needed to adjust the air quality to just meet the standard (draft REA, section 6.2.2.1). In study areas in which modeled concentrations at a very small number of receptors are substantially higher than

1 those at all other air quality receptors, these two different approaches can result in very different  
2 SO<sub>2</sub> concentrations across an area. In such study areas, in particular, the first approach generally  
3 results in much more significant reductions being applied to reduce SO<sub>2</sub> concentrations at the  
4 small group of highest modeled receptors such that concentrations at those receptors are just at or  
5 just below the standard and concentrations at the other receptors across the area are appreciably  
6 lower. Given that these two approaches to adjusting air quality can result in important  
7 differences in the magnitude of risk to at-risk populations in areas simulated to just meet the  
8 current standard, the tables below present estimates based on both approaches.<sup>43</sup>

9       Of the two types of risk metrics derived in the draft REA, we turn first to the results for  
10 the benchmark-based risk metric with regard to the percent of the study area populations with  
11 asthma estimated to experience at least one daily maximum 5-minute exposure per year at or  
12 above the different benchmark concentrations while at elevated ventilation (Table 3-3). Under air  
13 quality conditions just meeting the current standard across the three study areas, approximately  
14 20 to 25% of children with asthma, on average across the 3-year period, are estimated to  
15 experience one or more days per year with a 5-minute exposure at or above 100 ppb while  
16 breathing at elevated ventilation rates (Table 3-3). With regard to the 200 ppb benchmark, as  
17 many as 0.7 percent of the simulated population of children with asthma, on average across the  
18 3-year period, was estimated to experience a single day with a 5-minute exposure at or above  
19 200 ppb while breathing at elevated ventilation rates (Table 3-3). The percentage in a single year  
20 ranged up to 2.2% for a single day, while less than 0.1% of children with asthma were estimated  
21 to experience more than a single day with an exposure at or above 200 ppb while at elevated  
22 ventilation (draft REA, Tables 6-5 and 6-6). No simulated children with asthma were estimated  
23 to experience a day with a 5-minute exposure at or above 300 or 400 ppb. The estimates for  
24 adults are lower, generally due to the lesser amount and frequency of time spent outdoors (draft  
25 REA, section 5.2).

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<sup>43</sup> Details regarding these sensitivity analyses focused on the impact of the adjustment approach are presented in the draft REA, section 6.2.2.1.

**Table 3-3. Air quality conditions adjusted to just meet the current standard: Percent of simulated populations of children with asthma estimated to experience at least one daily maximum 5-minute exposure per year at or above indicated concentrations while at elevated ventilation.**

5-minute Exposure Concentration (ppb)	Percent (%) of Population of Children (5-18 years) with Asthma Average per year <sup>A</sup>		
	Fall River, MA	Indianapolis, IN	Tulsa, OK
≥ 100	19.4 – 26.7	<0.1 <sup>B</sup> – 0.1	0.1 – 0.4
≥ 200	<0.1 – 0.7	0 – <0.1	0
≥ 300	0	0	0

<sup>A</sup> The values presented in each cell are the average of the results for the three years simulated based on the two approaches to air quality adjustment (drawn from Table 6-5 of the draft REA).

<sup>B</sup> <0.1 is used to represent nonzero estimates below 0.1%. A value of zero (0) indicates there were no individuals having the selected exposure in any year.

We next consider the estimates for risk of lung function decrements in terms of a doubling or more in sRaw (Table 3-4). Under conditions just meeting the current standard in the three study areas, as many as 1.1% of children with asthma, on average across the 3-year period, were estimated to experience at least one day per year with a SO<sub>2</sub>-related increase in sRaw of 100% or more in the study area with the highest estimates (Table 3-4, Fall River). The corresponding percent estimated to experience two or more such days ranged as high as 0.6%, on average across the 3-year simulation period (draft REA, Table 6-8). Additionally, in the same study area, as much as 0.2% of the simulated populations of children with asthma, on average across the 3-year period, was estimated to experience a single day with a SO<sub>2</sub>-related increase in sRaw of 200% or more. The estimates for adults are very slightly lower, again, generally due to the lesser time spent outdoors (draft REA, section 5.3).

**Table 3-4. Air quality conditions adjusted to just meet the current standard: Percent of simulated population of children with asthma estimated to experience at least one day per year with a SO<sub>2</sub>-related increase in sRaw of 100% or more.**

Lung function decrement (increase in sRaw)	Percent (%) of Population of Children (5-18 years) with Asthma <sup>A</sup> Average per year		
	Fall River, MA	Indianapolis, IN	Tulsa, OK
≥ 100%	0.9 – 1.1	0	<0.1 <sup>B</sup> - <0.1
≥ 200%	0.1 – 0.2	0	0
<sup>A</sup> The values presented in each cell are the average of the results for the three years simulated based on two approaches to air quality adjustment (drawn from Table 6-7 of the draft REA). <sup>B</sup> <0.1 is used to represent nonzero estimates below 0.1%. A value of zero (0) indicates there were no individuals estimated to have the selected decrement in any year.			

In understanding these results, we note that the three study areas selected provide a variety of circumstances with regard to population exposure to short-term peak concentrations of SO<sub>2</sub> in ambient air. These three study areas reflect different combinations of different types of SO<sub>2</sub> emissions sources, including utilities using fossil fuels and non-utility sources, and provide three different patterns of exposure to SO<sub>2</sub> concentrations in a populated area in the U.S. In this way, the three areas provide a variety of examples of exposure patterns that can be informative to the EPA's consideration of potential exposures and risks that may be associated with air quality conditions occurring under the current SO<sub>2</sub> standard. As indicated by the discussion above and also recognized in section 3.2.2.3 below, there is variability in the estimated magnitude of exposure and associated risk across study areas and uncertainties associated with these estimates.

In developing the air quality scenarios for the current standard in the three study areas, the draft REA recognizes that these scenarios of adjusted air quality provide representations of the pattern of air quality that might occur in each study area under conditions that just meet the current standard. Where such conditions include relatively large spatial extents of higher concentrations – i.e., areas with design values in proximity to the level of the standard – that overlap with the more populated parts of the study area, exposure and risk results are relatively higher (draft REA, section 5.4). Among the three study areas, this best describes the Fall River study area, which is an area where source characteristics contribute to a sizeable spread of source-influenced relatively higher concentrations that coincide or overlap with locations where people reside and/or frequent. This association between concentrations and population in Fall River is illustrated in Figure 5-4 of the draft REA. Inclusion of an area with these characteristics in the REA provides some insight into the potential exposure and risk associated with other areas across the U.S. with similar characteristics and is therefore particularly informative to evaluation of the level of protection provided by the standard.

1 The other two study areas (Indianapolis and Tulsa) provide examples of areas where the  
2 higher SO<sub>2</sub> concentrations that result from the sizeable SO<sub>2</sub> sources in the study area do not  
3 strongly coincide with parts of the area in which people reside and/or frequent (draft REA,  
4 section 5.4). This relationship between SO<sub>2</sub> concentrations and population in these two areas is  
5 illustrated in Figures 5-5 and 5-6 of the draft REA. Accordingly, the corresponding exposure and  
6 risk estimates for these areas are lower than those estimated for the Fall River study area, even  
7 though the populations are larger (draft REA, sections 5.1 and 5.4).

8 As discussed above, among the three study areas, the Fall River study area presents the  
9 exposure circumstances associated with highest SO<sub>2</sub>-related exposures and risk for the current  
10 standard air quality scenario. Because of this, we recognize that the Fall River study area is of  
11 particular importance in considering the adequacy of the protection afforded by the current  
12 standard. Recognizing this, we note that the draft REA indicates that the percent of children with  
13 asthma that might be expected to experience 5-minute SO<sub>2</sub> concentrations at or above the 200  
14 ppb benchmark concentration, in an urban area that just meets the current standard, may be as  
15 high as 0.7%, on average across the three years, and 2.2% in a single-year period. With regard to  
16 the lung function risk, the draft REA indicates the percent of children that might be expected to  
17 experience at least a doubling of specific airway resistance, under conditions just meeting the  
18 current standard, may be as high as 1.1%, on average across the three-year period, and 1.9% in a  
19 single year. Thus, these results indicate that, in the single year with the highest concentrations  
20 across the 3-year period, nearly 98% of the population of children with asthma in the Fall River  
21 study area, would not be expected to experience a day with a 5-minute exposure at or above the  
22 200 ppb and 400 ppb benchmarks and would not be expected to experience as much as a  
23 doubling in sRaw, a magnitude roughly consistent with the level of protection that was described  
24 in establishing the now-current standard in 2010 (as summarized in section 3.1.1.2.4 above).<sup>44</sup>  
25 On average across the 3-year period, the corresponding percentage is nearly 99% (Tables 3-3 and  
26 3-4, above).

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<sup>44</sup> Although the 2009 REA did not include an air quality scenario representing the now-current standard, among the scenarios it did include were single-year air quality scenarios representing standard levels of 100 and 50 ppb. For the single-year scenario representing a standard level of 100 ppb in the study area with the highest population exposure and risk, the 2009 REA estimated 2.7% of children with asthma to experience at least one day with exposure at or above 200 ppb, while at elevated ventilation (2.1-2.9% to experience one or more SO<sub>2</sub>-attributable increases in sRaw of at least 100%); this estimate was 0.09% for the scenario representing a standard level of 50 ppb (0.4-0.9% to experience one or more SO<sub>2</sub>-attributable increases in sRaw of at least 100%) (2009 REA, Table 9-8 and Appendix B). While we recognize a number of differences between the 2009 REA and the quantitative modeling and analyses performed in the current draft REA, we note that the single year estimates for the Fall River study area in the current draft REA fall between the estimates for the two most similar air quality scenarios assessed in the last review.

### 3.2.2.3 Uncertainties

In this section, we consider the uncertainties associated with the quantitative estimates of exposure and risk, including those recognized by the characterization of uncertainty in the draft REA (draft REA, section 6.2). The characterization in the draft REA is based on an approach intended to identify and compare the relative impact that important sources of uncertainty may have on the exposure and risk estimates. The approach used has been applied in REAs for past NAAQS reviews for ozone, nitrogen oxides, carbon monoxide (U.S. EPA, 2008; 2010; 2014) and SO<sub>x</sub> (U.S. EPA, 2009). In the characterization of uncertainty for the current analysis, the draft REA utilized a qualitative uncertainty characterization approach adapted from the WHO approach for characterizing uncertainty in exposure assessment (WHO, 2008) accompanied by quantitative sensitivity analyses of key aspects of the assessment approach. This characterization and analyses are described in detail in chapter 6 of the draft REA. The approach used in the draft REA varies from that of WHO (2008) in that the draft REA approach placed a greater focus on evaluating the direction and the magnitude of the uncertainty (i.e., qualitatively rating how the source of uncertainty, in the presence of alternative information, may affect the estimated exposures and health risk results).

The characterization and analyses in the draft REA involve consideration of the various types of inputs and approaches that together result in the exposure and risk estimates for the three study areas. In so doing, the draft REA considers the limitations and uncertainties underlying these inputs and approaches and the extent of their influence on the resultant exposure/risk estimates. Consistent with the WHO (2008) guidance, the overall impact of the uncertainty is scaled by considering the extent or magnitude of the impact of the uncertainty as implied by the relationship between the source of the uncertainty and the exposure/risk output. The draft REA also evaluated the direction of influence, indicating how the source of uncertainty was judged to affect the exposure/risk estimates (e.g., likely to over- or under-estimation).

- **What are the key uncertainties associated with the exposure and risk estimates, including those of particular significance with regard to drawing conclusions as to the adequacy of the protection afforded by the current SO<sub>2</sub> standard?**

Based on the uncertainty characterization and associated analyses in the draft REA and consideration of associated policy implications, we recognize several areas of uncertainty as particularly important in our consideration of the exposure and risk estimates, as was also the case in the last review. Generally, these areas include estimation of the spatial distribution of SO<sub>2</sub> concentrations across each study area under air quality conditions just meeting the existing standard, including the fine-scale temporal pattern of 5-minute concentrations. We additionally recognize the uncertainty with regard to population groups and exposure concentrations for which the health effects evidence base is limited or lacking.

1 With regard to the spatial distribution of SO<sub>2</sub> concentrations, the draft REA recognizes  
2 some uncertainty associated with the approach used to adjust the air quality surface to  
3 concentrations just meeting the current standard. Accordingly, the draft REA has investigated the  
4 potential quantitative impact of this uncertainty on the exposure and risk estimates by deriving  
5 estimates based on an alternative adjustment approach (described in section 6.2.2.1 of the draft  
6 REA). Given the results of this sensitivity analysis, we have considered estimates from both  
7 approaches in summarizing the draft REA estimates in section 3.2.2.2 above. Additionally, we  
8 recognize uncertainty in the estimates of 5-minute concentrations in ambient air across the  
9 modeling receptors in each study area. While the ambient air monitoring dataset available to  
10 inform these estimates is much expanded in this review over the dataset available in the last  
11 review, we are still drawing on relationships occurring at one location and over one range of  
12 concentrations to estimate the fine-scale temporal pattern in concentrations at other locations.  
13 This is an important area of uncertainty in the draft REA results because the ambient air 5-  
14 minute concentrations are integral to the 5-minute estimates of exposure. While we recognize  
15 this as an important area of uncertainty, the approach used has taken into account the currently  
16 available information and is considered to provide a reasonable representation of fine-scale  
17 temporal variability in the three study areas.

18 An additional area of uncertainty affecting our interpretation of the exposure and risk  
19 results for the set of study areas assessed concerns our understanding of, and the prevalence  
20 across the U.S. of, the different exposure circumstances they represent. As noted in section  
21 3.2.2.2, the circumstances particularly pertinent to consideration of the adequacy of protection of  
22 the current standard include those in which areas where populations reside and/or exercise  
23 overlap with concentrations of SO<sub>2</sub> that are near, albeit just under, the level of the standard. Such  
24 circumstances are influenced by source characteristics and meteorological conditions, as well as  
25 housing and recreational area patterns in urban areas. While there is some uncertainty in our  
26 understanding of the prevalence of the exposure circumstances represented by the three study  
27 areas, including those for the Fall River study area, the available information indicates there to  
28 be many densely-populated areas in the U.S. in which there are facilities with sizeable SO<sub>2</sub>  
29 emissions (e.g., Appendix F).<sup>45</sup>

30 We also recognize an important area of uncertainty that is particular to our interpretation  
31 of the lung function risk estimates. This area concerns estimates of lung function risk derived for  
32 exposure concentrations below those represented in the evidence base. The exposure-response

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<sup>45</sup> Although source characteristics and meteorological conditions - in addition to magnitude of emissions - influence the distribution of concentrations in ambient air, Appendix F focuses on the distribution of large sources, rather than ambient concentrations, due to limitations in the available information with regard to spatial (and temporal) patterns of SO<sub>2</sub> concentrations in the proximity of such sources in urban areas (second draft ISA, section 2.5.2.2).

1 function on which the primary risk estimates are based generates non-zero predictions of a  
2 percent of the at-risk population exposure expected to experience a day with at least a doubling  
3 of sRaw for all exposures experienced while at elevated ventilation. In considering these  
4 estimates, we recognize that the uncertainty in the response estimates increases substantially with  
5 decreasing exposure concentration below those supported by study data. In so doing, we note the  
6 contribution to the risk estimates of exposure concentrations below 200 ppb. Additionally, we  
7 note that the assessment focuses on the daily maximum 5-minute exposure during elevated  
8 ventilation, summarizing results in terms of the days on which the magnitude of such exposure  
9 exceeds a benchmark or contributes to increased sRaw. While the health effects evidence  
10 indicates the lack of a cumulative effect of multiple exposures over several hours or a day  
11 (second draft ISA, section 5.2.1.2), and a reduced response to repeated exercising exposure  
12 events over an hour (second draft ISA, section 5.2.1.2; Kehrl et al., 1987), information is  
13 somewhat limited with regard to the length of time after recovery from one exposure by which a  
14 repeat exposure would elicit a similar effect as that of the initial event.

15 Another area of uncertainty, which remains from the last review and is important to our  
16 consideration of the draft REA results, concerns the extent to which the quantitative results  
17 represent the populations at greatest risk of effects associated with exposures to SO<sub>2</sub> in ambient  
18 air. As recognized in sections 3.2.1.1 and 3.2.1.4, the controlled human exposure study evidence  
19 base does not include studies of children younger than 12 years old or studies of people with  
20 more severe asthma.<sup>46</sup> The limited evidence that informs our understanding of potential risk to  
21 these groups indicates the potential for them to experience greater effects than other population  
22 groups with asthma under similar exposure circumstances, as summarized in section 3.2.1.4  
23 above. Further we note the lack of information on the factors contributing to increased  
24 susceptibility to SO<sub>2</sub>-induced bronchoconstriction among some people with asthma. Thus, there  
25 is uncertainty associated with our interpretation of the exposure/risk estimates with regard to the  
26 extent to which they represent the populations at greatest risk of SO<sub>2</sub>-related respiratory effects  
27 that is important to consideration of the exposure and risk results with regard to the adequacy of  
28 protection provided by the current standard.

29 In summary, among the multiple uncertainties and limitations in data and tools that affect  
30 the quantitative estimates of exposure and risk and their interpretation in the context of  
31 considering the current standard, we recognize several here as particularly important. These  
32 include uncertainties related to estimation of 5-minute concentrations in ambient air; the lack of  
33 information from controlled human exposure studies for the lower, more prevalent,

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<sup>46</sup> We additionally recognize that limitations in the activity pattern information for children younger than five years old precluded their inclusion in the populations of children simulated in the draft REA.

1 concentrations of SO<sub>2</sub> and limited information regarding multiple exposure episodes within a  
2 day; the prevalence of different exposure circumstances represented by the three study areas;  
3 and, characterization of particular subgroups of people with asthma that may be at greater risk.

#### 4 **3.2.2.4 Potential public health implications**

5 In considering public health implications of the quantitative exposure and risk estimates  
6 that may inform the Administrator's judgments in this area, this section discusses the information  
7 pertaining to the following question.

- 8 • **To what extent are the estimates of exposures and risks to at-risk populations that**  
9 **remain under conditions just meeting the current SO<sub>2</sub> standard important from a**  
10 **public health perspective?**

11 Several factors are important to consideration of public health implications. These  
12 include the magnitude or severity of the effects associated with the exposures estimated in the  
13 draft REA, as well as their adversity at the individual and population scale. Other important  
14 considerations include the size of the population estimated to experience such effects or to  
15 experience exposures associated with such effects. These considerations are discussed below.

16 Based on the currently available evidence which is largely consistent with that available  
17 in the last review (as summarized in section 3.2.1 above), the quantitative exposure and risk  
18 analyses focus on the potential for lung function decrements in people with asthma exposed to  
19 SO<sub>2</sub> while at elevated ventilation. Additionally, we have again focused on estimates for two  
20 types of risk metrics, one involving comparison to benchmark concentrations and the second  
21 involving estimates of lung function risk with regard to moderate or greater increases in sRaw. In  
22 considering these estimates, we recognize that although the lung function decrements, which are  
23 related to bronchoconstriction, are expected to be transient, we additionally recognize that such  
24 decrements, while occurring, may contribute to a diminished reserve in lung function (second  
25 draft ISA, p. 1-17, section 5.2.1.2). For population groups already at diminished reserve, such as  
26 those with more severe asthma, this may be particularly important. Thus, the discussion here  
27 reflects consideration of the health evidence, and exposure and risk estimates, as well as the  
28 consideration of potential public health implications in previous NAAQS decisions and ATS  
29 policy statements (as also discussed in section 3.2.1.5).

30 In light of the conclusion that among all people with asthma, children may be particularly  
31 at risk (summarized in section 3.2.1.2 above) and the draft REA findings of higher exposures and  
32 risks for children (in terms of percent of that population), we have focused the discussion here on  
33 children. We recognize that the draft REA results for air quality conditions just meeting the  
34 current standards indicate that, on average across the 3-year period simulated (consistent with the  
35 form of the current standard), less than 1% of the simulated population of children with asthma

1 might be expected to experience a single day with a 5-minute exposure at or above 200 ppb  
2 while breathing at elevated ventilation rates. The draft REA simulations also estimated no  
3 children with asthma to experience any days in a 3-year simulation period with a 5-minute  
4 exposure at or above 300 or 400 ppb. With regard to the lowest benchmark considered (100 ppb),  
5 the draft REA also indicates that in some areas of the U.S., approximately 25% of children with  
6 asthma, on average across the 3-year period, might be expected to experience one or more days  
7 per year with a 5-minute exposure at or above 100 ppb while breathing at elevated ventilation  
8 rates, with higher percentages in some years. With regard to estimates of lung function  
9 decrements, the draft REA indicates that in some areas, approximately 1% of children with  
10 asthma, on average across a 3-year period, might be expected to experience at least one day per  
11 year with a SO<sub>2</sub>-related increase in sRaw of 100% or more; the estimate for two or more days is  
12 appreciably lower, at 0.4% (draft REA, Table 6-8). Additionally, under such conditions (just  
13 meeting the current standard), the estimated percent of children with asthma that might be  
14 expected to experience a single day per year with a SO<sub>2</sub>-related increase in sRaw of 200% or  
15 more, on average across the 3-year period, is 0.2% (Table 3-4).

16 In considering the severity of responses associated with the REA estimates, we take note  
17 of the health effects evidence for the different benchmark concentrations and judgments made  
18 with regard to the severity of these effects in the last review. As in the last review, we recognize  
19 that the responses documented for exposures of 400 ppb are frequently accompanied by  
20 respiratory symptoms and thus are appropriately considered to be adverse respiratory effects  
21 consistent with past and recent ATS position statements. With regard to the lower benchmark  
22 concentration of 200 ppb, we recognize that, while the responses documented in studies of  
23 exercising subjects with asthma are not consistently accompanied by respiratory symptoms,  
24 conclusions in past NAAQS reviews recognized that moderate decrements in lung function can  
25 be clinically significant in some individuals with asthma (75 FR 35526, June 22, 2010).  
26 Accordingly, the Administrator in the last review considered effects associated with exposures as  
27 low as 200 ppb to be adverse in light of CASAC advice,<sup>47</sup> ATS statements and conclusions in  
28 past NAAQS reviews. While noting the lack of information for some populations groups with  
29 asthma, including primary-school-age children and people with more severe asthma, we  
30 additionally recognize the uncertainty with regard to effects that might be associated with  
31 exposures as low as 100 ppb (as discussed in section 3.2.1.3 and 3.2.1.4 above).

32 As indicated in section 3.1 above, the at-risk population in this review is people with  
33 asthma, as was the case in the last review. Further, children with asthma are identified as

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<sup>47</sup> The CASAC letter on the first draft SO<sub>2</sub> REA to the Administrator stated: “CASAC believes strongly that the weight of clinical and epidemiology evidence indicates there are detectable clinically relevant health effects in sensitive subpopulations down to a level at least as low as 0.2 ppm SO<sub>2</sub>” (Henderson, 2008).

1 particularly at risk. The size of the at-risk population in the U.S. is substantial. As summarized in  
2 section 3.2.1.5, nearly eight percent of the U.S. population (more than 24 million people) and  
3 8.4% of U.S. children have asthma. The prevalence in U.S. child populations of different races or  
4 ethnicities ranges from 7.4% to 13.4% (Table 3-2 above). This is well reflected in the draft REA  
5 study areas in which the asthma prevalence ranged from 8% to 8.7% of the total populations and  
6 9.7% to 11.2% of the children, with the highest prevalence represented in the Fall River study  
7 area. In Fall River, the prevalence varies among census tracts, with the highest tract having a  
8 prevalence in boys of 21.5% (draft REA, Table 4-1).

9 In considering the three study areas and the variation in exposure/risk estimates among  
10 them, we recognize the Fall River study area to be particularly informative to consideration of  
11 public health risk associated with and public health protection provided by the current standard.  
12 This is because, as summarized in section 2.2.2.2 above, the source characteristics and  
13 population distribution in the area cause the locations of relatively higher SO<sub>2</sub> concentrations in  
14 ambient air across the area (i.e., those closest to just meeting the standard) to overlap with  
15 locations of higher population density. These exposure circumstances contribute to higher  
16 exposure and risk estimates than in the other study areas (draft REA, section 5.4), making  
17 estimates for Fall River important in considering the adequacy of protection provided by the  
18 current standard. Thus, we have given particular attention to estimates for this study area.

19 Although exposure and risk estimates were not available in the last review for air quality  
20 conditions just meeting the now-current standard, the findings and considerations summarized  
21 here are generally similar to those considered in the last review, and indicate a level of protection  
22 consistent with that described in the 2010 decision. The exposure and risk estimates for the three  
23 study areas assessed in the draft REA for this review reflect differences in exposure  
24 circumstances among those areas and illustrate the exposures and risk that might be expected to  
25 occur in other areas with such circumstances under air quality conditions that just meet the  
26 current standard. Thus, the draft REA estimates indicate the magnitude of exposure and risk that  
27 might be expected in some areas and illustrate the importance to consideration of the public  
28 health protection afforded by the current standard of those areas where locations of relatively  
29 higher SO<sub>2</sub> concentrations in ambient air across the area coincide with the locations of higher  
30 population density. These considerations, and others raised above, are important to conclusions  
31 regarding the public health significance of the draft REA results. We recognize that such  
32 conclusions also depend in part on public health policy judgments that will weigh in the  
33 Administrator's decision in this review with regard to the adequacy of protection afforded by the  
34 current standard. Such judgments include those concerning the public health significance of  
35 effects at exposures for which evidence is limited or lacking, such as effects at the lower

1 benchmark concentrations considered and lung function risk estimates associated with exposure  
2 concentrations lower than those tested in the controlled exposure studies.

### 3 **3.2.3 Preliminary Staff Conclusions on the Current Standard**

4 This section describes preliminary staff conclusions regarding the adequacy of the current  
5 primary SO<sub>2</sub> standard. These preliminary conclusions are based on considerations described  
6 above, and in the discussion below regarding the currently available scientific evidence (as  
7 summarized in the second draft ISA, and the ISA and AQCDs from prior reviews), and the risk  
8 and exposure information drawn from the draft REA. Conclusions in the final PA will draw upon  
9 the final ISA, developed in consideration of CASAC review and public comment on the second  
10 draft ISA, and on the final REA, developed in consideration of CASAC review and public  
11 comment on the draft REA. Further, staff conclusions presented in the final PA will take into  
12 account advice from the CASAC and public comment on the draft PA and on these preliminary  
13 conclusions.

14 Taking into consideration the discussions responding to specific questions above in this  
15 and the prior chapter, this section addresses the following overarching policy question.

- 16 • **Does the currently available scientific evidence- and exposure/risk-based**  
17 **information, as reflected in the ISA and REA, support or call into question the**  
18 **adequacy of the protection afforded by the current SO<sub>2</sub> standard?**

19 In considering this question, we recognize as an initial matter that, as is the case in  
20 NAAQS reviews in general, the extent to which the current primary SO<sub>2</sub> standard is judged to be  
21 adequate will depend on a variety of factors inclusive of science policy judgments and public  
22 health policy judgments. These factors include public health policy judgments concerning the  
23 appropriate benchmark concentrations on which to place weight, as well as judgments on the  
24 public health significance of the effects that have been observed at the exposures evaluated in the  
25 health effects evidence. The factors relevant to judging the adequacy of the standards also  
26 include the interpretation of, and decisions as to the weight to place on, different aspects of the  
27 results of the exposure assessment for the three areas studied and the associated uncertainties.  
28 Thus, we recognize that the Administrator's conclusions regarding the adequacy of the current  
29 standard will depend in part on public health policy judgments, science policy judgments  
30 regarding aspects of the evidence and exposure/risk estimates, as well as, judgments about the  
31 level of public health protection with an adequate margin of safety that is requisite under the  
32 Clean Air Act.

33 Our response to this question takes into consideration the discussions that address the  
34 specific policy-relevant questions in prior sections of this document (see sections 3.2.1-3.2.2)  
35 and the approach described in section 3.1 that builds on the approach from the last review. We

1 focus first on consideration of the evidence, including that newly available in this review, and the  
2 extent to which it alters key conclusions supporting the current standard. We then turn to  
3 consideration of the quantitative exposure and risk estimates drawn from the draft REA,  
4 including associated limitations and uncertainties, and the extent to which they indicate differing  
5 conclusions regarding the magnitude of risk, as well as level of protection from adverse effects,  
6 associated with the current standard. We additionally consider the key aspects of the evidence  
7 and exposure/risk estimates emphasized in establishing the now-current standard, and the  
8 associated public health policy judgments and judgments about the uncertainties inherent in the  
9 scientific evidence and quantitative analyses that are integral to decisions on the adequacy of the  
10 current primary SO<sub>2</sub> standard.

11 In considering the currently available evidence, staff gives great weight to the long-  
12 standing body of health effects evidence for SO<sub>2</sub>, augmented in some aspects since the last  
13 review, that provides the foundation of our understanding of the health effects of SO<sub>2</sub> in ambient  
14 air. In so doing, we give particular attention to the evidence from controlled human exposure  
15 studies that demonstrates that very short exposures to less than 1000 ppb SO<sub>2</sub>, while breathing at  
16 elevated ventilation rates, induces bronchoconstriction in some people with asthma; and,  
17 supports the identification of people with asthma as the population at risk from short-term peak  
18 concentrations in ambient air (second draft ISA; 2008 ISA; 1994 AQCD supplement).

19 Further, while the evidence base has been augmented since the time of the last review, we  
20 note that the newly available evidence does not lead to different conclusions regarding the  
21 primary health effects of SO<sub>2</sub> in ambient air or regarding exposure concentrations associated  
22 with those effects; nor does it identify different populations at risk of SO<sub>2</sub> -related effects. In this  
23 way, the health effects evidence available in this review is consistent with evidence available in  
24 the last review when the current standard was established. This strong evidence base continues to  
25 demonstrate a causal relationship between short-term SO<sub>2</sub> exposures and respiratory effects,  
26 particularly in people with asthma. This conclusion is primarily based on evidence from  
27 controlled human exposure studies available at the time of the last review that reported lung  
28 function decrements and respiratory symptoms in people with asthma exposed to SO<sub>2</sub> for 5 to 10  
29 minutes while breathing at elevated ventilation. Support is provided by the epidemiological  
30 evidence that is coherent with the controlled exposure studies. The epidemiological evidence,  
31 including that recently available, includes studies reporting positive associations for asthma-  
32 related hospital admissions and emergency department visits (of individuals of all ages,

1 including adults and children) with short-term SO<sub>2</sub> exposures (second draft ISA, section  
2 5.2.1.2).<sup>48</sup>

3 The health effects evidence newly available in this review also does not extend our  
4 understanding of the range of 5-minute exposure concentrations eliciting effects in people with  
5 asthma exposed while breathing at elevated ventilation rates beyond what was understood in the  
6 last review. As in the last review, 200 ppb remains the lowest concentration tested in exposure  
7 studies where study subjects are freely breathing in exposure chambers. At that exposure  
8 concentration, approximately eight to nine percent of study subjects with asthma, breathing at  
9 elevated ventilation rates, experienced moderate or greater lung function decrements following  
10 5- to 10-minute controlled exposures. The limited information available for lower exposure  
11 concentrations, while not amenable to direct quantitative comparisons, generally indicates  
12 somewhat lesser response. In considering what may be gleaned from the epidemiological  
13 evidence with regard to exposure concentrations eliciting effects, we recognize complications  
14 associated with interpretation of epidemiologic studies of SO<sub>2</sub> in ambient air that relate to  
15 whether measurements at the study monitors adequately represent the spatiotemporal variability  
16 in ambient SO<sub>2</sub> concentrations in the study areas and associated population exposures (second  
17 draft ISA, section 5.2.1.9).

18 In this review, as in the last review, we recognize some uncertainty with regard to  
19 exposure levels eliciting effects in some population groups not studied, such as individuals with  
20 severe asthma, as well as uncertainty in the extent of effects at exposure levels below those  
21 studied. Collectively, these aspects of the evidence and associated uncertainties contribute to a  
22 recognition that for SO<sub>2</sub>, as for other pollutants, the available evidence base in a NAAQS review  
23 generally reflects a continuum, consisting of ambient levels at which scientists generally agree  
24 that health effects are likely to occur, through lower levels at which the likelihood and magnitude  
25 of the response become increasingly uncertain.

26 As at the time of the last review, the exposure and risk estimates developed from  
27 modeling exposures to SO<sub>2</sub> emitted into ambient air are critically important to consideration of  
28 the potential for exposures and risks of concern under air quality conditions of interest, and  
29 consequently are critically important to judgments on the adequacy of public health protection  
30 provided by the current standard. In considering the public health implications of estimated  
31 occurrences of exposures of different magnitudes, we take note of guidance from the ATS, the  
32 CASAC's written advice and recommendations in past reviews, and judgments made by the EPA  
33 in considering similar effects in previous NAAQS reviews (75 FR 35526 and 35536, June 22,

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<sup>48</sup> While uncertainties remain related to the potential for confounding by PM or other co-pollutants and the representation of fine-scale temporal variation in personal exposures, the findings of the epidemiological evidence continue to provide support for the conclusion on the causal relationship (second draft ISA, section 5.2.1.2).

2010). As recognized in section 3.2.1.5, an additional publication by the ATS that further addresses judgments on what constitutes an adverse health effect of air pollution is newly available in this review (Thurston et al., 2017). The more recent statement expands upon the 2000 statement, that was considered in the last SO<sub>2</sub> NAAQS review, and recognizes additional considerations with regard to such judgments that remain consistent with the EPA's judgments in the 2010 review. In that review, the Administrator judged that the effects reported in exercising people with asthma following 5- to 10-minute SO<sub>2</sub> exposures at or above 200 ppb, and especially at or above 400 ppb (often accompanied by respiratory symptoms and for which the evidence is stronger), can result in adverse health effects (75 FR 35536, June 22, 2010). In so doing, she also recognized that effects reported for exposures below 400 ppb are less severe than those at and above 400 ppb (75 FR 35547, June 22, 2010).

In considering the draft REA analyses available in this review, we are aware of a number of ways in which these analyses differ from those available in the last review. In addition to the expansion in the number and type of study areas assessed, we note the number of improvements to input data and modeling approaches, including the availability of continuous 5-minute air monitoring data at monitors within two of the three study areas. The current draft REA extends the time period of simulation by including a 3-year simulation period consistent with the form established for the now-current standard. Further, the years simulated reflect more recent emissions and circumstances subsequent to the 2010 decision. In considering the draft REA results, we also take note of the array of emissions and exposure circumstances represented by the three study areas. As summarized in section 3.2.2 above, the areas fall into three different geographic regions of the U.S. They range in total population size from approximately 180,000 to approximately one half million, and vary in population demographic characteristics. Additionally, the types of large sources of SO<sub>2</sub> emissions represented in the three study areas vary with regard to emissions characteristics and include EGUs, petroleum refineries, glass-making facilities, secondary lead smelters (from battery recycling), and chemical manufacturing.

As at the time of the last review, people with asthma are the population at risk of SO<sub>2</sub>-related respiratory effects. Children with asthma may be particularly at risk (section 3.2.1.2 above). While there are more adults in the U.S. with asthma than children with asthma, the draft REA results in terms of percent of the simulated at-risk populations, indicates higher exposures and risks for children with asthma as compared to adults. This finding relates to children's greater frequency and duration of occasions outdoors (section 3.2.2.2 above). In light of these conclusions and findings, we have focused our consideration of the draft REA results here on children.

As can be seen by the variation in exposure estimates, the three study areas in the draft REA represent an array of exposure circumstances, including those contributing to relatively

1 higher and relatively lower exposures and associated risk. As recognized in the draft REA, the  
2 analyses there are not intended to provide a comprehensive national assessment. Rather, the  
3 analyses for this array of study areas are intended to indicate the magnitude of exposures and  
4 risks that may be expected in areas of the U.S. that just meet the current standard but that may  
5 differ in ways affecting population exposures of interest. In that way, the draft REA is intended  
6 to be informative to the EPA's consideration of potential exposures and risks associated with the  
7 current standard and the Administrator's decision on the adequacy of protection provided by the  
8 current standard. As discussed in sections 3.2.2.2 and 3.2.2.4 above, consideration of exposures  
9 occurring in those areas where locations of relatively higher SO<sub>2</sub> concentrations in ambient air  
10 across an area that just meets the current standard coincide with the locations of higher  
11 population density are particularly important to consideration of the public health protection  
12 afforded by the current standard.

13 With regard to the draft REA representation of air quality conditions associated with just  
14 meeting the current standard, while we note reduced uncertainty in a few aspects of the approach  
15 for developing this air quality scenario, we recognize the uncertainty associated with the  
16 application of adjustments to the highest model receptor in the study area. As summarized in  
17 sections 3.2.2.2 and 3.2.2.3 above, sensitivity analyses described in section 6.2.2 of the draft  
18 REA indicate the quantitative impact potentially associated with area of uncertainty. Given the  
19 importance of this aspect of the REA to consideration of the level of protection provided by the  
20 current standard, we have considered the results for each study area in terms of a range bounded  
21 on the low end by the results for the main analysis and on the upper end by those based on the  
22 alternative adjustment approach used in the sensitivity analysis. In this context, we note that  
23 across all three study areas, which provide an array of SO<sub>2</sub> emissions and exposure situations, the  
24 percent of children with asthma estimated to experience at least one day with as much as a  
25 doubling in sRaw (attributable to SO<sub>2</sub>), on average across the 3-year period, ranges from 0.9 to  
26 1.1%; the highest estimate is just under 2% for the highest single year. Less than 1% of children  
27 with asthma are estimated to experience, while at elevated ventilation, a daily maximum 5-  
28 minute exposure per year at or above 200 ppb, on average across the 3-year period, with a  
29 maximum of approximately 2% in the highest single year. Further, no child (or adult) with  
30 asthma is estimated to experience, while at elevated ventilation, a daily maximum 5-minute  
31 exposure per year at or above 400 ppb (in any of the three years simulated across the three study  
32 areas). Thus, in light of current ATS guidance, as well as conclusions and CASAC advice in  
33 prior NAAQS reviews, the draft REA exposure and risk estimates for the current review indicate  
34 that the current standard is likely to provide effective protection from SO<sub>2</sub>-related health effects  
35 to at-risk populations of children and adults with asthma.

1 In summarizing the information discussed thus far, we reflect on the key aspects of the  
2 2010 decision that established the current standard. As an initial matter, effects associated with  
3 5- to 10-minute exposures as low as 200 ppb of people with asthma while breathing at elevated  
4 ventilation were considered to be adverse; this judgment was based on consideration of CASAC  
5 advice and EPA decisions in prior NAAQS reviews, as well as ATS guidance. We note that the  
6 newly available information in this review includes an additional statement from ATS on  
7 adversity which is generally consistent with the earlier statement (available at the time of the  
8 2010 decision). While recognizing the differences between the current and past analyses,  
9 including the lack of an air quality scenario specific to the now-current standard in the last  
10 review, as well as uncertainties associated with such analyses, we note a rough consistency of the  
11 associated estimates when considering the array of study areas in both reviews. Overall, the  
12 newly available quantitative analyses appear to comport with the conclusions reached in the last  
13 review regarding control expected to be exerted by the now-current 1-hour standard on 5-minute  
14 exposures of concern. With regard to the results for the REA in the last review (which were for a  
15 single-year simulation), the 2010 decision recognized those results to indicate that a one-hour  
16 standard of 75 ppb might be expected to protect more than 97% of children with asthma (and  
17 somewhat less than 100%) from experiencing exposures at or above a 200 ppb benchmark  
18 concentration, and more than 99% of that population group from experiencing exposures at or  
19 above a 400 ppb benchmark. Single-year results for study areas assessed in the current draft REA  
20 indicate protection of approximately 98 to more than 99% of the populations of children with  
21 asthma from experiencing exposures at or above a 200 ppb benchmark concentration and of all  
22 of the study area at-risk populations from exposures at or above 400 ppb. Additionally, the 2010  
23 decision also took note of the magnitude of the ambient air SO<sub>2</sub> concentrations in U.S.  
24 epidemiological studies of associations between ambient air concentrations and emergency  
25 department visits and hospital admissions, for which the effect estimate remained positive and  
26 statistically significant in copollutant models with PM. In considering these studies, the  
27 Administrator judged that the level chosen for the new 1-hour standard provided an adequate  
28 margin of safety. No additional such studies are available in the current review. Thus, in  
29 considering the key aspects of the decision in the last review, we find the currently available  
30 information to be consistent with that on which the decision establishing the current standard was  
31 based.

32 Based on all of the above, and taking into consideration related information, limitations  
33 and uncertainties, such as those recognized above, we draw preliminary conclusions regarding  
34 the extent to which the newly available information in this review supports or calls into question  
35 the adequacy of protection afforded by the current standard. In considering the conclusions that  
36 may be supported by the exposure and risk estimates, we take note of the more than 24 million

1 people with asthma in the U.S., including more than 6 million children. We additionally note the  
2 uncertainties or limitations of the current evidence base with regard to the exposure levels at  
3 which effects may be elicited in some population groups (e.g., children with asthma and  
4 individuals with severe asthma), as well as the severity of the effects. In so doing, we recognize  
5 that the controlled human exposure studies, on which the depth of our understanding of SO<sub>2</sub>-  
6 related health effects is based, do not provide information with regard to responses in people  
7 with more severe asthma or in children younger than 12 years. Additionally, some aspects of our  
8 understanding continue to be limited; among these aspects are the potential for effects in some  
9 people with asthma exposed to concentrations below 200 ppb, as well as the potential for other  
10 air pollutants to affect responses to SO<sub>2</sub>. In light of this we note the draft REA results for the  
11 lowest benchmark that indicate that in some areas of the U.S. with air quality conditions that just  
12 meet the current standard, approximately 20 to 25% of children with asthma may experience  
13 one or more exposures, on average across a 3-year period, to concentrations at or above 100 ppb  
14 while at elevated ventilation. Thus, the evidence and exposure/risk information related to the  
15 lowest exposures studied lead us to conclude that the combined consideration of the body of  
16 evidence and the quantitative exposure estimates continue to provide support for a standard as  
17 protective as the current one.

18 Further, we recognize that conclusions regarding the adequacy of the current standard  
19 depend in part on public health policy judgments identified above and judgments about the level  
20 of public health protection with an adequate margin of safety. In so doing, we take note of the  
21 long-standing health effects evidence that documents the effects of SO<sub>2</sub> exposures as short as a  
22 few minutes on people with asthma that are exposed while breathing at elevated ventilation rates  
23 and recognize that such effects have been documented in the lowest concentration studied in  
24 exposure chambers with appropriate clean-air controls (200 ppb). In so doing, we recognize the  
25 limitations, and associated uncertainty, in the evidence available for lower exposure  
26 concentrations (e.g., 100 ppb), as was the case in the last review, and we note the lower  
27 responses reported. Thus, in focusing on the potential for 5-minute exposures at and above 200  
28 ppb, that have been previously recognized as adverse (June 22, 2010; 75 FR 35547), we take  
29 note of the draft REA results that indicate the current standard may be expected to protect  
30 approximately 98% to nearly 99% of at-risk populations with asthma from experiencing any  
31 days with such exposures, in a single- and 3-year period, respectively. We additionally note the  
32 draft REA finding of no children (or adults) estimated to experience any days with a 5-minute  
33 exposure of 300 ppb or higher. In light of ATS guidance, CASAC advice and EPA conclusions  
34 in past NAAQS reviews, these results indicate effective protection of at-risk populations from  
35 SO<sub>2</sub>-related health effects that we note is consistent with the level of protection specified when  
36 the standard was set. Thus, we reach the preliminary conclusion that the currently available

evidence and quantitative information, including the associated uncertainties, do not call into question the adequacy of protection provided by the current standard, and thus support consideration of retaining the current standard, without revision.

In summary, the newly available health effects evidence, critically assessed in the ISA as part of the full body of evidence, reaffirms conclusions on the respiratory effects recognized for SO<sub>2</sub> in the last review. Further, we observe the general consistency of the current evidence with the evidence that was available in the last review with regard to key aspects on which the current standard is based. We additionally note the quantitative exposure and risk estimates for conditions just meeting the current standard that indicate a similar level of protection, for at-risk populations from respiratory effects considered to be adverse, as that described in the last review for the now-current standard. We also recognize, as in the last review, the limitations and uncertainties associated with the available information. Collectively, these considerations (including those discussed above) provide the basis for the preliminary staff conclusion that consideration should be given to retaining the current standard, without revision. Accordingly, and in light of this preliminary staff conclusion that it is appropriate to consider the current standard to be adequate, we have not identified any potential alternative standards for consideration in this review.

### **3.3 KEY UNCERTAINTIES AND AREAS FOR FUTURE RESEARCH AND DATA COLLECTION**

In this section, we highlight key uncertainties associated with reviewing and establishing the primary NAAQS for sulfur oxides. Such key uncertainties and areas for future research, model development, and data gathering are outlined below. In some cases, research in these areas can go beyond aiding standard setting to aiding in the development of more efficient and effective control strategies. We note, however, that a full set of research recommendations to meet standards implementation and strategy development needs is beyond the scope of this discussion. Rather, listed below are key uncertainties, research questions and data gaps that have been thus far highlighted in this review of the primary standard.

- A critical aspect of our consideration of the evidence and the quantitative dose estimates is our understanding of SO<sub>2</sub> effects below the lowest concentrations studied in controlled human exposure studies. Additional information related in several areas would reduce uncertainty in our interpretation of the available information for purposes of risk characterization. These areas include the following.
  - Our understanding of whether and to what extent some population groups, including children or people with severe asthma, are more responsive to peak SO<sub>2</sub> exposures (or responsive to lower concentrations), while breathing at elevated ventilation rates, than the groups that have been studied.

- 1           – A better understanding of the effects and the shape of the exposure-response  
2           relationship at lower 5-minute exposure concentrations (i.e., below 200 ppb)  
3           would help to reduced uncertainty in our estimates of lung function effects and,  
4           accordingly, in characterizing SO<sub>2</sub>-related health effects.
- 5           – Little information is available on the factors contributing to the susceptibility to  
6           lower concentrations of SO<sub>2</sub> of a subgroup of people with asthma, termed  
7           “responders” in the second draft ISA (second draft ISA, section 5.2.1.2; Johns and  
8           Linn, 2011).
- 9           – There is also only very limited evidence regarding the potential influence of  
10          history of exposure and co-occurring exposure to other air pollutants, including  
11          particulate matter. Further research is needed in this area to better inform our  
12          characterization of health risk related to SO<sub>2</sub>.
- 13       • An understanding of the fine-scale spatial and temporal gradients of ambient air SO<sub>2</sub>  
14       concentrations in residential areas, as well as near sources of SO<sub>2</sub> emissions, is a key  
15       element in our assessment of exposure and risk. Additional information in this area is  
16       needed. Current limitations in this area additionally contribute to uncertainty in  
17       characterization of ambient air SO<sub>2</sub> levels in the risk assessment and the resulting  
18       exposure and risk estimates. Further characterization of the fine-scale spatial and  
19       temporal variation in ambient air SO<sub>2</sub> concentrations in different environments and  
20       related to different sources, as well as different air quality conditions that just meet the  
21       existing standard, would help to reduce this uncertainty.
- 22       • Uncertainties with regard to other aspects of the health effects evidence include that  
23       regarding what can be gleaned from the epidemiologic studies showing an association  
24       between short-term SO<sub>2</sub> exposures and asthma-related hospital admission and emergency  
25       department visits. Uncertainty remains regarding the extent of copollutant confounding in  
26       these studies, particularly by PM. Additionally, there is uncertainty related to the  
27       representation of exposure through fixed site monitors and capturing peak SO<sub>2</sub>  
28       concentrations that limits the informativeness of the ambient air concentrations analyzed  
29       in the studies to standards reviews.
- 30       • While the CHAD is much expanded over the last review, limited information and  
31       associated uncertainty remain in several aspects of the available data. Additional  
32       information would reduce uncertainty in these aspects of our exposure and risk estimates.
  - 33           – Collection and analysis of multiday activity patterns that consider the attributes  
34           most influential to determining long-term activity patterns. Further research  
35           would assist in better evaluating and improving existing approaches used to  
36           generate longitudinal activity profiles (as discussed in the draft REA, section  
37           4.1.5.1).
  - 38           – Activity data for some population subgroups, such as people with severe asthma  
39           and very young children.

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## APPENDIX A

### PREPARATION OF DATA FILES FOR GENERATION OF FIGURES IN CHAPTER 2

The raw data came from pre-generated AQS extract files. Files are located at [http://www.epa.gov/airquality/airdata/ad\\_data.html](http://www.epa.gov/airquality/airdata/ad_data.html). Documentation of files is located at <http://aqsdrl.epa.gov/aqsweb/aqstmp/airdata/FileFormats.html>. Hourly Data Files were used. A separate Hourly Data File for each parameter and year combination was run. The type of SO<sub>2</sub> data is determined by the parameter code and duration code and is coded as follows:

- 1-hour values data - parameter code = 42401 and duration code = 1
- 5-minute data (12 observations per hour) - parameter code = 42401 and duration code = H
- 5-minute data (hourly max) – parameter code = 42406 and duration code = 1

For the 1-hour data at a Site/POC to be used, it must have met the following completeness criteria:

- 75% or more of the hourly observations in a day (18 or more) must be present.
- 75% or more of the days in a quarter must be present and complete:
- 1<sup>st</sup> Quarter – 68 observations or 69 observations in leap year
  - 2<sup>nd</sup> Quarter – 69 observations
  - 3<sup>rd</sup> Quarter – 69 observations
  - 4<sup>th</sup> Quarter – 69 observations
- 4 quarters for each of at least 3 of the 5 years (2011-2015) must be present and complete. For this analytical purpose, the three years do not have to be consecutive. This dataset was prepared in June 2016.

After completeness criteria were applied, the following data screens were also performed to account for some outliers in the 5-minute data:

- Only 5 minute data with a corresponding hourly value in AQS (parameter 42401 and duration code 1) were kept.
- Only 5 minute values with an hourly mean value under 120% of the hourly value in AQS (parameter 42401 and duration code 1) were kept.
- Only hours where a 5-minute max hourly value (AQS parameter 42406 and duration code 1) was reported and fell between 1 and 12 times the AQS hourly value (parameter 42401 and duration code 1) were kept.

## APPENDIX B

### ADDITIONAL INFORMATION ON DATASETS PRESENTED IN FIGURE 2-8

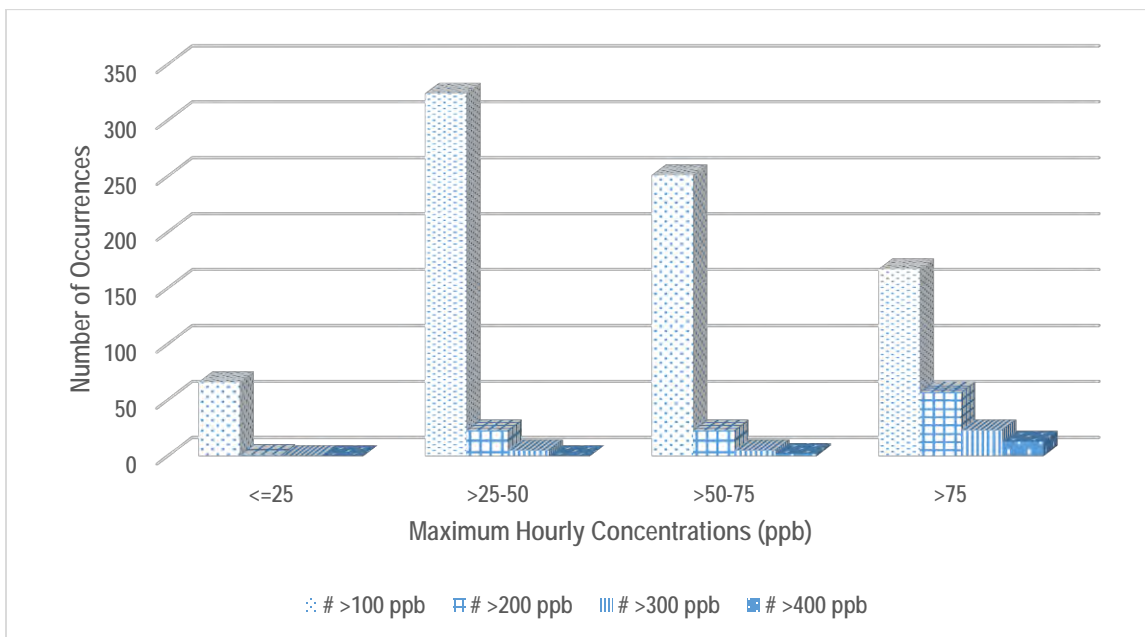
**Table B-1. Summary statistics (in ppb) for distributions of daily maximum 5-minute SO<sub>2</sub> concentrations on days with differing daily maximum 1-hour SO<sub>2</sub> concentrations.**

Bin	Daily Maximum 1-hour Concentration (ppb)			
	<=25	>25-50	>50-75	>75
N	315964	5486	1521	1435
25 <sup>th</sup> percentile	1	46.6	95	170.1
Median	2.0	62	122.6	220
Mean	5	74	140	259
75 <sup>th</sup> percentile	5.2	88	167.2	294.25
95 <sup>th</sup> percentile	22.0	152.5	259	502.9
99 <sup>th</sup> percentile	44	225.6	389	822.5
When the three data sets for sites with DVs at or below 75 ppb are combined, the 99 <sup>th</sup> percentile is 58.3 ppb and the 99.9 <sup>th</sup> percentile is 150 ppb.				

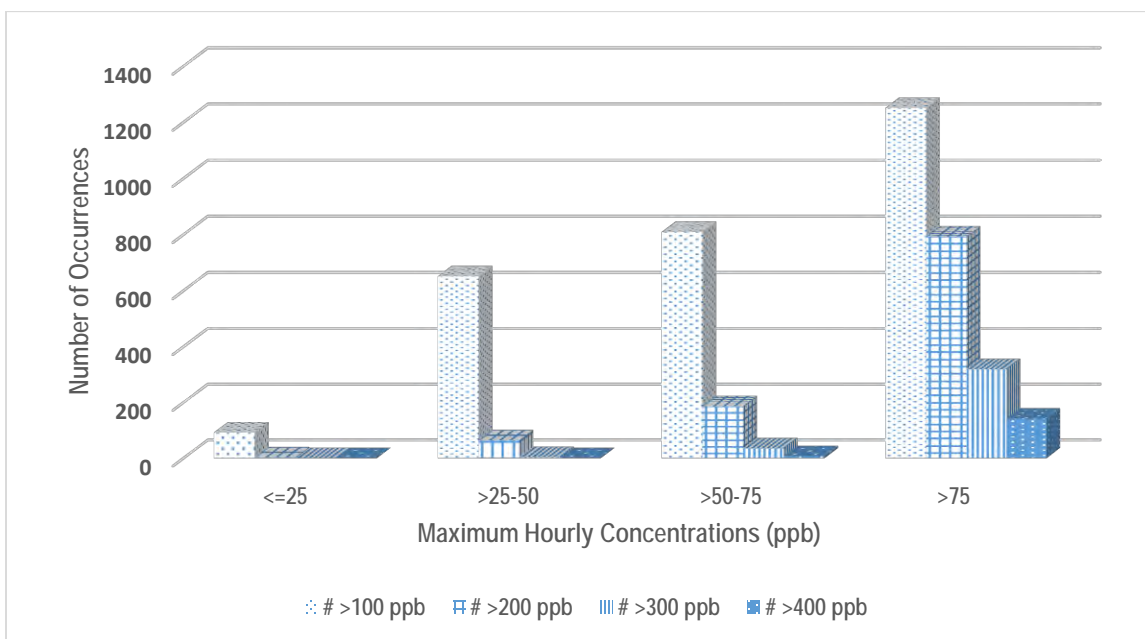
**Table B-2. Summary statistics (in ppb) for distributions of daily maximum 5-minute SO<sub>2</sub> concentrations at sites with differing design values.**

Bin	Design Value (ppb)			
	<=25	>25-50	>50-75	>75
N	225863	59011	18504	21028
25 <sup>th</sup> percentile	0.8	2	2	2.3
Median	1.7	5	7	8
Mean	3	10	20	41
75 <sup>th</sup> percentile	3.8	13	26	40
95 <sup>th</sup> percentile	12.2	37	82.6	199.6
99 <sup>th</sup> percentile	28.3	68	146.1	365

**Figure B-1. Monitoring data for sites meeting the current standard: Frequency of daily maximum 5-minute values on days with differing daily maximum 1-hour concentrations (2013-2015).**



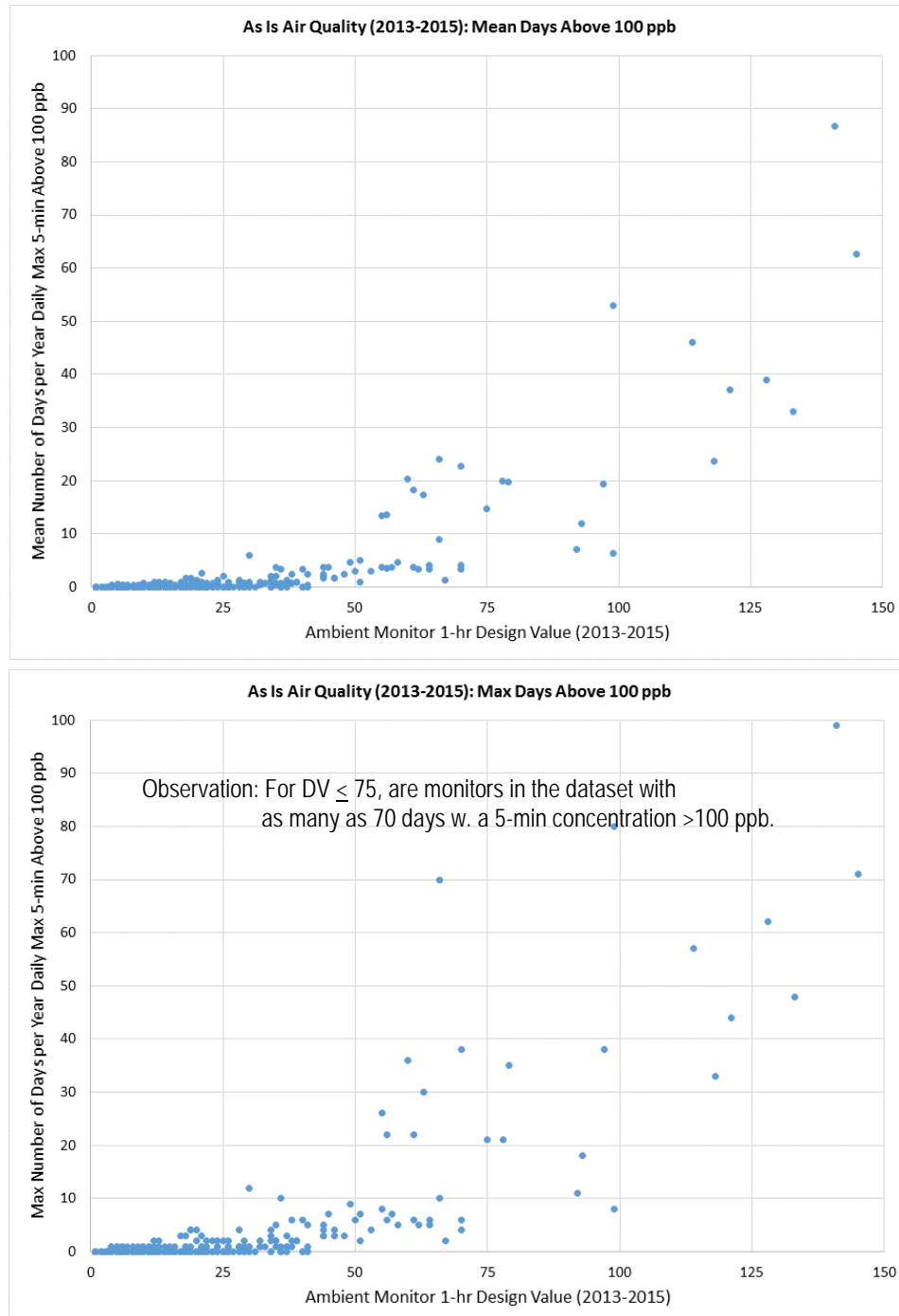
**Figure B-2. Monitoring data for sites not meeting the current standard: Frequency of daily maximum 5-minute values on days with differing daily maximum 1-hour concentrations (2013-2015).**



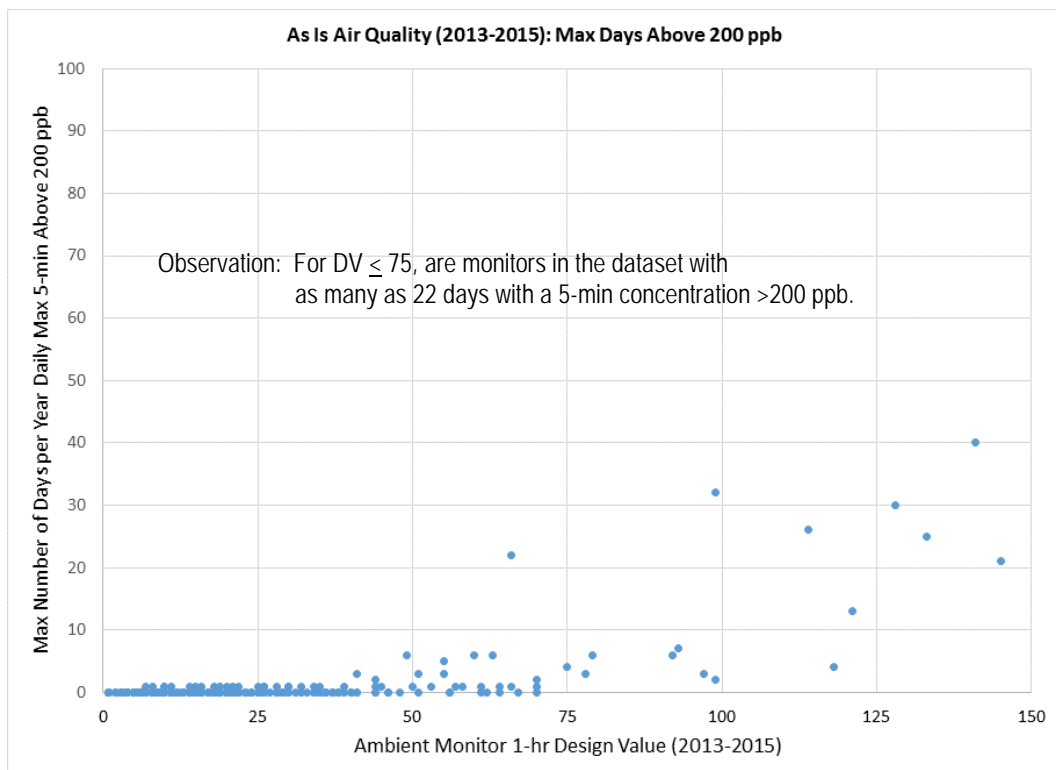
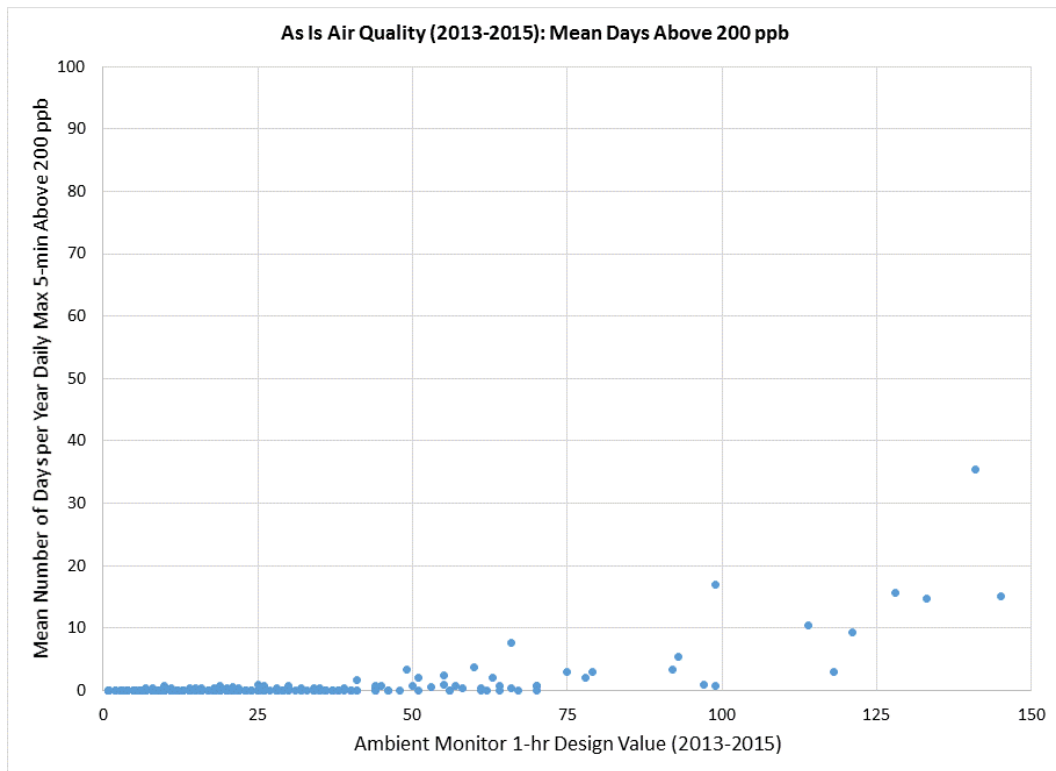
## APPENDIX C

### OCCURRENCES OF 5-MINUTE SO<sub>2</sub> CONCENTRATIONS OF INTEREST IN THE RECENT AMBIENT AIR MONITORING DATA (2013-2015)

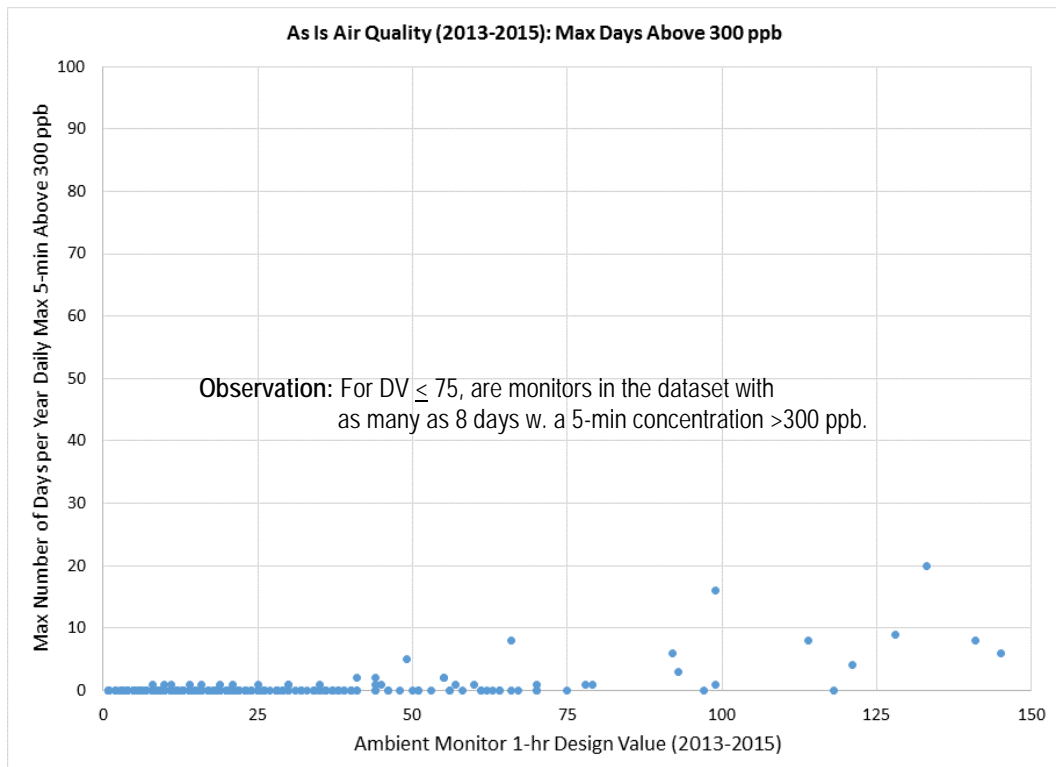
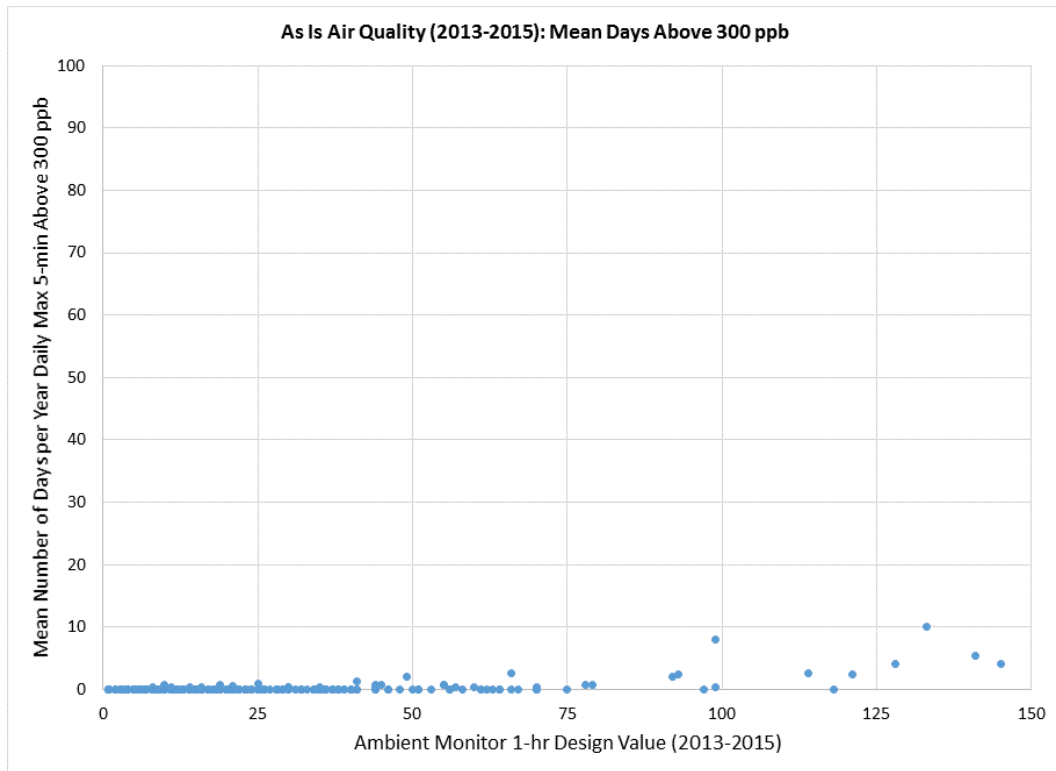
**Figure C-1. As is (unadjusted) SO<sub>2</sub> monitoring data (2013-2015).** Mean number of days/year (top panel) and maximum number of days/year (bottom panel) with daily maximum 5-minute concentrations of SO<sub>2</sub> above 100 ppb.



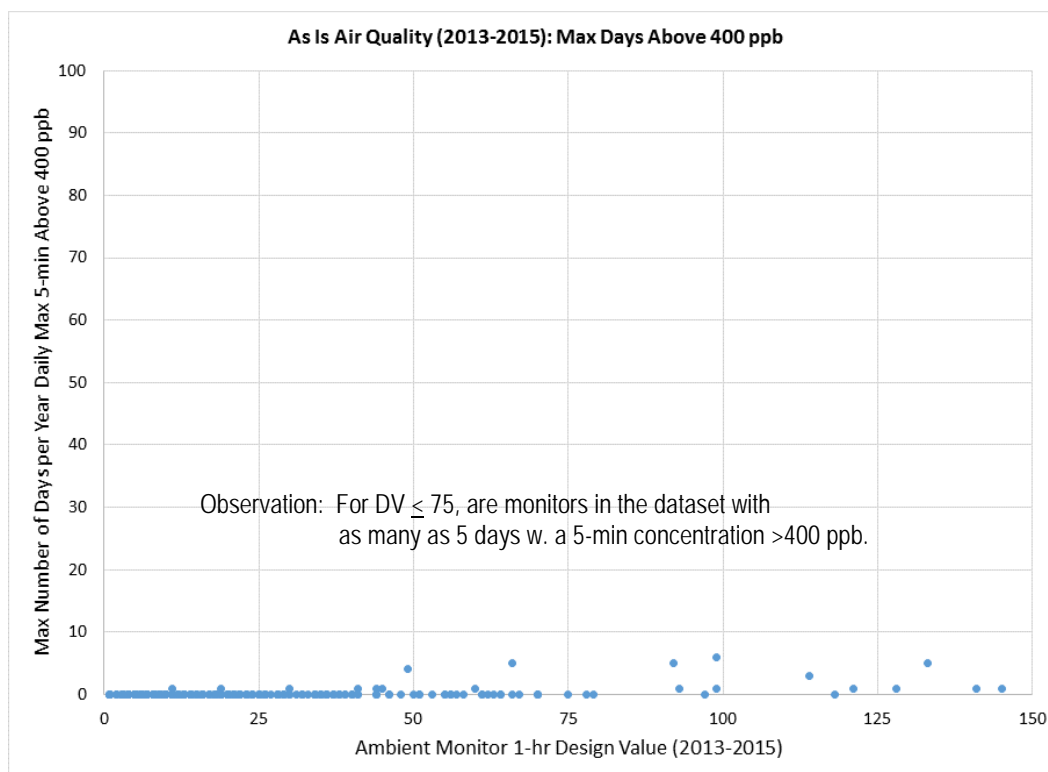
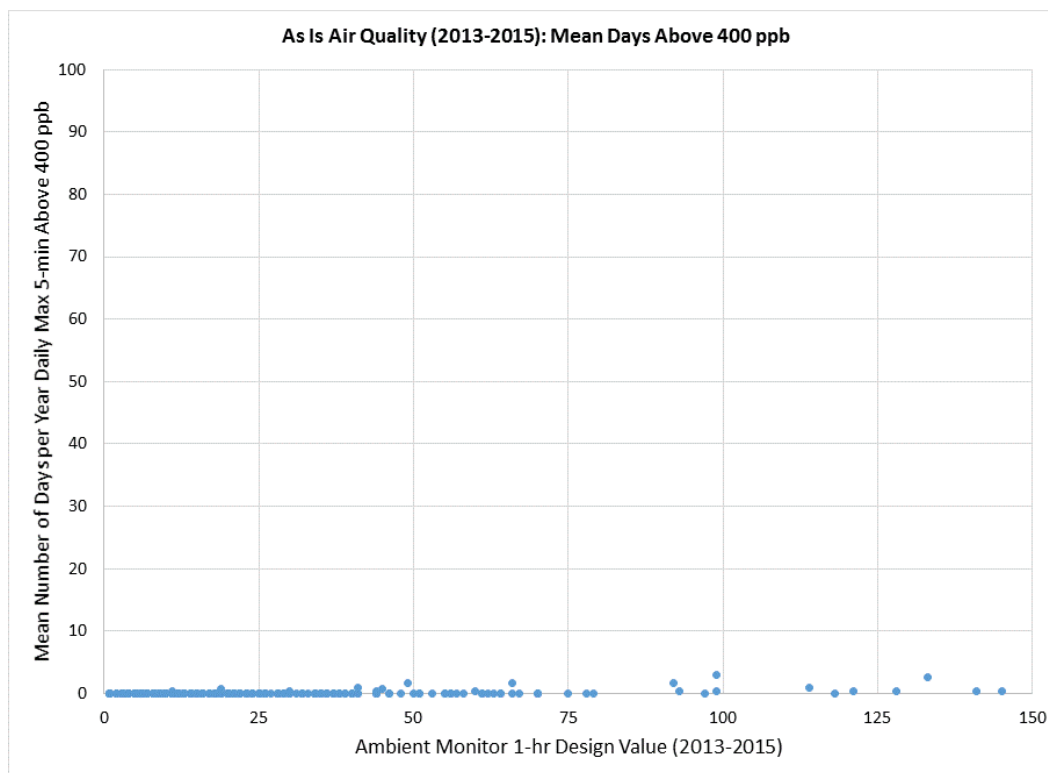
**Figure C-2. As is (unadjusted) SO<sub>2</sub> monitoring data (2013-2015).** Mean number of days/year (top panel) and maximum number of days/year (bottom panel) with daily maximum 5-minute concentrations of SO<sub>2</sub> above 200 ppb.



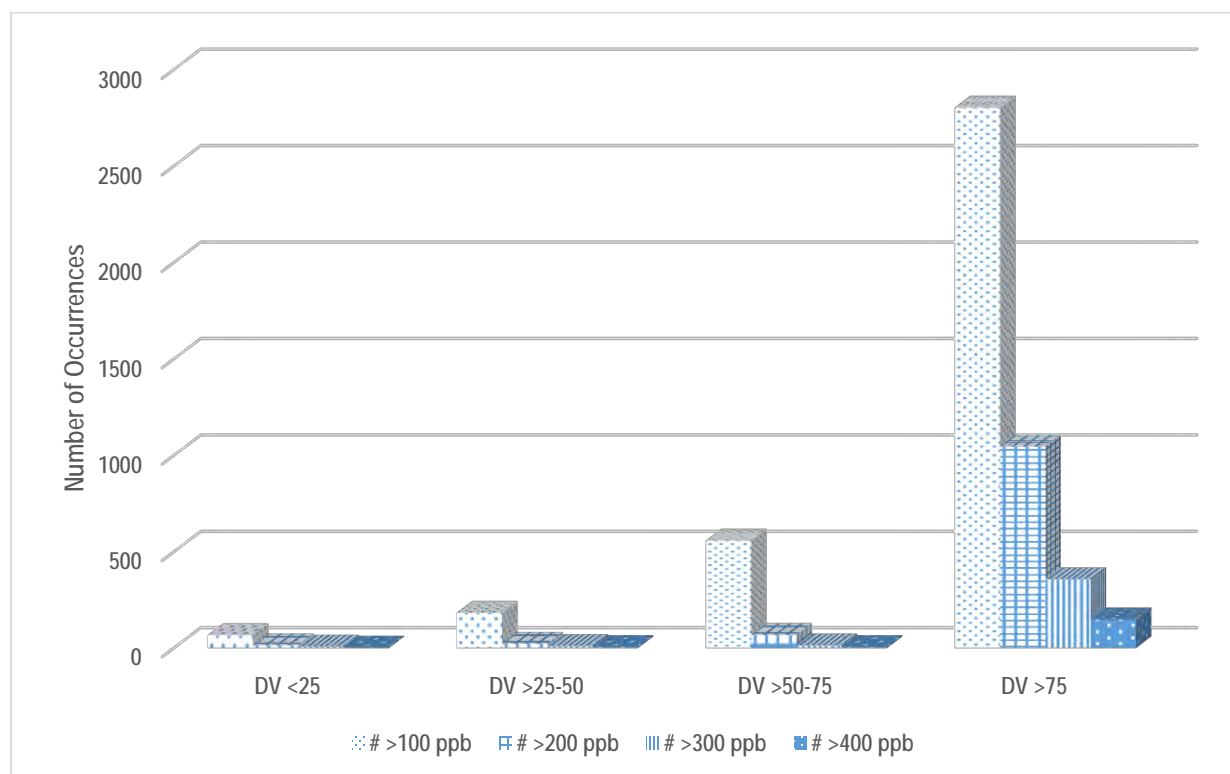
**Figure C-3. As is (unadjusted) SO<sub>2</sub> monitoring data (2013-2015).** Mean number of days/year (top panel) and maximum number of days/year (bottom panel) with daily maximum 5-minute concentrations of SO<sub>2</sub> above 300 ppb.



**Figure C-4. As is (unadjusted) SO<sub>2</sub> monitoring data (2013-2015).** Mean number of days/year (top panel) and maximum number of days/year (bottom panel) with daily maximum 5-minute concentrations of SO<sub>2</sub> above 400 ppb.

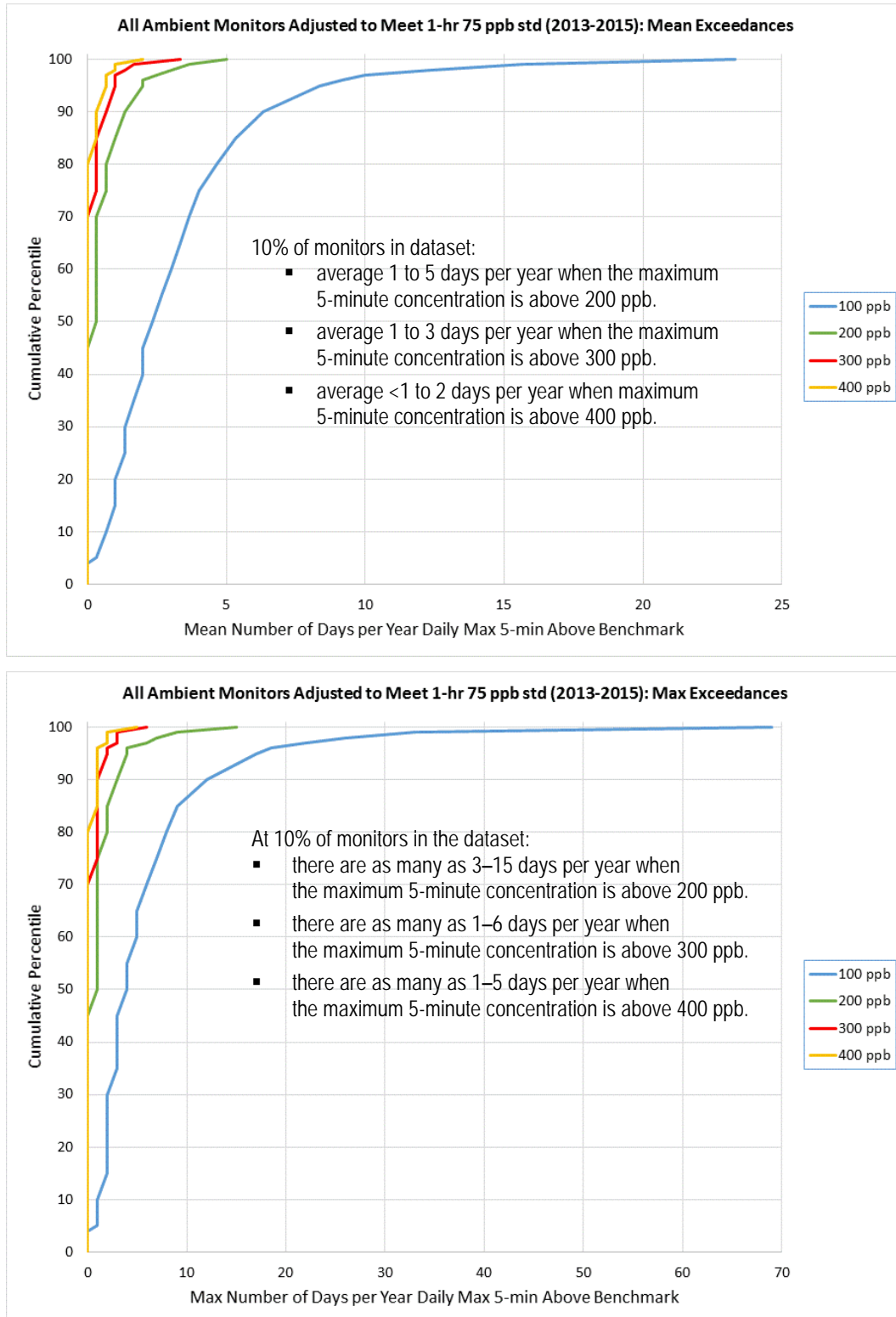


1 **Figure C-5. Monitoring data (2013-2015), unadjusted.** Total number of days across 3-year  
 2 period with daily maximum 5-minute concentrations of SO<sub>2</sub> above 100, 200, 300 and 400 ppb  
 3 across monitors grouped by design value.



4

**Figure C-6. Monitoring data (2013-2015) adjusted to just meet the current standard (75 ppb as a 3-year average of annual 99<sup>th</sup> percentile 1-hour daily maximum concentrations).** Mean number of days/year (top panel) and maximum number of days/year (bottom panel) with daily maximum 5-minute concentrations of SO<sub>2</sub> above 100, 200, 300 and 400 ppb.



## **APPENDIX D**

### **AIR QUALITY INFORMATION FOR GEOGRAPHICAL AREAS OF THREE SELECTED U.S. EPIDEMIOLOGICAL STUDIES**

**Table D-1. Air quality information for geographical areas of the three U.S. epidemiological studies for which the SO<sub>2</sub> effect estimates for hospital admissions or emergency department visits (for asthma or other respiratory disease) and areawide 24-hour average SO<sub>2</sub> concentrations remained positive and statistically significant in copollutant models with particulate matter.**

Study Information								Ambient Air Quality <sup>A</sup>			
Study Area	Study Time Period	Study Reference	SO <sub>2</sub> Concentration Metric Associated with Health Outcome	Assignment of Monitors to Study Subjects for Study Analyses	Study-reported SO <sub>2</sub> Concentrations, <sup>B</sup> 24-hour average (ppm)		99 <sup>th</sup> percentile of daily maximum 1-hour concentrations across study period at highest monitor in study dataset (ppb)	Annual 99 <sup>th</sup> percentile of daily maximum 1-hour concentrations at monitor yielding highest design value (ppb)		Design Value for Current NAAQS (3-year average of annual 99 <sup>th</sup> percentile daily maximum 1-hour concentrations), ppm (monitor ID)	
					Mean	Upper Percentiles					
Bronx County, NY	Jan 1999-Dec 2000	ATSDR 2006 <sup>C</sup>	24-hr ave	2 monitors collecting data in series	12	-	78 <sup>D</sup>	1999	-	E	
								2000			
New York City, NY	Jan 1999-Dec 2002	Ito et al 2007	24-hr ave	Average across all (19) monitors	7.8	75 <sup>th</sup> =10 95 <sup>th</sup> =17	82 <sup>F</sup>	1999	78	1999-2001	73 (36-061-0056)
								2000	71		
								2001	71	2000-2002	69 (36-061-0056)
								2002	65		
New Haven, CT	Jan 1988-Dec 1990	Schwartz, 1995	24-hr ave	Average across all (6) monitors	29.8	75 <sup>th</sup> =38.2 90 <sup>th</sup> =60.7	150 <sup>G</sup>	1988	159	1988-1990	147 (09-009-1123)
								1989	167		
								1990	116		
<p>A Air quality information provided here is drawn from monitors reporting to AQS, as documented in Appendix E). Design values are SO<sub>2</sub> concentrations for the study area in the statistical form of the standard, derived in accordance with 40 CFR, Part 50, Appendix T. Presented is the highest valid design value at a monitor reporting to AQS for specified 3-year period.</p> <p>B Ambient SO<sub>2</sub> concentrations in terms of study metric that are reported in the second draft ISA Table 5-9 (for ATSDR, 2006 and Ito et al., 2007) and Table 5-14 (for Schwartz, 1995). Where multiple monitors contribute data, these are the arithmetic mean and percentiles of the dataset of daily multi-monitor average concentrations for the full study period.</p> <p>C This study was cited as NY DOH, 2006 in the 2008 ISA.</p> <p>D This statistic is for combined dataset of 2 monitoring sites due to construction at the initial site (Thompson and Stewart, 2009). Data are from the first monitor (36-005-0073) for the period Jan 1 to July 14, 1999. Data are from the second monitor (36-005-0110), approximately ½ mile northeast of first, for the period Sept 2, 1999 to Nov 22, 2000.</p> <p>E Due to incomplete quarters or years, there is not a valid design value for a monitor in the Bronx any of the 3-year periods that include the study period.</p> <p>F This statistic is based on monitor 36-061-0080 (Thompson and Stewart, 2009), for which five quarters of data are available during the study period (from 1999 through first quarter of 2000).</p> <p>G This statistic is based on monitor 09-009-1123 (Thompson and Stewart, 2009), for which 12 quarters of data are available during the study period (1988 through 1990).</p>											

## REFERENCES

- ATSDR (Agency for Toxic Substances and Disease Registry). (2006). A study of ambient air contaminants and asthma in New York City: Part A and B. Atlanta, GA: U.S. Department of Health and Human Services.  
[http://permanent.access.gpo.gov/lps88357/ASTHMA\\_BRONX\\_FINAL\\_REPORT.pdf](http://permanent.access.gpo.gov/lps88357/ASTHMA_BRONX_FINAL_REPORT.pdf)
- Ito, K; Thurston, GD; Silverman, RA. (2007). Characterization of PM<sub>2.5</sub>, gaseous pollutants, and meteorological interactions in the context of time-series health effects models. *J Expo Sci Environ Epidemiol* 17: S45-S60. <http://dx.doi.org/10.1038/sj.jes.7500627>
- Schwartz, J; Morris, R. (1995). Air pollution and hospital admissions for cardiovascular disease in Detroit, Michigan. *Am J Epidemiol* 142: 23-35.
- Thompson, R; Stewart, M. (2009). *Air Quality Statistics for Cities Referenced in Key U.S. and Canadian Epidemiological Studies of Hospital Admissions and Emergency Department Visits for All Respiratory Causes and Asthma*. OAQPS Staff Memorandum to Ozone NAAQS Review, March 25. Washington, DC: Office of Air Radiation. (EPA docket number EPA-HQ-OAR-2007-0352). Available at:  
[https://www3.epa.gov/ttn/naaqs/standards/so2/data/2009\\_03\\_Thompson\\_AirQuality.pdf](https://www3.epa.gov/ttn/naaqs/standards/so2/data/2009_03_Thompson_AirQuality.pdf)

## **APPENDIX E**

### **DERIVATION OF DESIGN VALUES PRESENTED IN APPENDIX D**

User ID: DST

## DESIGN VALUE REPORT

Report Request ID: 1565153

Report Code: AMP480

Jun. 20, 2017

## GEOGRAPHIC SELECTIONS

Tribal Code	State	County	Site	Parameter	POC	City	AQCR	UAR	CBSA	CSA	EPA Region
	09	009									

## PROTOCOL SELECTIONS

Parameter Classification	Parameter	Method	Duration
DESIGN VALUE	42401		

## SELECTED OPTIONS

Option Type	Option Value
SINGLE EVENT PROCESSING	EXCLUDE REGIONALLY CONCURRED EVENTS
WORKFILE DELIMITER	,
USER SITE METADATA	STREET ADDRESS
MERGE PDF FILES	YES
QUARTERLY DATA IN WORKFILE	NO
AGENCY ROLE	PQAO

## DATE CRITERIA

Start Date	End Date
1990	1990

## APPLICABLE STANDARDS

Standard Description
SO2 1-hour 2010

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY  
AIR QUALITY SYSTEM  
PRELIMINARY DESIGN VALUE REPORT

Report Date: Jun. 20, 2017

- Notes:**
1. Computed design values are a snapshot of the data at the time the report was run (may not be all data for year).
  2. Some PM2.5 24-hour DVs for incomplete data that are marked invalid here may be marked valid in the Official report due to additional analysis.
  3. Annual Values not meeting completeness criteria are marked with an asterisk ('\*').

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY  
AIR QUALITY SYSTEM  
PRELIMINARY DESIGN VALUE REPORT

Report Date: Jun. 20, 2017

**Pollutant:** Sulfur dioxide(42401)

**Standard Units:** Parts per billion(008)

**NAAQS Standard:** SO2 1-hour 2010

**Statistic:** Annual 99th Percentile

**Design Value Year:** 1990

**REPORT EXCLUDES MEASUREMENTS WITH REGIONALLY CONCURRED EVENT FLAGS.**

**State Name:** Connecticut

**Level:** 75

Site ID	STREET ADDRESS	1990				1989				1988				3-Year	
		Comp.	99th	Cert&		Comp.	99th	Cert&		Comp.	99th	Cert&		Design	Valid
		Qtrrs	Percentile	Eval		Qtrrs	Percentile	Eval		Qtrrs	Percentile	Eval		Value	Ind.
09-009-0010	EGAN CENTER, MATHEW ST	3	114 *	Y		4	113			3	118 *			115	N
09-009-0017	LOMBARD STREET					3	112 *			4	113			113	N
09-009-1003	ANIMAL SHELTER, COMMERCE ST	4	68	Y		4	99			4	95			87	Y
09-009-1123	715 STATE STREET	4	116	Y		4	167			4	159			147	Y
09-009-2123	Bank St at Meadow St (see c	4	83	Y		4	97			4	85			88	Y
09-009-3008	LYDIA STREET EXTENTION	3	93 *	Y		4	110			4	100			101	Y

**Notes:** 1. Computed design values are a snapshot of the data at the time the report was run (may not be all data for year).

2. Some PM2.5 24-hour DVs for incomplete data that are marked invalid here may be marked valid in the Official report due to additional analysis.

3. Annual Values not meeting completeness criteria are marked with an asterisk ('\*').

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY  
AIR QUALITY SYSTEM  
PRELIMINARY DESIGN VALUE REPORT

Report Date: Jun. 20, 2017

CERTIFICATION EVALUATION AND CONCURRENCE FLAG MEANINGS

FLAG	MEANING
M	The monitoring organization has revised data from this monitor since the most recent certification letter received from the state.
N	The certifying agency has submitted the certification letter and required summary reports, but the certifying agency and/or EPA has determined that issues regarding the quality of the ambient concentration data cannot be resolved due to data completeness, the lack of performed quality assurance checks or the results of uncertainty statistics shown in the AMP255 report or the certification and quality assurance report.
S	The certifying agency has submitted the certification letter and required summary reports. A value of "S" conveys no Regional assessment regarding data quality per se. This flag will remain until the Region provides an "N" or "Y" concurrence flag.
U	Uncertified. The certifying agency did not submit a required certification letter and summary reports for this monitor even though the due date has passed, or the state's certification letter specifically did not apply the certification to this monitor.
X	Certification is not required by 40 CFR 58.15 and no conditions apply to be the basis for assigning another flag value
Y	The certifying agency has submitted a certification letter, and EPA has no unresolved reservations about data quality (after reviewing the letter, the attached summary reports, the amount of quality assurance data submitted to AQS, the quality statistics, and the highest reported concentrations).

- Notes:**
- 1. Computed design values are a snapshot of the data at the time the report was run (may not be all data for year).
  - 2. Some PM2.5 24-hour DVs for incomplete data that are marked invalid here may be marked valid in the Official report due to additional analysis.
  - 3. Annual Values not meeting completeness criteria are marked with an asterisk ('\*').

User ID: DST

## DESIGN VALUE REPORT

Report Request ID: 1565370

Report Code: AMP480

Jun. 21, 2017

## GEOGRAPHIC SELECTIONS

Tribal Code	State	County	Site	Parameter	POC	City	AQCR	UAR	CBSA	CSA	EPA Region
	36	005									
	36	047									
	36	061									
	36	081									
	36	085									

## PROTOCOL SELECTIONS

Parameter Classification	Parameter	Method	Duration
DESIGN VALUE	42401		

## SELECTED OPTIONS

Option Type	Option Value
SINGLE EVENT PROCESSING	EXCLUDE REGIONALLY CONCURRED EVENTS
WORKFILE DELIMITER	,
USER SITE METADATA	STREET ADDRESS
MERGE PDF FILES	YES
QUARTERLY DATA IN WORKFILE	NO
AGENCY ROLE	PQAO

## DATE CRITERIA

Start Date	End Date
2000	2002

## APPLICABLE STANDARDS

Standard Description
SO2 1-hour 2010

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY  
AIR QUALITY SYSTEM  
PRELIMINARY DESIGN VALUE REPORT

Report Date: Jun. 21, 2017

- Notes:**
1. Computed design values are a snapshot of the data at the time the report was run (may not be all data for year).
  2. Some PM2.5 24-hour DVs for incomplete data that are marked invalid here may be marked valid in the Official report due to additional analysis.
  3. Annual Values not meeting completeness criteria are marked with an asterisk ('\*').

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY  
AIR QUALITY SYSTEM  
PRELIMINARY DESIGN VALUE REPORT

Report Date: Jun. 21, 2017

**Pollutant:** Sulfur dioxide(42401)

**Design Value Year:** 2000

**Standard Units:** Parts per billion(008)

**REPORT EXCLUDES MEASUREMENTS WITH REGIONALLY CONCURRED EVENT FLAGS.**

**NAAQS Standard:** SO2 1-hour 2010

**Statistic:** Annual 99th Percentile

**Level:** 75

**State Name:** New York

Site ID	STREET ADDRESS	2000			1999			1998			3-Year	
		Comp. Qtrrs	99th Percentile	Cert& Eval	Comp. Qtrrs	99th Percentile	Cert& Eval	Comp. Qtrrs	99th Percentile	Cert& Eval	Design Value	Valid Ind.
36-005-0073	IS 155, 470 JACKSON AV.				2	68 *	Y	4	70	Y	69	N
36-005-0080	MORRISANIA CENTER, 1225-57	1	94 *		4	77	Y	4	69	Y	80	N
36-005-0083	200TH STREET AND SOUTHERN B	2	62 *								62	N
36-005-0110	IS 52 681 KELLY ST	4	86		1	98 *	Y				92	N
36-047-0011	301 GREENPOINT AVENUE				3	51 *	Y	4	42	Y	47	N
36-047-0076	PS 321 180 7TH AV,	0	36 *		4	54	Y	3	59 *	Y	50	N
36-061-0010	MABEL DEAN HIGH SCH.ANNEX,	3	72 *		4	79	Y	3	64 *	Y	72	N
36-061-0056	PS 59, 228 E. 57TH STREET,	4	71		4	78	Y	4	69	Y	73	Y
36-081-0097	56TH AVE AT SPRINGFIELD BLV	4	50		4	53	Y	2	52 *	Y	52	N
36-085-0067	SUSAN WAGNER HS, 1200 MAN	1	54 *		4	46	Y	4	46	Y	49	N

- Notes:**
1. Computed design values are a snapshot of the data at the time the report was run (may not be all data for year).
  2. Some PM2.5 24-hour DVs for incomplete data that are marked invalid here may be marked valid in the Official report due to additional analysis.
  3. Annual Values not meeting completeness criteria are marked with an asterisk ('\*').

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY  
AIR QUALITY SYSTEM  
PRELIMINARY DESIGN VALUE REPORT

Report Date: Jun. 21, 2017

**Pollutant:** Sulfur dioxide(42401)

**Design Value Year:** 2001

**Standard Units:** Parts per billion(008)

**REPORT EXCLUDES MEASUREMENTS WITH REGIONALLY CONCURRED EVENT FLAGS.**

**NAAQS Standard:** SO2 1-hour 2010

**Statistic:** Annual 99th Percentile

**Level:** 75

**State Name:** New York

Site ID	STREET ADDRESS	2001			2000			1999			3-Year	
		Comp. Qtrtrs	99th Percentile	Cert& Eval	Comp. Qtrtrs	99th Percentile	Cert& Eval	Comp. Qtrtrs	99th Percentile	Cert& Eval	Design Value	Valid Ind.
36-005-0073	IS 155, 470 JACKSON AV.							2	68 *	Y	68	N
36-005-0080	MORRISANIA CENTER, 1225-57				1	94 *		4	77	Y	86	N
36-005-0083	200TH STREET AND SOUTHERN B	4	71	Y	2	62 *					67	N
36-005-0110	IS 52 681 KELLY ST	3	81 *	Y	4	86		1	98 *	Y	88	N
36-047-0011	301 GREENPOINT AVENUE							3	51 *	Y	51	N
36-047-0076	PS 321 180 7TH AV,				0	36 *		4	54	Y	45	N
36-061-0010	MABEL DEAN HIGH SCH.ANNEX,	2	69 *	Y	3	72 *		4	79	Y	73	N
36-061-0056	PS 59, 228 E. 57TH STREET,	4	71	Y	4	71		4	78	Y	73	Y
36-081-0097	56TH AVE AT SPRINGFIELD BLV	4	50	Y	4	50		4	53	Y	51	Y
36-081-0124	Queens College 65-30 Kiss	1	57 *	Y							57	N
36-085-0067	SUSAN WAGNER HS, 1200 MAN				1	54 *		4	46	Y	50	N

- Notes:**
1. Computed design values are a snapshot of the data at the time the report was run (may not be all data for year).
  2. Some PM2.5 24-hour DVs for incomplete data that are marked invalid here may be marked valid in the Official report due to additional analysis.
  3. Annual Values not meeting completeness criteria are marked with an asterisk ('\*').

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY  
AIR QUALITY SYSTEM  
PRELIMINARY DESIGN VALUE REPORT

Report Date: Jun. 21, 2017

**Pollutant:** Sulfur dioxide(42401)  
**Standard Units:** Parts per billion(008)  
**NAAQS Standard:** SO2 1-hour 2010  
**Statistic:** Annual 99th Percentile

**Design Value Year:** 2002  
**REPORT EXCLUDES MEASUREMENTS WITH REGIONALLY CONCURRED EVENT FLAGS.**  
**Level:** 75  
**State Name:** New York

Site ID	STREET ADDRESS	2002			2001			2000			3-Year	
		Comp. Qtrtrs	99th Percentile	Cert& Eval	Comp. Qtrtrs	99th Percentile	Cert& Eval	Comp. Qtrtrs	99th Percentile	Cert& Eval	Design Value	Valid Ind.
36-005-0080	MORRISANIA CENTER, 1225-57							1	94 *		94	N
36-005-0083	200TH STREET AND SOUTHERN B	4	62	Y	4	71	Y	2	62 *		65	N
36-005-0110	IS 52 681 KELLY ST	4	67	Y	3	81 *	Y	4	86		78	N
36-047-0076	PS 321 180 7TH AV,							0	36 *		36	N
36-061-0010	MABEL DEAN HIGH SCH.ANNEX,				2	69 *	Y	3	72 *		71	N
36-061-0056	PS 59, 228 E. 57TH STREET,	4	65	Y	4	71	Y	4	71		69	Y
36-081-0097	56TH AVE AT SPRINGFIELD BLV				4	50	Y	4	50		50	N
36-081-0124	Queens College 65-30 Kiss	4	57	Y	1	57 *	Y				57	N
36-085-0067	SUSAN WAGNER HS, 1200 MAN							1	54 *		54	N

**Notes:** 1. Computed design values are a snapshot of the data at the time the report was run (may not be all data for year).  
2. Some PM2.5 24-hour DVs for incomplete data that are marked invalid here may be marked valid in the Official report due to additional analysis.  
3. Annual Values not meeting completeness criteria are marked with an asterisk ('\*').

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY  
AIR QUALITY SYSTEM  
PRELIMINARY DESIGN VALUE REPORT

Report Date: Jun. 21, 2017

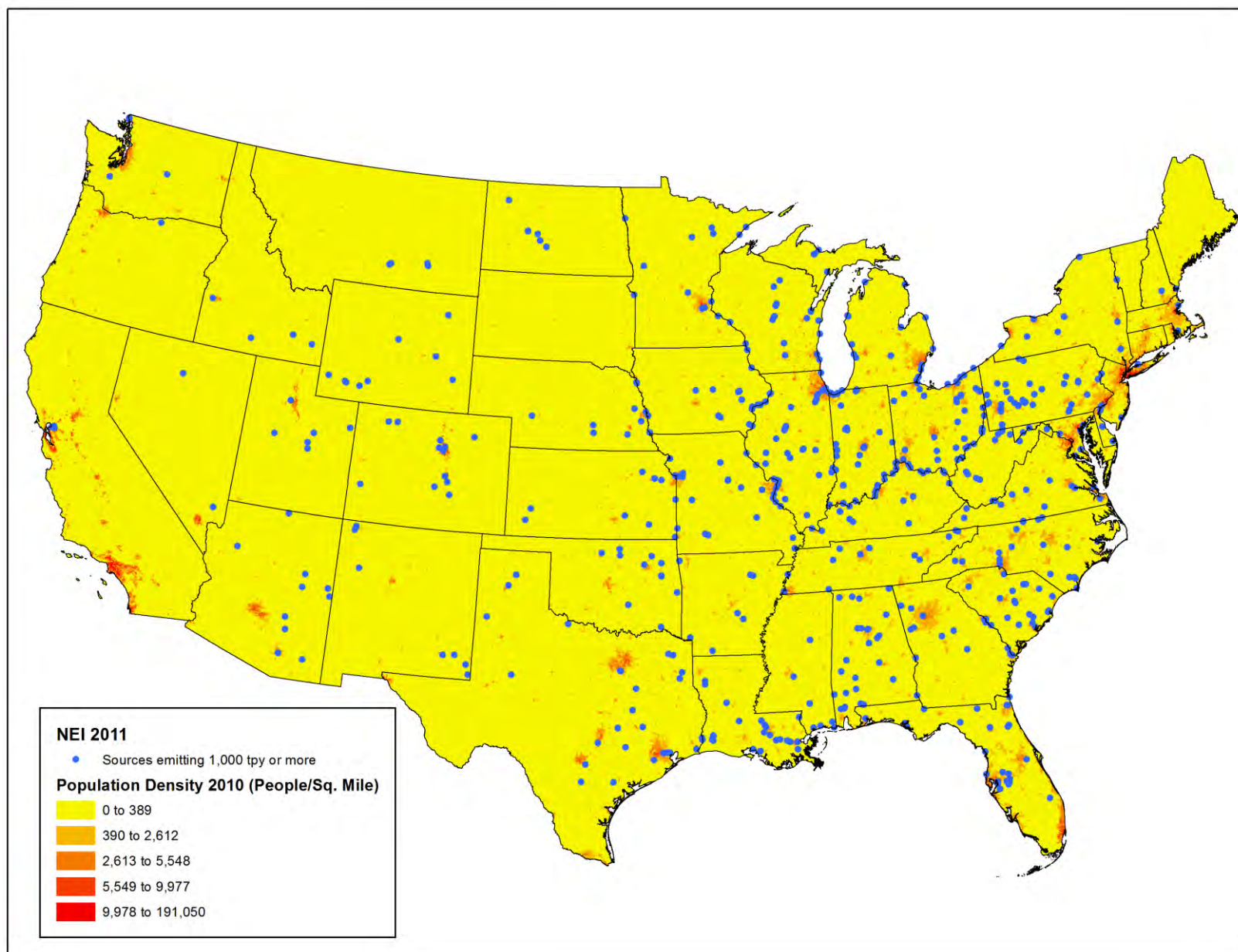
CERTIFICATION EVALUATION AND CONCURRENCE FLAG MEANINGS

FLAG	MEANING
M	The monitoring organization has revised data from this monitor since the most recent certification letter received from the state.
N	The certifying agency has submitted the certification letter and required summary reports, but the certifying agency and/or EPA has determined that issues regarding the quality of the ambient concentration data cannot be resolved due to data completeness, the lack of performed quality assurance checks or the results of uncertainty statistics shown in the AMP255 report or the certification and quality assurance report.
S	The certifying agency has submitted the certification letter and required summary reports. A value of "S" conveys no Regional assessment regarding data quality per se. This flag will remain until the Region provides an "N" or "Y" concurrence flag.
U	Uncertified. The certifying agency did not submit a required certification letter and summary reports for this monitor even though the due date has passed, or the state's certification letter specifically did not apply the certification to this monitor.
X	Certification is not required by 40 CFR 58.15 and no conditions apply to be the basis for assigning another flag value
Y	The certifying agency has submitted a certification letter, and EPA has no unresolved reservations about data quality (after reviewing the letter, the attached summary reports, the amount of quality assurance data submitted to AQS, the quality statistics, and the highest reported concentrations).

**Notes:** 1. Computed design values are a snapshot of the data at the time the report was run (may not be all data for year).  
2. Some PM2.5 24-hour DVs for incomplete data that are marked invalid here may be marked valid in the Official report due to additional analysis.  
3. Annual Values not meeting completeness criteria are marked with an asterisk ('\*').

## **APPENDIX F**

### **GEOGRAPHIC DISTRIBUTION OF CONTINENTAL U.S. FACILITIES EMITTING MORE THAN 1,000 TPY SO<sub>2</sub> AND POPULATION DENSITY BASED ON U.S. CENSUS TRACTS**

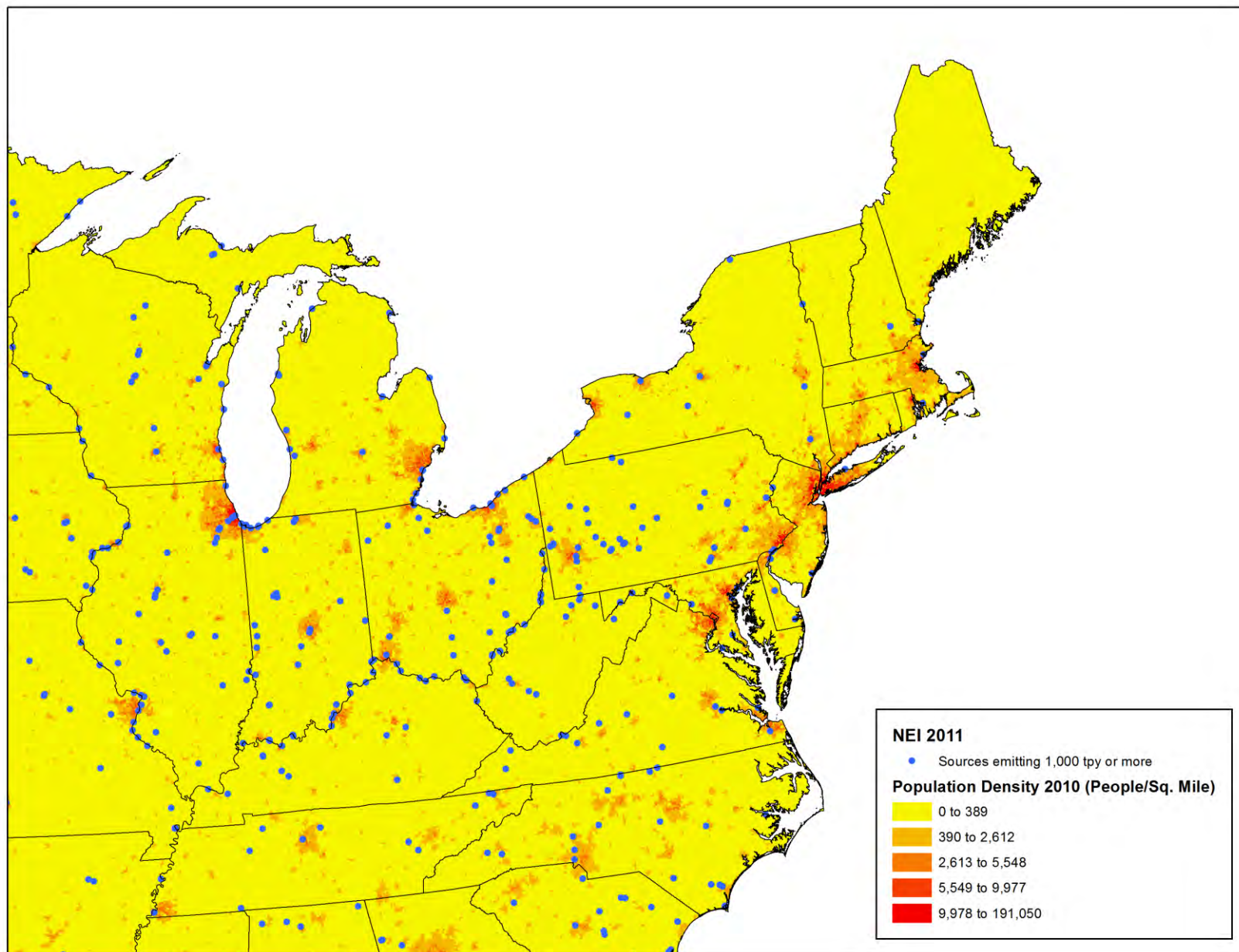


**Figure F-1. Continental U.S.: Facilities emitting more than 1,000 tpy SO<sub>2</sub> (n=619 in 2011 NEI) and population density.**

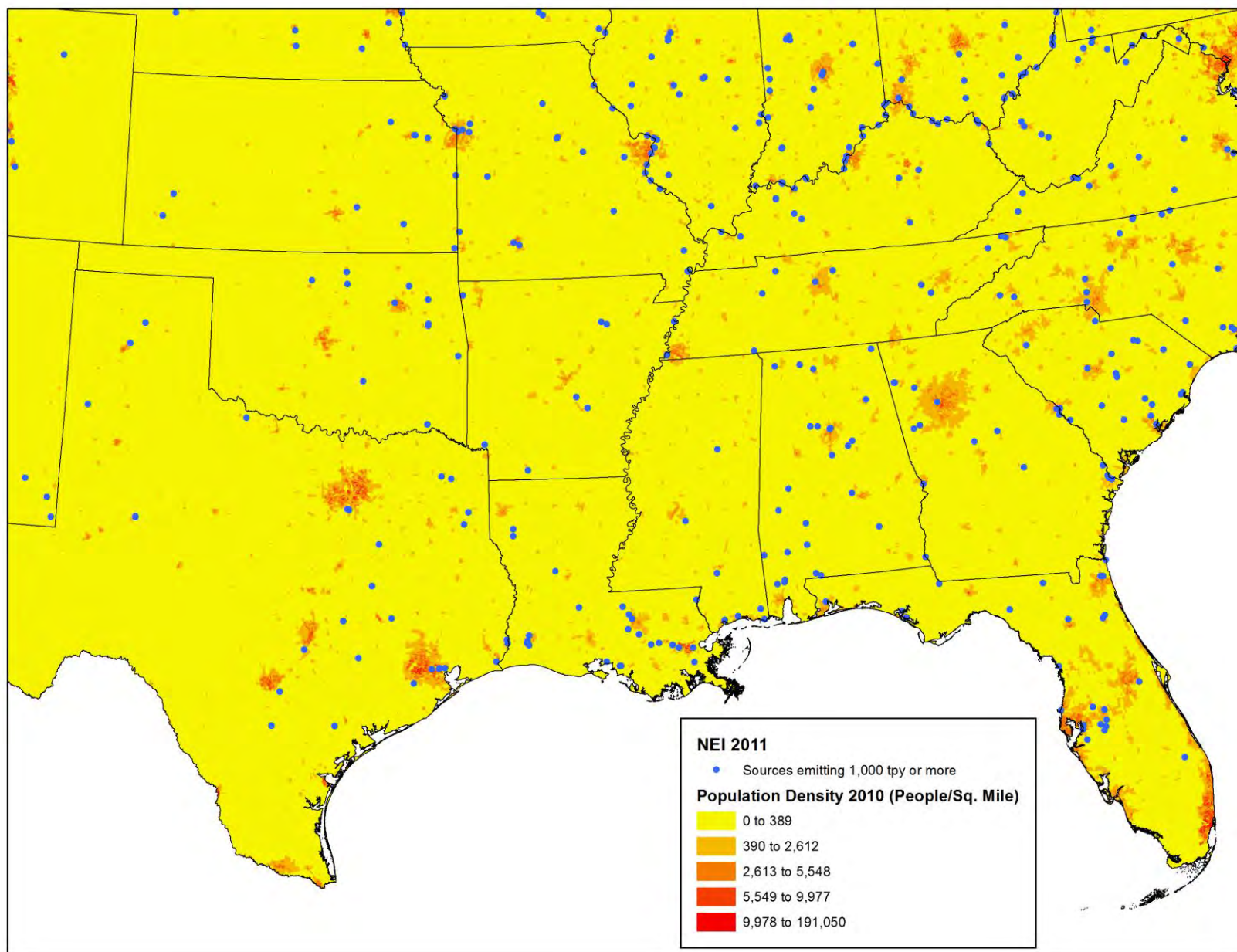
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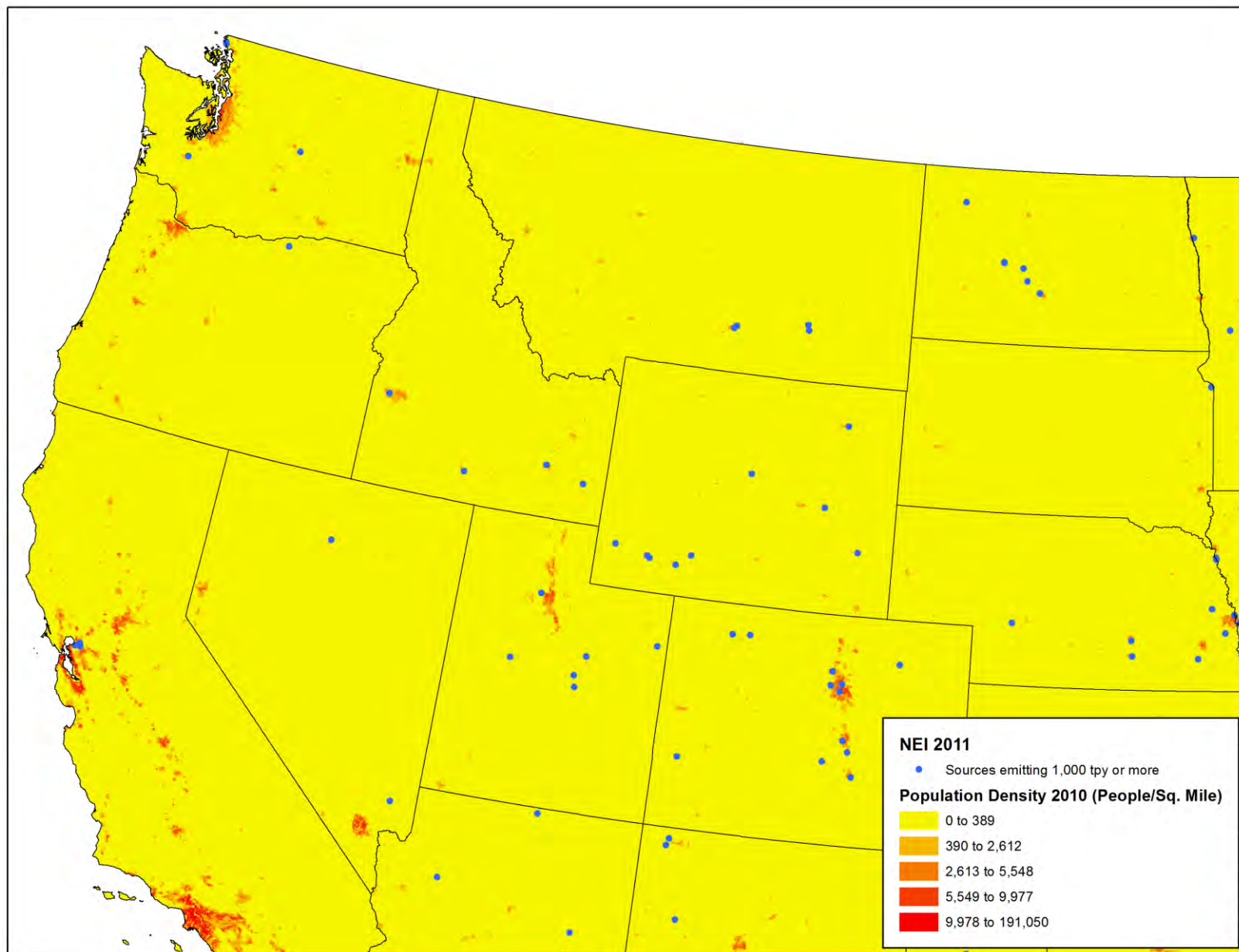
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**Figure F-2. Northeast U.S.: Facilities emitting more than 1,000 tpy SO<sub>2</sub> and population density.**



**Figure F-3. Southeast U.S.: Facilities emitting more than 1,000 tpy SO<sub>2</sub> and population density.**

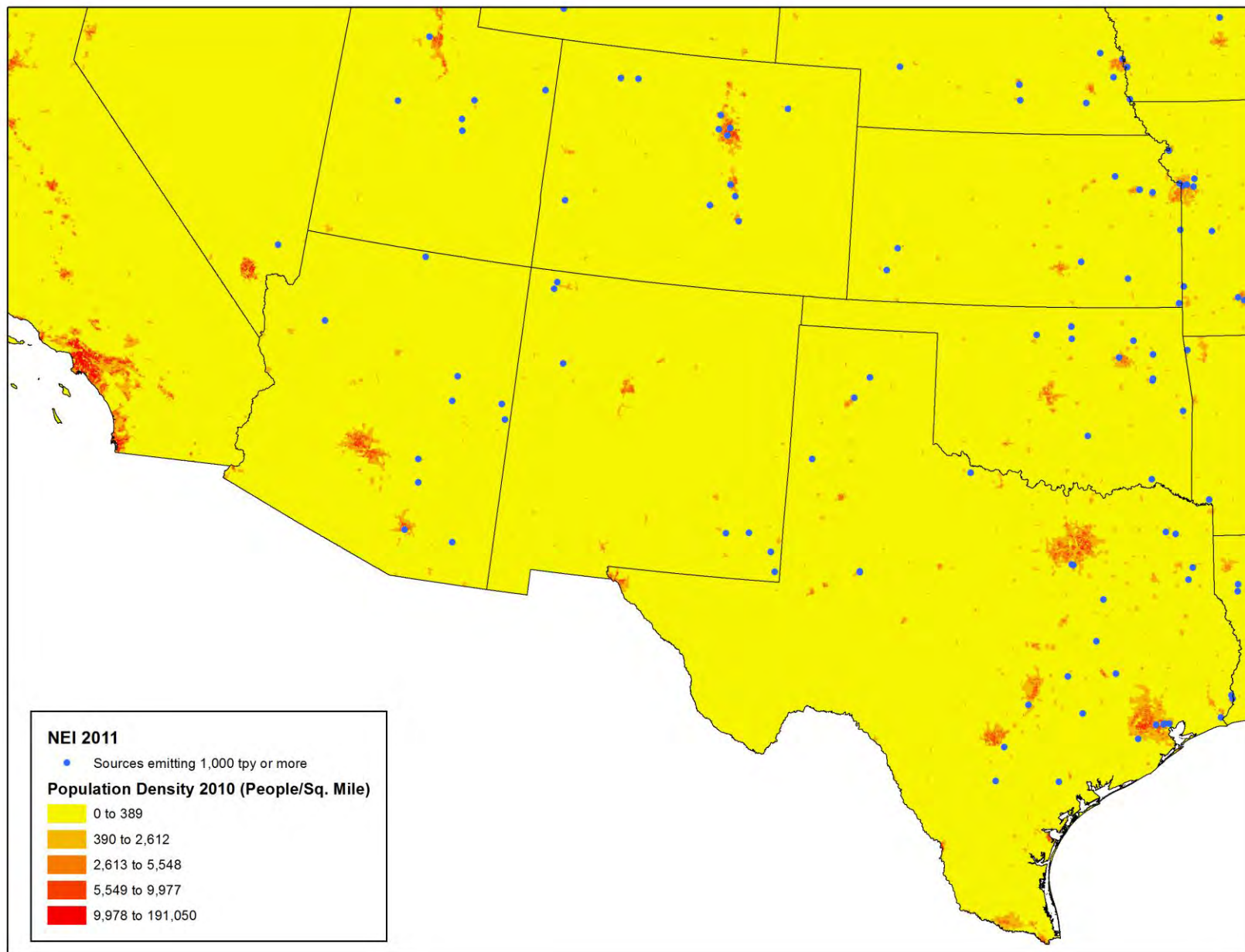


**Figure F-4. Northwest U.S.: Facilities emitting more than 1,000 tpy SO<sub>2</sub> and population density.**

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**Figure F-5. Southwest U.S.: Facilities emitting more than 1,000 tpy SO<sub>2</sub> and population density.**

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