Review of the National Ambient Air Quality Standards for Lead:

Policy Assessment of Scientific and Technical Information

OAQPS Staff Paper

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U.S. Environmental Protection Agency
Office of Air Quality Planning and Standards
Research Triangle Park, North Carolina

DISCLAIMER

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An earlier draft of this document was formally reviewed by the Clean Air Scientific Advisory Committee (CASAC) and made available for public comment. This document has been informed by the expert advice and comments received from CASAC, as well as by public comments submitted by independent scientists, officials from State and local air pollution organizations, environmental groups, and industry groups and companies.

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1 INTRODUCTION

1.1 PURPOSE

This OAQPS Staff Paper, prepared by staff in the U.S. Environmental Protection Agency's (EPA) Office of Air Quality Planning and Standards (OAQPS), presents factors relevant to EPA's current review of the primary (health-based) and secondary (welfare-based) lead (Pb) national ambient air quality standards (NAAQS) that were originally established in 1978. In this document, OAQPS staff evaluates the policy implications of the key studies and scientific information contained in the final document, *Air Quality Criteria for Lead* (USEPA, 2006a; henceforth referred to as the CD), prepared by EPA's National Center for Environmental Assessment, and presents and interprets results from several quantitative analyses (e.g., human exposure analyses, human health risk assessments and environmental assessments) that we believe should also be considered in EPA's review of the Pb NAAQS.[1] Further, this document presents OAQPS staff conclusions and recommendations on a range of policy options that we believe are appropriate for the Administrator to consider concerning whether, and if so how, to revise the primary and secondary Pb NAAQS.

The policy assessment presented in this Staff Paper is intended to help "bridge the gap" between the scientific assessment contained in the CD and the judgments required of the EPA Administrator in determining whether it is appropriate to retain or revise the NAAQS for Pb. In evaluating the adequacy of the current standard and a range of policy alternatives, OAQPS staff has considered the available scientific evidence and quantitative risk-based analyses, together with related limitations and uncertainties, and has focused on the information that is most pertinent to evaluating the basic elements of air quality standards: indicator[2], averaging time, form[3], and level. These elements, which together serve to define each standard, must be considered collectively in evaluating the health and welfare protection afforded by the Pb standards. The information, conclusions, and staff recommendations presented in this Staff Paper have been informed by comments and advice received from an independent scientific review committee, the Clean Air Scientific Advisory Committee (CASAC), in their reviews of an earlier draft of this document and drafts of related technical support documents, as well as comments on these earlier draft documents submitted by public commenters.

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[1] The terms "staff" and "we" throughout this document refer to OAQPS staff.

[2] The "indicator" of a standard defines the chemical species or mixture that is to be measured in determining whether an area attains the standard.

[3] 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.

While this Staff Paper should be of use to all parties interested in the Pb NAAQS review, it is written with an expectation that the reader has some familiarity with the technical discussions contained in the CD. Further, we note that this document, which contains conclusions and recommendations of OAQPS staff, does not necessarily reflect the views of the Agency.

1.2 BACKGROUND

1.2.1 Legislative Requirements

Two sections of the Clean Air Act (Act) govern the establishment and revision of the NAAQS. Section 108 (42 U.S.C. 7408) directs the Administrator to identify and list each air pollutant that "in his judgment, cause or contribute to air pollution which may reasonably be anticipated to endanger public health and welfare" and whose "presence . . . in the ambient air results from numerous or diverse mobile or stationary sources" and to issue air quality criteria for those that are listed. Air quality criteria are 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 ambient air . . .". Section 108 also states that the Administrator "shall, from time to time . . . revise a list" that includes these pollutants, which provides the authority for a pollutant to removed from or added to the list of criteria pollutants.

Section 109 (42 U.S.C. 7409) directs the Administrator to propose and promulgate "primary" and "secondary" NAAQS for pollutants listed under section 108. Section 109(b)(1) defines a primary standard as one "the attainment and maintenance of which in the judgment of the Administrator, based on [air quality] criteria and allowing an adequate margin of safety, are requisite to protect the public health."[4] A secondary standard, as defined in Section 109(b)(2), must "specify a level of air quality the attainment and maintenance of which, in the judgment of the Administrator, based on 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."[5]

The requirement that primary standards include an adequate margin of safety was intended to address uncertainties associated with inconclusive scientific and technical

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[4] 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)

[5] Welfare effects as defined in section 302(h) (42 U.S.C. 7602(h)) 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."

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. *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). Both kinds of uncertainties are components of the risk associated with pollution at levels below those at which human health effects can be said to occur with reasonable scientific certainty. Thus, in selecting primary standards that include an adequate margin of safety, the Administrator is seeking not only to prevent pollution levels that have been demonstrated to be harmful but also to prevent lower pollutant levels that may pose an unacceptable risk of harm, even if the risk is not precisely identified as to nature or degree.

In selecting a margin of safety, EPA considers such factors as the nature and severity of the health effects involved, the size of the sensitive population(s) at risk, and the kind and degree of the uncertainties that must be addressed. The selection of any particular approach to providing an adequate margin of safety is a policy choice left specifically to the Administrator's judgment. *Lead Industries Association v. EPA*, *supra*, 647 F.2d at 1161-62.

In setting standards that are "requisite" to protect public health and welfare, as provided in section 109(b), EPA's task is to establish standards that are neither more nor less stringent than necessary for these purposes. In so doing, EPA may not consider the costs of implementing the standards. See generally *Whitman v. American Trucking Associations*, 531 U.S. 457, 471, 475-76 (2001).

Section 109(d)(1) of the Act requires that "not later than December 31, 1980, and at 5-year intervals thereafter, the Administrator shall complete a thorough review of the criteria published under section 108 and the national ambient air quality standards . . . and shall make such revisions in such criteria and standards and promulgate such new standards as may be appropriate . . . ." Section 109(d)(2) requires that an independent scientific review committee "shall complete a review of the criteria . . . and the national primary and secondary ambient air quality standards . . . and shall recommend to the Administrator any new . . . standards and revisions of existing criteria and standards as may be appropriate . . . ." Since the early 1980's, this independent review function has been performed by the Clean Air Scientific Advisory Committee (CASAC) of EPA's Science Advisory Board.

1.2.2 History of Lead NAAQS Reviews

On October 5, 1978 EPA promulgated primary and secondary NAAQS for lead under section 109 of the Act (43 FR 46246). Both primary and secondary standards were set at a level of 1.5 micrograms per cubic meter ($\mu g/m^3$), measured as Pb in total suspended particulate matter (TSP), not to be exceeded by the maximum arithmetic mean concentration averaged over a

calendar quarter. This standard was based on the 1977 *Air Quality Criteria for Lead* (USEPA, 1977).

A review of the Pb standards was initiated in the mid-1980s. The scientific assessment for that review is described in the 1986 *Air Quality Criteria for Lead* (USEPA, 1986a), the associated Addendum (USEPA, 1986b) and the 1990 Supplement (USEPA, 1990a). As part of the review, the Agency designed and performed human exposure and health risk analyses (USEPA, 1989), the results of which were presented in a 1990 Staff Paper (USEPA, 1990b). Based on the scientific assessment and the human exposure and health risk analyses, the 1990 Staff Paper presented options for the Pb NAAQS level in the range of 0.5 to 1.5 $\mu g/m^3$, and suggested the second highest monthly average in three years for the form and averaging time of the standard (USEPA, 1990b). After consideration of the documents developed during the review and the significantly changed circumstances since Pb was listed in 1976, as noted above, the Agency did not propose any revisions to the 1978 Pb NAAQS. In a parallel effort, the Agency developed the broad, multi-program, multimedia, integrated *U.S. Strategy for Reducing Lead Exposure* (USEPA, 1991). As part of implementing this strategy, the Agency focused efforts primarily on regulatory and remedial clean-up actions aimed at reducing Pb exposures from a variety of nonair sources judged to pose more extensive public health risks to U.S. populations, as well as on actions to reduce Pb emissions to air.

1.2.3 Current Lead NAAQS Review

EPA initiated the current review of the air quality criteria for Pb on November 9, 2004 with a general call for information (69 FR 64926). A project work plan (USEPA, 2005a) for the preparation of the CD was released in January 2005 for CASAC and public review. EPA held a series of workshops in August 2005, with invited recognized scientific experts to discuss initial draft materials that dealt with various lead-related issues being addressed in the Pb air quality criteria document. These workshops helped to inform the preparation of the first draft CD (USEPA, 2005b), which was released for CASAC and public review in December 2005 and discussed at a CASAC meeting held on February 28-March 1, 2006.

A second draft CD (USEPA, 2006b) was released for CASAC and public review in May 2006, and discussed at the CASAC meeting on June 28, 2006. A subsequent draft of *Chapter 7 - Integrative Synthesis* (Chapter 8 in the final CD), released on July 31, 2006, was discussed at an August 15, 2006 CASAC teleconference. The final CD was released on September 30, 2006 (USEPA, 2006a). While the CD focuses on new scientific information available since the last review, it appropriately integrates that information with scientific criteria from previous reviews.

In February 2006, EPA released the *Plan for Review of the National Ambient Air Quality Standards for Lead* (USEPA 2006c) that described Agency plans and a timeline for reviewing

the air quality criteria, developing human exposure and risk assessments and an ecological risk assessment, preparing a policy assessment, and developing the proposed and final rulemakings.

In May 2006, EPA released for CASAC and public review a draft *Analysis Plan for Human Health and Ecological Risk Assessment for the Review of the Lead National Ambient Air Quality Standards* (USEPA, 2006d), which was discussed at a June 29, 2006 CASAC meeting. CASAC panel members' views were received at and subsequent to the meeting (Henderson, 2006), and considered in the implementation of the human health and ecological risk assessments. The May 2006 assessment plan discussed two assessment phases: a pilot phase and a full-scale phase. The pilot phase of both the human health and ecological risk assessments was presented in the draft *Lead Human Exposure and Health Risk Assessments and Ecological Risk Assessment for Selected Areas* (ICF, 2006; henceforth referred to as the pilot phase or first draft Risk Assessment Report) which was released for CASAC and public review in December 2006. The first draft Staff Paper, also released in December 2006, discussed the pilot assessments and the most policy-relevant science from the CD. These documents were reviewed by CASAC and the public at a public meeting on February 6-7, 2007.

Taking into consideration comments on the first draft Risk Assessment Report and the first draft Staff Paper from CASAC (Henderson, 2007a) and the public, staff conducted full-scale human exposure and health risk assessments, although no further work was done on the environmental assessment due to resource limitations. The full-scale human exposure and health risk assessments were presented in a second draft Risk Assessment Report (USEPA, 2007a) which was released in July 2007 for review by CASAC at a meeting held on August 28-29, 2007. Taking into consideration CASAC comments (Henderson, 2007b) and public comments on that document, staff conducted additional human exposure and health risk assessments, which are presented in a final Risk Assessment Report (USEPA, 2007b) and discussed in this Staff Paper.

The schedule for completion of this review is governed by a judicial order resolving a lawsuit filed in May 2004, alleging that EPA had failed to complete the current review within the period provided by statute. *Missouri Coalition for the Environment, v. EPA* (No. 4:04CV00660 ERW, Sept. 14, 2005). The order that now governs this review, entered by the court on September 14, 2005, provides that EPA will finalize the Staff Paper no later than November 1, 2007. The order also specifies that EPA sign, for publication, notices of proposed and final rulemaking concerning its review of the Pb NAAQS no later than May 1, 2008 and September 1, 2008, respectively. Since this review of the lead NAAQS was initiated, EPA has adopted a new process for reviewing NAAQS that eliminates issuance of a Staff Paper and adds publication of an advance notice of proposed rulemaking (ANPR). In applying this new process to this review of the lead NAAQS, in addition to the issuance of this Staff Paper consistent with the judicial

order, EPA also plans to publish an ANPR around the end of November 2007. To ensure that the ordered final rulemaking deadline will be met, EPA has set an interim target date for a proposed rulemaking of March 2008.

1.3 GENERAL APPROACH AND ORGANIZATION OF THE DOCUMENT

The policy assessment in this Staff Paper document is based on staff's evaluation of the policy implications of the scientific evidence reviewed in the CD and results of quantitative analyses based on that evidence, as well as the views presented by CASAC and various stakeholders. Taken together, this information informs various conclusions and the identification of a range of policy options to address public health and welfare effects associated with exposure to ambient Pb resulting from emissions to the ambient air.

Following this introductory chapter, this Staff Paper is organized into three main parts: the characterization of ambient Pb; Pb-related health effects and primary Pb NAAQS; and Pb-related welfare effects and secondary Pb NAAQS. The content of these parts is discussed more fully below.

The characterization of ambient Pb is presented in Chapter 2 and includes information on Pb properties, current Pb air quality patterns, historic trends, and background levels. In recognition of the multimedia nature of Pb and the distribution into other media of Pb emitted into the air, Chapter 2 also includes information on Pb in media other than air including outdoor dust, soil, surface water and sediment. This chapter provides a frame of reference for exposure and risk analyses and subsequent discussion of the Pb NAAQS and alternative forms of Pb standards.

Chapters 3 through 5 comprise the second main part of this document, dealing with human health and primary standards. Chapter 3 presents an overview of key policy-relevant health effects evidence, major health-related conclusions from the CD, and an examination of issues related to the quantitative assessment of health risks. Chapter 4 describes the scope and methods used in conducting human exposure and health risk assessments and presents key results from those assessments together with a discussion of uncertainty and variability in the results. Chapter 5 includes staff conclusions and policy recommendations on the adequacy of the current primary standard and on an appropriate range of alternative primary standards for the Administrator's consideration, together with a discussion of the science and public health policy judgments underlying such standards.

Chapter 6 comprises the third main part of this document. Chapter 6 presents a policy-relevant assessment of Pb welfare effects evidence and describes the scope and methods used in conducting the environmental risk assessment, as well as results from the pilot environmental assessment. This chapter includes staff conclusions and policy recommendations on the

adequacy of the current secondary standard and on an appropriate range of alternative secondary standards for the Administrator's consideration, together with a discussion of the science and public welfare policy judgments underlying such standards.

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2 CHARACTERIZATION OF AMBIENT LEAD

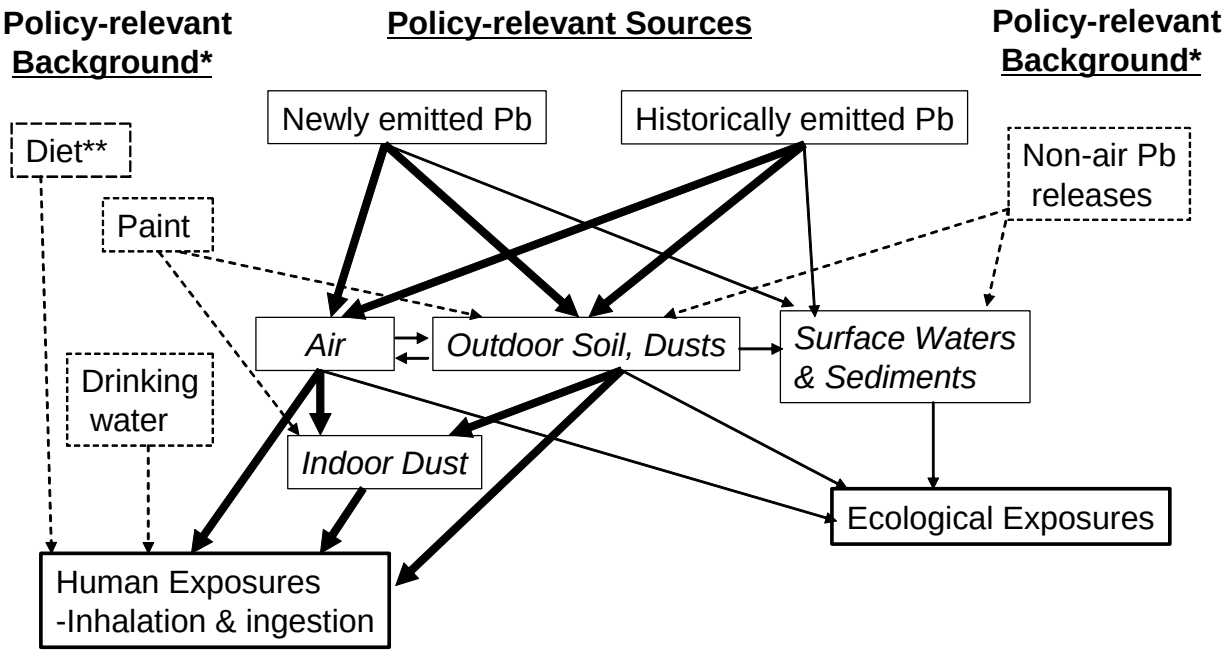
2.1 INTRODUCTION/BACKGROUND

The focus for this Pb NAAQS review is on Pb derived from those sources emitting Pb to ambient air. The multimedia and persistent nature of Pb, however, contributes several complexities to the review.

First, exposures to Pb emitted into the air occur via multiple pathways. As described in the CD, "The multimedia aspects of Pb exposure can be seen in that Pb emissions to the air contribute to Pb concentrations in water, soil and dusts; Pb in soil and dust also can make important contributions to Pb concentrations in ambient air" (CD, p. 3-1).

Inhalation exposures can result from Pb emitted to the ambient air recently or from Pb emitted in the past that has deposited from air to soil or dust and then become resuspended in the ambient air. Further, Pb emitted into the ambient air can contribute to ingestion exposures (associated with indoor dust, outdoor soil/dust, agricultural products and surface water) of recently deposited Pb and of Pb that was deposited in the past. Consequently, exposure to Pb is multipathway, and we are considering both airborne Pb, as it contributes to exposures through direct inhalation of particles containing Pb, and also Pb that has deposited from air to dusts, soil and other environmental media and that contributes to exposures through ingestion. Further, we are considering that Pb, once deposited, may be resuspended in the air, contributing to inhalation exposures or, upon redeposition, to ingestion exposures. Thus, as illustrated in Figure 2-1, pathways that are directly relevant to a review of the NAAQS include both newly emitted Pb from currently operating sources, and Pb emitted in the past, either from currently operating sources or historic sources, which are collectively referred to as "policy-relevant sources".

Due to limited data, models, and time available, however, we are not able to fully and completely characterize all of the various complexities associated with Pb exposure pathways. For example, Figure 1-1 illustrates that people are also exposed to Pb that originates from nonair sources, including leaded paint or drinking water distribution systems (see CD, Sections 3.3 and 3.5). For purposes of this review, the Pb from these nonair sources is collectively referred to as "policy-relevant background". Additionally Pb in diet and drinking water sources may derive from Pb emitted into the ambient air (i.e., policy-relevant sources), however, we have not explicitly described the current contribution from air pathways to these exposure pathways in this chapter; these exposure pathways are described in the CD (Section 3.4).



*Policy-relevant background sources and pathways are indicated by dashed lines.
**Dietary exposure should not be considered to be limited to policy-relevant background, as it reflects a combination of Pb introduced into food items during processing (policy-relevant background), as well as Pb associated with atmospheric deposition (policy-relevant sources).

Figure 2-1. Principal pathways of human and ecological exposure to Pb. Among the policy-relevant pathways, heavy arrows indicate the predominant human exposures.

2.1.1 Properties of Ambient Lead

Due to its physicochemical properties, Pb exists in the environment predominantly in solid form. Consequently upon emission into the air, Pb deposits onto surfaces or exists in the atmosphere as a component of atmospheric aerosol (CD, Section 2.1). The various Pb compounds that are naturally occurring in the environment or are introduced by anthropogenic activities include oxides, chlorides (or other halides), sulfates, and sulfides (see CD, Table 2-5). A more complete discussion of the physical and chemical properties of Pb and Pb compounds is provided in the CD (Section 2.1). The Pb NAAQS pertains to the Pb content of all Pb compounds that may be emitted to air (see Section 2.3.1 for discussion of collection and analysis methods).

The relative presence of Pb among the various environmentally occurring compounds influences its distribution within the environment, and the relative bioavailability of these

compounds has implications for human and ecological exposures and risks (CD, Sections 4.2.1, 8.1.3 and 8.2.3). With regard to human exposures and risk, this is reflected in the exposure modeling described in Chapter 4. Lead speciation and bioavailability are discussed further with regard to environmental effects in Chapter 6.

2.1.2 Fate and Transport of Pb Particles

The atmosphere is the major environmental transport pathway for anthropogenic Pb (CD, p 2-52). Lead can be transported in the atmosphere and undergo secondary dispersal via the deposition and resuspension of particles containing Pb. As described in the CD (Section 2.3.1), airborne Pb particles generally have a bimodal distribution with the greater mass of Pb found in the fine fraction (CD, p. 2-52). Since small particles are much slower to deposit than larger particles, Pb can be transported great distances in the atmosphere. Thus, Pb is widely dispersed, as evidenced by detection of Pb even in the most remote places such as the arctic region (CD, pp. 2-52, 3-3).

Airborne concentrations of species emitted from a point source are frequently described by a Gaussian distribution. Gaussian models are, in general, reasonably accurate for small geographic scales, e.g., within ~50 km of the source (CD, p. 2-53). The rate and direction of dispersion are dependent both on pollutant characteristics and meteorological conditions. Important meteorological factors influencing dispersion include wind speed, surface roughness, inversion frequency, inversion duration, and temperature. Results are site specific. For long range transport modeling, Lagrangian trajectory or Eulerian grid models are commonly employed. These models determine how a parcel of air moves relative to the moving fluid and a fixed coordinate system, respectively. Retrospective air mass trajectories based on hybrid models are also used. Results of a study using such an approach have reported finding airborne Pb in a less industrial country originating from emissions in several distant countries (CD, p. 2-54).

Wet and dry deposition are the ultimate paths by which Pb particles are removed from the atmosphere. Dry deposition is the process by which Pb particles are delivered from the atmosphere onto surfaces in the absence of precipitation. Factors that govern dry deposition are the level of atmospheric turbulence, especially in the layer nearest the ground, particle size distributions and density, and the nature of the surface itself, such as smooth or rough. In the commonly used model formulation for dry deposition, it is assumed that the dry deposition flux is directly proportional to the local concentration of the pollutant species, at some reference height above the surface (e.g., 10 m or less), multiplied by the deposition velocity (CD, p. 2-55). The concentration is computed by the dispersion models mentioned above, depending on local versus regional or global applications. Estimates of dry deposition velocity constitute the

primary output of a large number of dry deposition models that have been developed during the past ten years and most of these rely on so-called “resistance schemes”. The advantage of this deposition velocity representation is that all the complexities of the dry deposition process are bundled in a single parameter, but the disadvantage is that because this parameter addresses a variety of processes, it is difficult to specify properly. A large range of Pb deposition velocities (0.05 to 1.3 cm/s) has been reported (CD, pp. 2-55 to 2-57 and Table 2-21).

Wet deposition, or the delivery of a pollutant to the ground in precipitation, is the process by which airborne pollutants are scavenged by precipitation and removed from the atmosphere. The flux of a depositing species can be defined as the product of the rate of precipitation and the concentration of the chemical species in the precipitation (CD, pp. 2-57 to 2-59). Wet deposition is affected by: 1) nucleation scavenging (removal by direct incorporation into new cloud droplets); 2) in-cloud scavenging (removal by incorporation into existing cloud droplets); and 3) precipitation washout (removal by rain as it is falling to the ground). The size of particles can influence wet deposition rates. Large particles are scavenged by precipitation more efficiently than smaller particles (CD, p. 2-59). Lead, beyond the influence of individual sources, is found primarily in the submicron size range, and consequently does not undergo wet deposition as easily as many of the crustal elements (CD, p. 2-59). Models vary in how they treat wet deposition. Gaussian models focus solely on washout aspects, mainly because this process is dominant within the 50 km limit of model applicability. Regional and global models have more comprehensive treatment of wet deposition. Lead concentrations in precipitation have shown a pronounced downward trend from the 1970s into the 1990s, presumed primarily due to the phase-out of leaded fuel (CD, pp. 2-60 to 2-61 and Table 2-22).

The resuspension of soil-bound Pb particles and contaminated road dust can be a significant source of airborne Pb (CD, Section 2.3.3, and p. 2-62). Studies of emissions in southern California indicate that Pb in resuspended road dust may represent between 40% and 90% of Pb emissions in some areas (CD, p. 2-65). Lead concentrations in suspended soil and dust, however, vary significantly (CD, p. 2-65). In general, the main drivers of particle resuspension are typically mechanical stressors such as vehicular traffic, construction and agricultural operations, and to a lesser extent, the wind. Understanding the physics of resuspension from natural winds requires analyzing the wind stresses on individual particles and although this analysis can be accurate on a small scale, predicting resuspension on a large scale generally focuses on empirical data for soil movement due to three processes: saltation, surface creep, and suspension (CD, pp. 2-62 to 2-63). Further, rather than a continuous process, resuspension may occur as a series of events. Short episodes of high wind speed, dry conditions, and other factors conducive to resuspension may dominate annual averages of upward flux (CD, p. 2-65). These factors complicate emissions estimates.

2.2 SOURCES AND EMISSIONS TO THE ATMOSPHERE

In this section we describe the available information on sources and emissions of Pb into the ambient air. The section does not provide a comprehensive list of all sources of Pb, 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 provide some characterization of their emissions and distribution within the U.S.

The primary data source for this discussion is the National Emissions Inventory (NEI) for 2002 (USEPA, 2007a). As a result of Clean Air Act requirements, emissions standards implemented for a number of source categories since then are projected to result in considerably lower emissions at the current time or in the near future.

It is noted that the Pb emissions estimates in the NEI, and presented in this chapter, are a mixture of estimates specific to Pb (regardless of the compound in which it may have been emitted) and estimates specific to the Pb compounds emitted. That is, emissions estimates for some of the point sources are in terms of mass of Pb compounds, whereas the nonpoint source and mobile source emissions estimates are in terms of mass of the Pb only. For the point sources, approximately 80% are reported as mass of Pb and most of the other 20% are reported as mass of Pb compounds. The high molecular weight of Pb (as compared to elements with which it is associated in Pb compounds), however, reduces the impact of this reporting inconsistency.

Historical trends in emissions are described in Section 2.2.1, information on the various types of Pb sources is presented in Section 2.2.2, the number and geographic distribution of sources is discussed in Section 2.2.3 and the larger Pb point sources are identified in Section 2.2.4. The data sources for, limitations of and our confidence in the information summarized here are described in Section 2.2.5.

2.2.1 Trends in National Emissions: 1980 to 2002

Table 2-1 shows a downward trend in Pb emissions from the fuel combustion, industrial process and solid waste sectors from 1980 through 2002, as well as the dramatic reduction in Pb emissions in the transportation sector due to the removal of Pb from gasoline. While the most dramatic reductions occurred prior to 1990, Pb emissions were further reduced by about 65% (from approximately 5,000 to approximately 1,700 tpy) between 1990 and 2002 (USEPA 1990; USEPA, 2007a). The greatest emission reductions were from mobile sources, primary and secondary Pb and copper smelting, pulp and paper manufacturing, inorganic paint pigment production and steel wire products. As discussed in the CD (Section 2.2.4), reductions in Pb emissions from mobile sources include some associated with the latter period of the “phase-out”

of leaded gasoline. From a national inventory perspective, the stationary source categories that have the largest emissions in the 2002 NEI are summarized briefly in Appendix 2A.

Table 2-1. Trend in Pb emissions (tpy) from 1980 to 2002.

	1980	1985	1990	1995	1999	2002
Transportation	64,706	18,973	1,197	564	-	491[a]
Fuel Combustion	4,299	515	500	490	-	377
Industrial Processes	3,938	2,531	2,474	2,271	-	719
Solid Waste	1,210	871	804	604	-	110
Total	74,153	22,890	4,975	3,929	3303	1,697

[a] This value is not yet reflected in 2002 NEI (vers 3); it will be reflected in version 4, estimated for 2008 release.
Note: Estimates for 1980-1995 are from http://www.epa.gov/airtrends/econ-emissions.html.
Detailed categorization of the 1999 NEI is not available.
Estimates for 2002 are from Version 3 of the 2002 National Emissions Inventory, US EPA (USEPA, 2007a). The estimates for 2002 differ from those in Table 2-8 of the CD due to changes in the 2002 NEI subsequent to publication of the CD.

2.2.2 Types of Pb Sources

Lead is emitted from a wide variety of source types, some of which are small individually but the cumulative emissions of which are large, and some for which the opposite is true. The categories of Pb sources estimated in the 2002 NEI to emit –as a category- more than 5 tons per year (tpy) of Pb are listed in Table 2-2. The main sources of emissions in the 2002 NEI are comprised primarily of combustion-related emissions and industrial process-related emissions. Point source emissions account for about 66% of the national Pb emissions in the 2002 NEI. The point source emissions are roughly split between combustion and industrial processes, while mobile, nonroad sources (emissions associated with general aviation aircraft leaded fuel) account for 29%.

2.2.2.1 Stationary Sources

Table 2-2 presents emissions estimates for stationary sources grouped into descriptive categories. Presence and relative position of a source category on this list does not necessarily provide an indication of the significance of the emissions from individual sources within the source category. A source category, for example, may be composed of many small (i.e., low-emitting) sources, or of just a few very large (high-emitting) sources. Such aspects of a source category, which may influence its potential for human and ecological impacts, are included in the short descriptions of the largest stationary source categories presented in Appendix 2A. The relative sizes of stationary sources represented in the NEI and the geographic distribution of the larger sources are presented in Sections 2.2.2.1 and 2.2.3, respectively.

Table 2-2. Source categories emitting greater than 5 tpy of Pb in the 2002 NEI.

Source Category Description	Total Emissions (tpy)[a]
ALL CATEGORIES [b]	1,697[b]
Mobile sources	491[c]
Industrial/Commercial/ Institutional Boilers & Process Heaters	190
Utility Boilers	168[d]
Iron and Steel Foundries	110
Primary Lead Smelting	59
Hazardous Waste Incineration	47
Secondary Lead Smelting	43
Military Installations	33
Municipal Waste Combustors	33
Integrated Iron & Steel Manufacturing	32
Pressed and Blown Glass and Glassware Manufacturing	32
Stainless and Non Stainless Steel Manufacturing: EAF	32
Mining	31
Lead Acid Battery Manufacturing	27
Secondary Nonferrous Metals	24
Portland Cement Manufacturing	22
Primary Copper Smelting	22
Primary Metal Products Manufacturing	21
Industrial and Commercial Machinery Manufacturing	18
Fabricated Metal Products Manufacturing	14
Electrical and Electronics Equipment Manufacturing	12
Waste Disposal - Solid Waste Disposal	11
Industrial Inorganic Chemical Manufacturing	10
Pulp & Paper Production	10
Sewage Sludge Incineration	10
Mineral Products Manufacturing	9
Secondary Aluminum Production	9
Synthetic Rubber Manufacturing	9
Secondary Copper Smelting	8
Transportation Equipment Manufacturing	8
Ferroalloys Production	7
Nonferrous Foundries	7
Stationary Reciprocating Internal Combustion Engines	7
Commercial and Industrial Solid Waste Incineration	6
Primary Nonferrous Metals--Zinc, Cadmium and Beryllium	6
Residential Heating	6
Asphalt Processing and Asphalt Roofing Manufacturing	5

[a]Some values here differ from those in the CD (Table 2-8) due to changes in the 2002 NEI subsequent to CD publication. Additionally, values just above 5 tpy have been rounded to 5.
[b]Includes 91 tpy Pb emissions from 109 smaller categories (57 tpy in MACT categories and 34 tpy in non MACT).
[c] This value is not yet reflected in 2002 NEI (vers 3); it will be reflected in version 4, estimated for 2008 release.
[d] This estimate of 168 tons, which is based on the 2002 NEI, has uncertainties and differs from estimates in some other studies and inventories. For example, the estimated lead emissions reported to the U.S. EPA's Toxic Release Inventory for year 2004 is about 90 tons for this sector, and the projected estimate for year 2010 presented in the 1998 EPA Utility Air Toxics Study Report to Congress (U.S. EPA, 1998) is 92 tons.

2.2.2.2 Mobile Sources

Thirty-five years ago, combustion of leaded gasoline was the main contributor of Pb to the air. In the early 1970s, EPA set national regulations to gradually reduce the Pb content in gasoline. In 1975, unleaded gasoline was introduced for motor vehicles equipped with catalytic converters. EPA banned the use of leaded gasoline in highway vehicles after December 1995. While Pb is not added to jet fuel that is used in commercial aircraft, military aircraft, or other turbine engine aircraft, currently lead is still added to aviation gasoline (commonly referred to as "avgas") used in most piston-engine aircraft and some types of race cars. Lead emissions from the combustion of avgas are discussed below. Vehicles used in racing are not regulated by the EPA under the Clean Air Act and can therefore use alkyl-Pb additives to boost octane. EPA has formed a voluntary partnership with the National Association for Stock Car Auto Racing (NASCAR) with the goal of permanently removing alkyl-Pb from racing fuels used in the Nextel Cup, Busch and Craftsman Truck Series (CD, p. 2-50). In January of 2006, NASCAR agreed to switch to unleaded fuel in its race cars and trucks beginning in 2008. NASCAR initiated this switch in 2007.

Lead is also present as a trace contaminant in gasoline and diesel fuel and is a component of lubricating oil (CD, pp. 2-45 to 2-48). Inventory estimates from these sources are not currently available. Additional mobile sources of Pb include brake wear, tire wear, and loss of Pb wheel weights (CD, pp. 2-48 to 2-50). Emission rates for Pb from brake wear have been published but inventory estimates have not yet been developed from these data (Schauer et al., 2006). Robust estimates of Pb from tire wear and wheel weights are not available. Currently, Pb from combustion of leaded avgas is the only mobile source of Pb included in the 2002 NEI.

Currently, there are two main types of leaded avgas used, 100 Octane and 100 Octane Low Lead (100 LL), which can contain up to 1.12 grams Pb per liter (g/L) (0.009347 pounds per gallon, lb/gal) and 0.56 g Pb/L (0.004673 lb/gal), respectively (ASTM D 910). The vast majority of leaded avgas used is 100LL. In 2002 approximately 280 million gallons of avgas were supplied to the U.S. (DOE, 2006) contributing an estimated 491 tons of lead to the air and comprising 29% of the national Pb inventory.[1]

Lead emission estimates from piston-engine aircraft in the 2002 NEI are allocated to 3,410 airports located throughout the United States (USEPA, 2007b). These Pb emissions are

[1] Lead emissions from general aviation are calculated as the product of the fuel consumed, the concentration of Pb in the fuel and the factor 0.75 to account for an estimated 25% of Pb being retained in the engine and/or exhaust system of the aircraft. The estimate of 25% Pb retention was derived from estimates from light-duty gas vehicles operating on leaded fuel and is an upper-bound estimate of the amount of Pb retained in a piston-engine aircraft. Smaller retention values would proportionally increase the overall mobile source Pb inventory.

allocated to each airport based on its percentage of piston-engine operations nationwide. These operations for 2002 can be found in the *Terminal Area Forecast* (TAF) system, which is the official forecast of aviation activity at FAA facilities. Airport-specific Pb emissions estimates in the NEI include Pb emitted during the entire flight (i.e., not limited to the landing and take-off cycle and local operations). EPA is using this allocation approach for Pb because it is important to account for all of the Pb emitted by avgas use. There is currently not an alternative approach for incorporating all the Pb emissions from aircraft into the NEI. EPA understands that allocating lead emissions to airports from operations outside the landing-takeoff cycle and local flying operations has a tendency to overstate the local emissions near airports because longer duration (e.g., itinerant) flights emit lead at altitude as well as in the local area near the airport.

Airport-specific Pb emissions estimates in the 2002 NEI do not include the following airport-related sources of Pb: evaporative losses of Pb from fuel storage and distribution, military aircraft combustion emissions, and the small amounts of tetraethyl-lead (TEL) discarded on the tarmac by pilots after their fuel check. Lead emissions from fuel storage and distribution are estimated to total 0.3 tons nationally and are included in the NEI, but not assigned to specific airports. Data regarding military piston engine aircraft emissions are supplied to EPA by states. The 2002 version 3 inventory estimates for this category did not include state-submitted data, but future updates to the NEI will include these estimates.

These current NEI estimates provide a valuable comparison with other ambient sources of Pb. Future upgrades to these estimates and assessments specific to individual airports could include more refined local data including characteristics of local operations (e.g., landings and take-offs), Pb retention in piston engines, and fuel consumption rates.

Among the airports in the 2002 NEI where piston-engine aircraft operate, approximately one percent of US airports listed have estimated Pb emissions of greater than one ton per year, a greater percentage has estimated Pb emissions between one ton and 0.1 ton per year, while the majority of airports are estimated to have Pb emissions less than 0.1 ton per year. Table 2-3 below demonstrates these estimated emission ranges.

Table 2-3. Lead emissions from leaded aviation gas use in the 2002 NEI version 3.

Emissions Range (tpy)	Number of Airports	Total Emissions (tpy)
< 0.1	2,104	76.7
0.1 to 1.0	1,270	367.5
> 1	36	47.1
Summary	3,410	491.3

2.2.2.3 Resuspension of Previously Deposited Pb and other Sources

Resuspension of soil-bound Pb particles and contaminated road dust has been reported to be a significant source of airborne Pb (CD, Section 2.3.3, and p. 2-62). Quantitative estimates of resuspension-related emissions, however, are not included in the 2002 NEI. Studies of emissions in southern California indicate that Pb in resuspended road dust may represent between 40% and 90% of Pb emissions in that area (CD, p. 2-65). Lead concentrations in suspended soil and dust, however, vary significantly (CD, p. 2-65). In general, the main drivers of particle resuspension are typically mechanical stressors such as vehicular traffic, construction and agricultural operations, and to a lesser extent, the wind. Lead resuspended in soil near roadways that was in place during the use of leaded gasoline may be a notable emissions source if or when such soil is disturbed (e.g., road widening or building construction).

Understanding the physics of resuspension from natural winds requires analyzing the wind stresses on individual particles and although this analysis can be accurate on a small scale, predicting resuspension on a large scale generally focuses on empirical data for soil movement due to three processes: saltation, surface creep, and suspension (CD, pp. 2-62 to 2-63). Rather than a continuous process, resuspension may occur as a series of events. Short episodes of high wind speed, dry conditions, and other factors conducive to resuspension may dominate annual averages of upward flux (CD, p. 2-65). All of these factors complicate emissions estimates (CD, Section 2.2.1) such that quantitative estimates for these processes remain an area of significant uncertainty.

Other sources not currently included in the NEI are emissions of Pb from natural sources, such as wind-driven resuspension of soil with naturally occurring Pb, sea salt spray, volcanoes, wild forest fires, and biogenic sources (CD, Section 2.2.1). Estimates for these emissions, some of which have significant variability (CD, p. 2-13) have not been developed for the NEI, as quantitative estimates for these processes remain an area of significant uncertainty.

2.2.3 Number and Geographic Distribution of Sources

The geographic distribution and magnitude of Pb emissions in the U.S. from all sources identified in the 2002 NEI is presented in Figure 2-2, in terms of emissions density (defined here as tons per area, square mile, per county). This presentation indicates a broad distribution of Pb emissions across the U.S., with the highest emitting counties scattered predominantly within a broad swath from Minnesota to southern New England and southward.

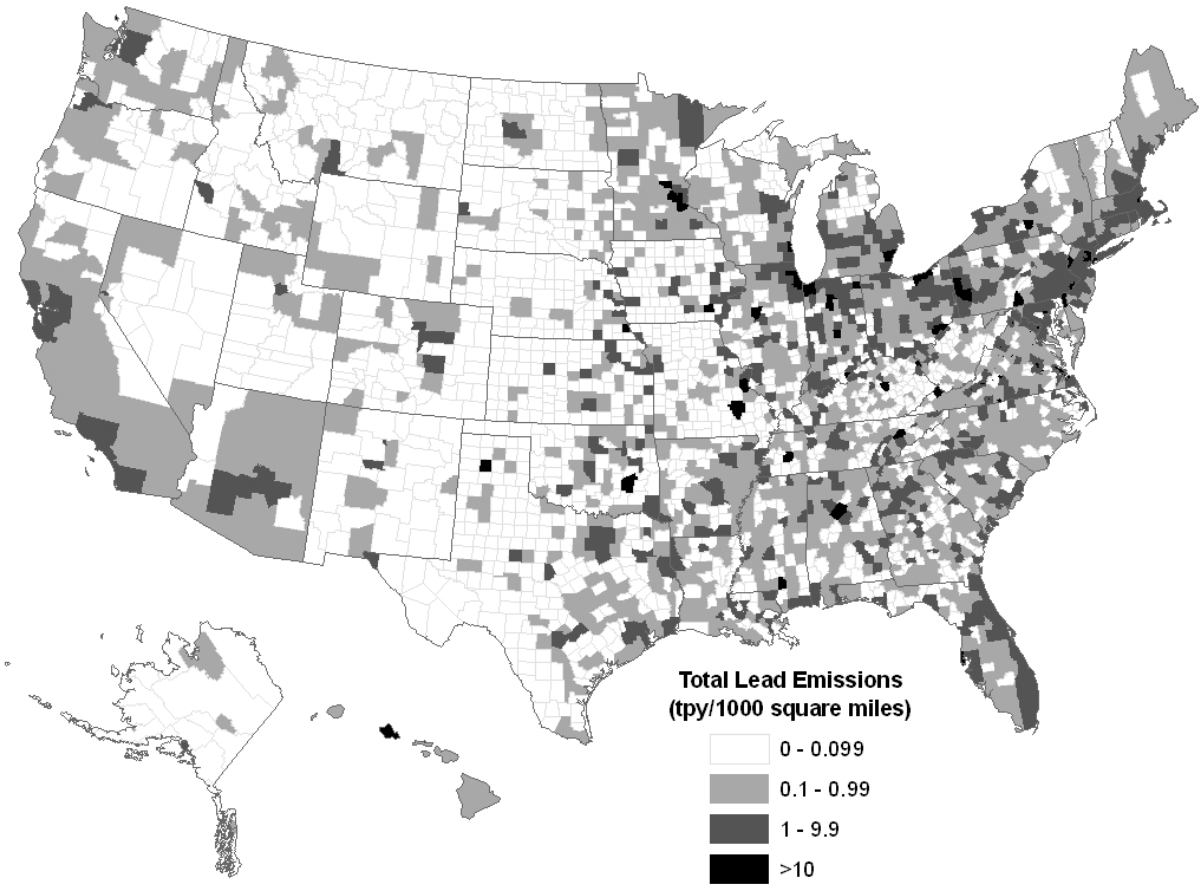


Figure 2-2. Emissions density from all Pb sources in the 2002 NEI.

Within the NEI, emissions from stationary sources may be associated with specific "points" (i.e., point sources) or with activities estimated to occur with some frequency within an "area" such as a county (area sources) or with mobile sources (see Section 2.2.2.2). Emissions from all stationary sources represented in the NEI are presented in Figure 2-3, in terms of emissions density (tons per area, square mile, per county).

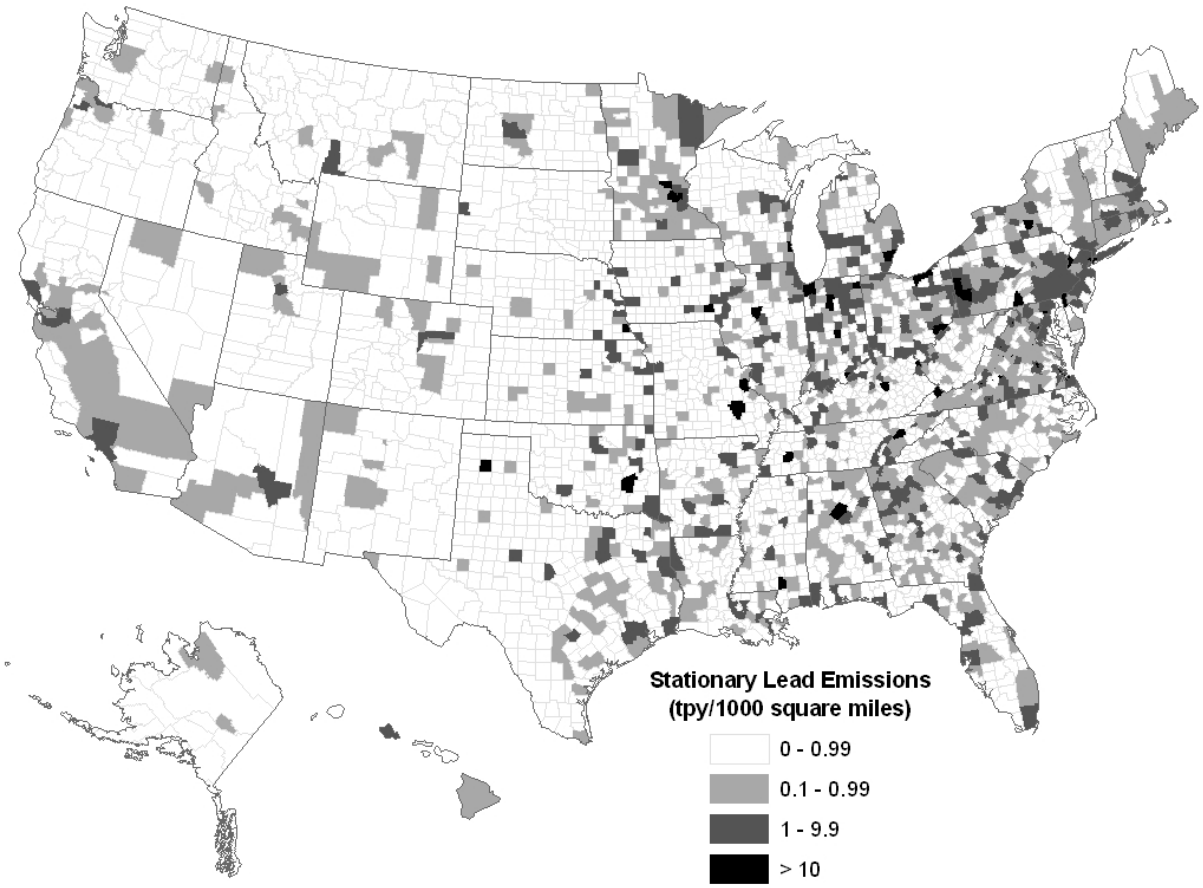


Figure 2-3. Emissions density from all stationary sources of Pb in 2002 NEI.

There are some 13,067 point sources (industrial, commercial or institutional) in the 2002 NEI, each with one or more processes that emit Pb to the atmosphere (Table 2-4). Most of these sources emit less than 0.1 tpy Pb. There are approximately 1,300 point sources of Pb in the NEI with estimates of emissions greater than or equal to 0.1 tpy and these point sources, combined, emit 1058 tpy, or 94% of the Pb point source emissions. In other words, 94% of Pb point source emissions are emitted by the largest 10% of these sources.

Table 2-4. Size distribution of point sources within the 2002 NEI and associated estimated emissions.

Emissions Range (tpy)	Number of Sources	Total Emissions (tpy)	Average Emissions per Source (tpy)
< 0.1	11,800	73	<0.01
0.1 to 1.0	1,028	326	0.3
1.0 to 5	210	421	2
> 5	29	301	10
Summary	13,067	1121	

2.2.4 Largest Pb Point Sources in the 2002 NEI

While Section 2.2.2 described source categories that rank highest due to cumulative national Pb emissions, this section is intended to consider Pb emissions on the individual source level. The geographic distribution of point sources estimated to emit greater than 1 tpy is presented in Figure 2-4.

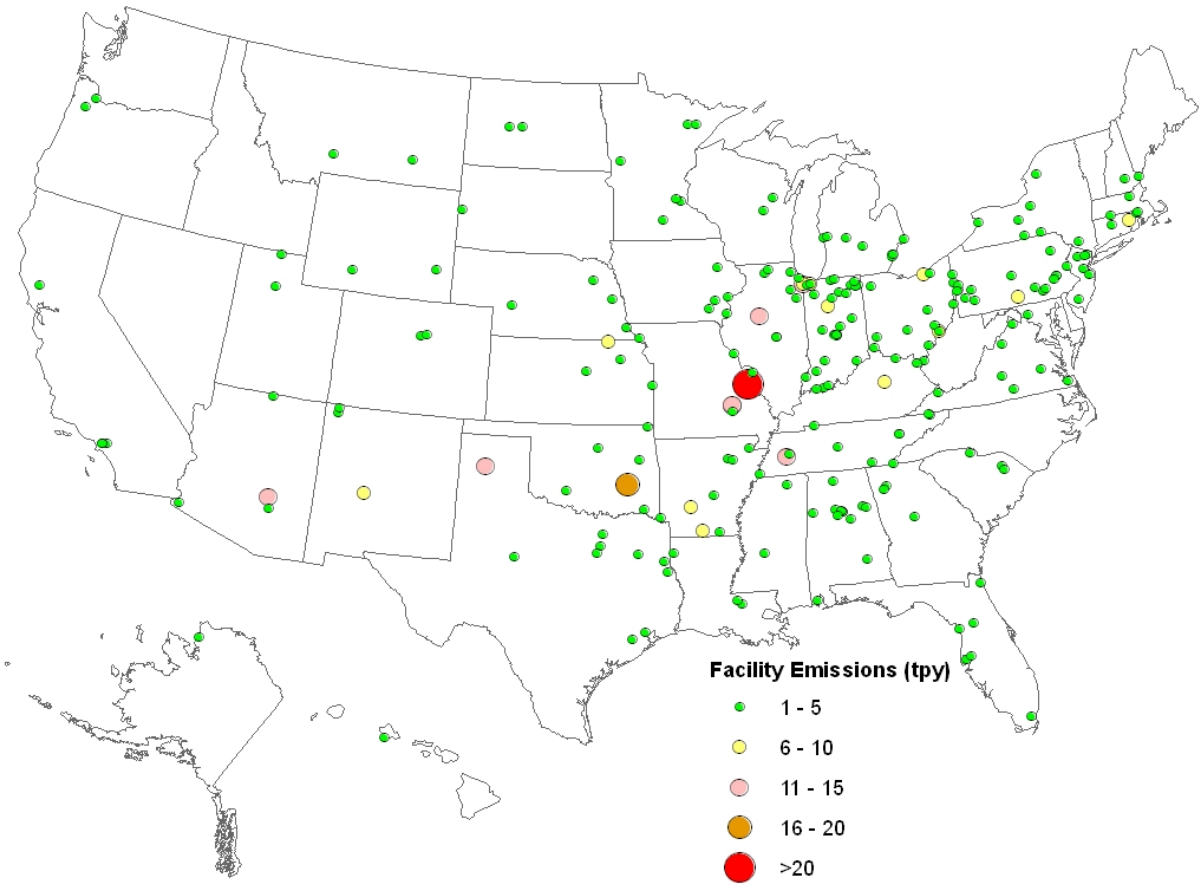


Figure 2-4. Geographic distribution of point sources with >1 tpy Pb emissions in 2002 NEI.

As mentioned in Section 2.2.3, the 2002 NEI includes 30 facilities with emissions estimated to be greater than or equal to 5 tons per year (see Table 2-4). Most of these sources (Table 2-5) are metallurgical industries, followed by waste disposal facilities and manufacturing processes.

Table 2-5. Point Sources with Pb emissions in 2002 NEI greater than or equal to 5 tpy.

Source Category Name	State	County Name	2002 Point Emissions (TPY)[a]
Primary Lead Smelting	MO	Jefferson County	58.8
Military Installation	OK	Pittsburg County	17.2
Mining	MO	Reynolds County	15.4
Copper Refining[b]	TX	Potter County	13.9
Primary Copper Smelting	AZ	Gila County	12.8
Electric Arc Furnaces	IL	Peoria County	12.5
Secondary Lead Smelting	MO	Iron County	12.4
Integrated Iron & Steel Manufacturing	IN	Lake County	11.3
Pressed and Blown Glass and Glassware Manufacturing	TN	Madison County	10.9
Military Installation	PA	Franklin County	10.4
Hazardous Waste Incineration	AR	Union County	10.2[c]
Lead Acid Battery Manufacturing	KY	Madison County	9.9
Industrial and Commercial Machinery Manufacturing	KS	Marshall County	8.2
Synthetic Rubber Products Manufacturing - Fabric Coating	IN	Cass County	7.4
Commercial and Industrial Solid Waste Incineration	AR	Clark County	7.3
Iron and Steel Foundries	OH	Cuyahoga County	7.3
Integrated Iron & Steel Manufacturing	IN	Porter County	7.2
Integrated Iron & Steel Manufacturing	IN	Lake County	6.1
Mineral Products Manufacturing	NM	Socorro County	6.1
Commercial and Industrial Solid Waste Incineration	CT	Windham County	5.8
Ferroalloys Production	OH	Washington County	5.7
Nonferrous Foundries	NE	Nemaha County	5.5
Portland Cement Manufacturing	MD	Frederick County	5.4
Hazardous Waste Incineration	OH	Lorain County	5.4
Coke Oven	VA	Buchanan County	5.1
Iron and Steel Foundries	IA	Jefferson County	5.1
Mining	MO	Reynolds County	5

[a] (USEPA, 2007)
[b] This entry is included in the total provided for "secondary nonferrous metals" in Table 2-2.
[c] Following compliance with the MACT standards in 2008, Pb emissions are estimated to be 0.7 tpy.

2.2.5 Data Sources, Limitations and Confidence

The Pb emissions information presented in the previous sections is drawn largely from EPA's NEI for 2002 (USEPA, 2007a). The NEI is based on information submitted from State, Tribal and local air pollution agencies and data obtained during the preparation of technical support information for EPA's hazardous air pollutant regulatory programs. EPA has recently developed version 3 of the NEI for 2002 and that version is anticipated to be posted on the EPA's CHIEF website soon at (http://www.epa.gov/ttn/chief/net/2002inventory.html). The information presented in this document is based on version 3.

The process of identifying sources that emit Pb into the air has been ongoing since before the Clean Air Act of 1970. The comprehensiveness of emission inventories generally, and the NEI, specifically, depends upon knowledge of source types emit Pb, their locations and their operating characteristics, as well as the reporting of this information to the inventory. As noted above, the NEI relies on information that is available from a variety of sources for this information. There are numerous steps, each with its own uncertainties, associated with the development of this information for use in the emissions inventory. First, the categories emitting Pb must be identified. Second, the sources' processes and control devices must be known. Third, the activity throughputs and operating schedules of these sources must be known. Finally, we must have emission factors to relate emissions to the operating throughputs, process conditions and control devices. The process, control device, throughputs and operating schedules are generally available for each source. However, the emission factors represent average emissions for a source type and average emissions may differ significantly from source to source. In some cases, emissions testing provides source-specific information. In others, emissions factors must be estimated from similar sources or source categories or other information. More information on emission factors and the estimation of emissions is found in the introduction to EPA's Compilation of Air Pollutant Emissions Factors (USEPA, 2006a). Further information on emission factors is available at: http://www.epa.gov/ttn/chief/ap42/.

The NEI is limited with regard to Pb emissions estimates for some sources such as resuspended road dust (Section 2.2.2.3), biomass burning and trace levels of Pb in motor fuel and lubricating oil (Section 2.2.2.2), and others. We have not yet developed estimates for the NEI of Pb emissions associated with resuspension of Pb residing in roadway dust and nearby surface soil. Emissions estimates are also not yet in the NEI for the miscellaneous categories of on-road emissions (e.g., combustion of fuel with Pb traces, lubricating oil, mechanical wear of vehicle components, etc.) and Pb that may be emitted from wildfires.

The 2002 NEI underwent extensive 3-month external review, including a review of the process for developing the inventory which includes extensive quality assurance and quality control steps (QA/QC). For example, we created a QA/QC process and tracking database to provide feedback reports to point source data providers at regular intervals during the QA of the data. The feedback reports included the following 4 QC reports: data integrity, latitude/longitudes QC, stack parameters QC, and emissions QC. Further, there was additional QA/QC conducted for emission inventory information for facilities that are included in the Risk and Technology Review (RTR) source categories (60FR14734). As a result we have strong confidence in the quality of the data for these facilities. Version 3 of the 2002 NEI used in RTR has undergone additional peer review and QA/QC based on comments received to Docket # EPA-HQ-OAR-2006-0859.

In summary, generic limitations to the 2002 NEI include the following:

- Consistency: The 2002 NEI for Pb is a composite of emissions estimates generated by state and local regulatory agencies, industry, and EPA. Because the estimates originated from a variety of sources, as well as for differing purposes, they will in turn vary in quality, whether Pb is reported for particular source types, method of reporting compound classes, level of detail, and geographic coverage.
- Variability in Quality and Accuracy of Emission Estimation Methods: The accuracy of emission estimation techniques varies with pollutants and source categories. In some cases, an estimate may be based on a few or only one emission measurement at a similar source. The techniques used and quality of the estimates will vary between source categories and between area, major, and mobile source sectors. Generally, the more review and scrutiny given to emissions data by states and other agencies, the more certainty and accuracy there is in that data.

2.3 AIR QUALITY MONITORING DATA

The EPA has been measuring Pb in the atmosphere since the 1970s. For the most part, Pb concentrations have decreased dramatically over that period. This decrease is primarily attributed to the removal of Pb from gasoline; however, some individual locations still have Pb concentrations above the level of the NAAQS. The following sections describe the ambient Pb measurement methods, the sites and networks where these measurements are made, as well as how the ambient Pb concentrations vary geographically and temporally.

Ambient air Pb concentrations are measured by four monitoring networks in the United States, all funded in whole or in part by EPA. These networks provide Pb measurements for three different size classes of airborne particulate matter (PM): total suspended PM (TSP), PM less than or equal to 2.5 μm in diameter ($PM_{2.5}$), and PM less than or equal to 10 μm in diameter (PM_{10}). The networks include the Pb TSP network, the $PM_{2.5}$ Chemical Speciation Network (CSN), the Interagency Monitoring of Protected Visual Environments (IMPROVE) network, and the National Air Toxics Trends Stations (NATTS) network. The subsections below describe each network and the Pb measurements made at these sites.

In addition to these four networks, various organizations have operated other sampling sites yielding data on ambient air concentrations of Pb, often for limited periods and/or for primary purposes other than quantification of Pb itself. Most of these data are accessible via EPA's Air Quality System (AQS): http://www.epa.gov/ttn/airs/airsaqs/. In an effort to gather as much air toxics data, including Pb, into one database, the EPA and State and Territorial Air Pollution Program Administrators and the Association of Local Air Pollution Control Officials (STAPPA/ALAPCO) created the Air Toxics Data Archive. The Air Toxics Data Archive can be accessed at: http://vista.cira.colostate.edu/atda/.

2.3.1 Ambient Pb Measurement Methods

A number of methods are used to collect Pb and measure Pb concentrations in the atmosphere. Most methods use similar sample collection approaches. Ambient air is drawn through an inlet for a predetermined amount of time (typically 24 hours) and the PM is collected on a suitable filter media. After the sample has been collected, the filter may be used to determine the mass of PM collected prior to then being used for determination of Pb. The filter is chemically extracted and analyzed to determine the Pb concentration in the particulate material. The concentration of Pb found in the atmosphere, in $\mu g/m^3$, is calculated based on the concentration of Pb in the volume extracted, the size of the collection filter, and the volume of air drawn through the filter.

The primary factors affecting the measurements made are the sampling frequency, duration of sampling, type of inlet used, volume of air sampled, and the method of analyzing the filter for Pb content. The following paragraphs describe how these factors affect the Pb measurements.

2.3.1.1 Inlet Design

In ambient air monitors, a number of inlet designs have been developed that allow certain particle size ranges to be sampled. The inlets use either impaction or cyclone techniques to remove particles larger than a certain size (the size cutpoint) from the sample stream. Three particle size cutpoints are used in ambient Pb measurements including TSP, $PM_{2.5}$, PM_{10}. The TSP inlet is designed to allow as much suspended particulate into the sampling device as possible while protecting against precipitation and direct deposition on to the filter (nominally 25 to 45 micrometers) (USEPA, 2004c).

Sampling systems employing inlets other than the TSP inlet will not collect Pb contained in the PM larger than the size cutpoint. Therefore, they do not provide an estimate of the total Pb in the ambient air. This is particularly important near sources which may emit Pb in the larger PM size fractions (e.g., fugitive dust from materials handling and storage).

2.3.1.2 Volume of Air Sampled

The amount of Pb collected is directly proportional to the volume of air sampled. Two different sampler types have evolved for PM and Pb sampling – a high-volume and a low-volume sampler. High-volume samplers draw between 70 and 100 m^3/hr of air through an 8 inch by 10 inch filter (0.05 m^2 filter area). Low-volume samplers typically draw 1 m^3/hr through a 47 mm diameter filter (0.002 m^2 filter area). Currently all Federal Reference Method (FRM) and Federal Equivalence Method (FEM) for Pb-TSP are based on high-volume samplers.

2.3.1.3 Sampling Frequency

The frequency of Pb sampling used in the U.S. varies between one sample every day (1 in 1 sampling) to the more common frequency of one sample every 6 days (1 in 6 sampling). Semi-continuous methods for the measurement of ambient metals (including Pb) are currently being explored which would allow for more frequent sampling (as frequent as 1 sample per hour), but much more work is needed on these methods before they can be deployed in a network setting.

More frequent sampling reduces the uncertainty in estimates of quarterly or annual averages associated with temporal variations in ambient concentrations. However, the costs of sampling and analysis are directly tied to sample frequency. As such, it is necessary to evaluate the reduction in measurement error versus the increase in sampling and analysis costs when selecting the required sampling frequency. A discussion of the observed temporal variation of Pb measurements is given later in this section.

2.3.1.4 Sample Analysis

After the samples have been collected on filters and the filters have been weighed, the filters are analyzed for Pb content. A number of analytical methods can be used to analyze the filters for Pb content including x-ray fluorescence analysis (XRF), proton-induced x-ray emission (PIXE), neutron activation analysis (NAA), atomic absorption (AA), or inductively-coupled plasma mass spectrometry (ICP/MS) (CD, pp. 2-80 to 2-81). A detailed discussion of these methods was given in the 1986 CD (USEPA, 1986), and the reader is referred to that document for more information on these analytical methods. A search conducted on the AQS database[2] shows that the method detection limits for all of these analytical methods (coupled with the sampling methods) are very low, ranging from 0.01 $\mu g/m^3$ to as low as 0.00001 $\mu g/m^3$, and are more than adequate for determining compliance with the current NAAQS.

2.3.2 Pb-TSP

This network is comprised of state and locally managed Pb monitoring stations which measure Pb in TSP, i.e., particles up to 25 to 45 microns. These stations use samplers and laboratory analysis methods which have either FRM or FEM status. The FRM and FEM method descriptions can be found in the U.S. Code of Federal Regulations, Section 40 part 50, Appendix G. Sampling is conducted for 24-hour periods, with a typical sampling schedule of 1 in 6 days. Some monitoring agencies "composite" samples by analyzing several consecutive samples together to save costs and/or increase detection limits.

[2] EPA's AQS can be accessed at http://www.epa.gov/ttn/airs/airsaqs/

2.3.2.1 Monitor Locations

The locations of Pb-TSP sites in operation between 2003 and 2005 are shown in Figure 2-5. State and local agencies are required to operate two Pb-TSP monitors in any area which has exceeded the NAAQS in the last two years (40 CFR 58 Appendix D). State and local agencies have the latitude to operate more monitors beyond the minimum requirement. Agencies which operate these sites report the data to EPA's AQS where they are accessible via several web-based tools. EPA's series of annual air quality trends reports have used data from this network to quantify trends in ambient air Pb concentrations. The most recent Trends report for Pb-TSP can be found at http://www.epa.gov/airtrends/lead.html.

A review of the Pb-TSP network's coverage of the highest Pb emitting sources (as identified in the current version of the 2002 NEI) was conducted as part of preparing this document. This review indicates that many of the highest Pb emitting sources in the 2002 NEI do not have nearby Pb-TSP monitors. This review indicates that only 2 of 27 facilities (both Pb smelters[3]) identified as emitting greater than 5 tpy have a Pb-TSP monitor within 1 mile. The lack of monitors near large sources indicates we are likely currently underestimating the extent of occurrences of relatively higher Pb concentrations. Additionally, none of the 189 Pb-TSP sites included in the 2003-2005 analysis described in Sections 2.3.2.3 and 2.3.2.4 are located within a mile of airports identified in the NEI as an airport where piston-engine aircraft operate (i.e., aircraft that still use leaded aviation fuel).[4]

[3] Primary and secondary smelters were the source types given particular priority at the time of the last Pb NAAQS review (USEPA, 1990; USEPA, 1991).

[4] While there are limited historical data (going back to 1993) in AQS for 12 Pb-TSP monitoring sites operating within one mile of 11 of these airports, time constraints have limited the extent of our analysis here of these data or of other such data that may be available elsewhere.

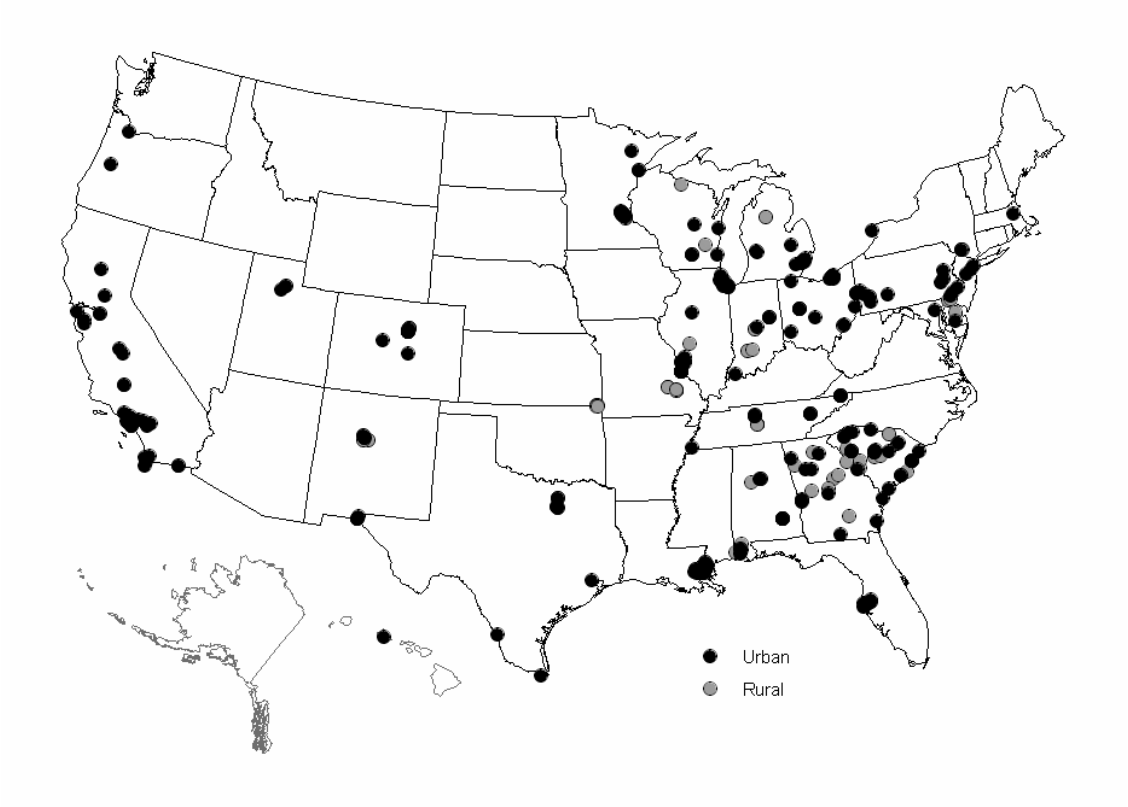


Figure 2-5. Pb-TSP monitoring sites: 2003-2005.

The number of sites in the Pb-TSP network has decreased significantly since the 1980s (see Figure 2-6). The number of sites in the network reached its highest point in 1981 (946 sites). About 250 sampling sites operated during 2005. This decline in the number of Pb-TSP sites is attributable to the dramatic decrease in Pb concentrations observed since the 1980s and the need to fund new monitoring objectives (e.g., $PM_{2.5}$ and ozone monitoring). Lead-TSP sites in lower concentration areas were shut down to free up resources needed for monitoring of other pollutants such as $PM_{2.5}$ and ozone.

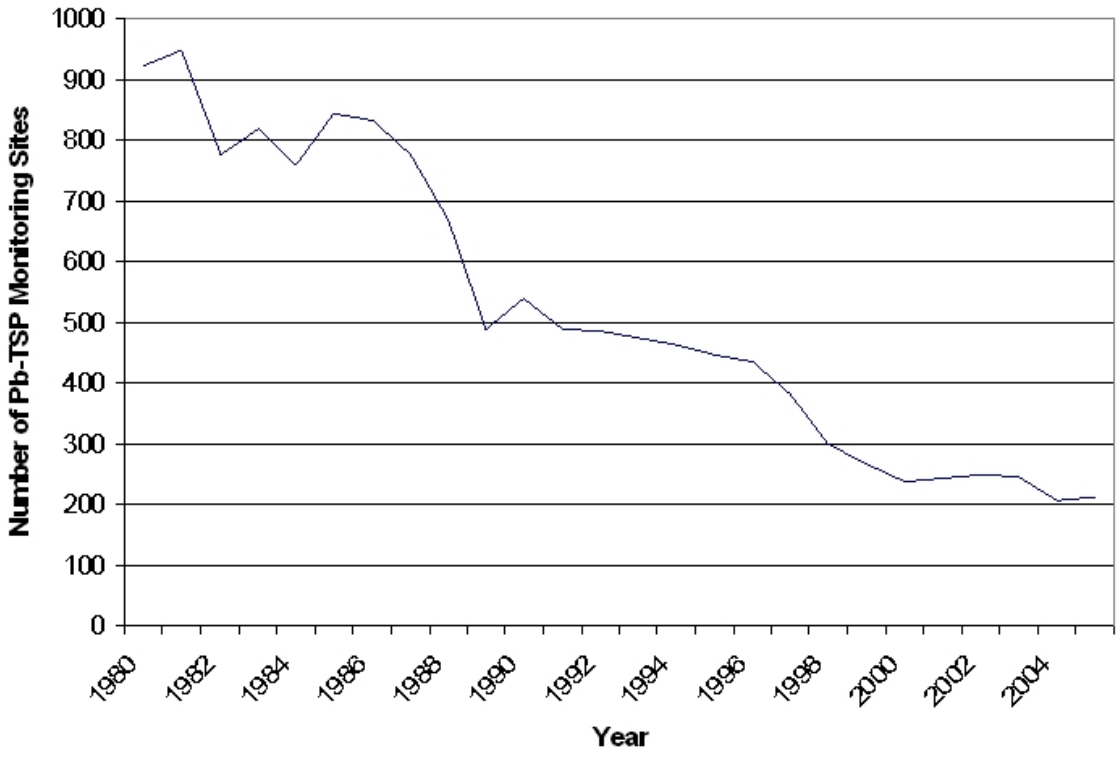


Figure 2-6. Change in the number of Pb-TSP monitoring sites from 1980 to 2005.

2.3.2.2 Historical Trend

Airborne concentrations of Pb in the United States have fallen dramatically over the last 30 years due largely to the phase-out of leaded gasoline additives. Figure 2-7 shows national trends in airborne TSP Pb concentrations for two subsets of NAAQS FRM monitoring sites not considered to be "source-oriented" (see Section 2.3.2.3.3), one for 1980 through 1989 (representing 168 sites), and the second for 1990 through 2006 (representing 44 sites). Two separate graphs were used to characterize the overall (1980 through 2006) long-term trend due to data limitations associated with a single graph; only 15 sites met the inclusion criterion for the entire 27 year period. The data in both graphs are plotted in terms of the site-level maximum quarterly arithmetic mean for each year (the form of the current NAAQS) and are shown in relation to the current NAAQS of 1.5 $\mu g/m^3$ (maximum quarterly average). The monitors used in this analysis are typically population-oriented urban monitors that are not source-oriented. The left plot shows an 86 percent decrease in national average maximum quarterly means from 1980 to 1989 and the right plot shows a 54 percent decrease in the same statistic from 1990 to 2006. The combination of these equates to an overall decline of about 94 percent from 1980 to 2006. The single, much smaller subset of sites that cover the full 27-year period (n=15) showed a 95

percent decrease in the same metric from 1980 through 2006. Since the early 1980's, major declines over several orders of magnitude have been observed not only in urban areas, but also in rural regions and remote locations. The sharp decline through the 1980s has also been observed in Pb associated with fine particles (less than or equal to 2.5 microns) at remote and rural sites throughout the United States and have been attributed to the phase out of leaded gasoline (Eldred and Cahill, 1994).

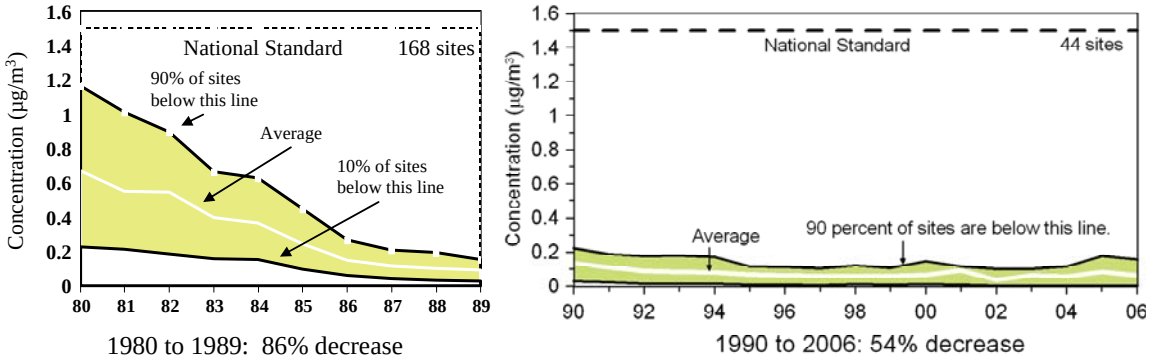


Figure 2-7. Airborne Pb -TSP concentrations, averaged across continuously operating monitoring sites: 1980-1989 and 1990-2006. (Sources: left plot - AQS data extracted 10/10/07; right plot – EPA 2007.)

2.3.2.3 Data Analysis Details

Lead-TSP data collected in 2003-2005 (parameter code 12128, durations '7' and 'C') were extracted from EPA's AQS on May 22, 2007. Most of the monitors reporting data for that timeframe utilized FRM or FEM, and therefore, are candidates for comparisons to the NAAQS. Some of the Pb-TSP monitors, however, were placed for nonregulatory purposes (e.g., for toxics monitoring initiatives) and utilize methods other than a FRM or FEM. Although measurements from these monitors cannot be compared to the NAAQS for purposes of non-attainment decisions, they were considered worthy for inclusion in this national Pb-TSP characterization. The non-FRM/FEM Pb-TSP methods typically have lower reported uncertainties and detection limits than the FRM/FEM. Detection limits vary somewhat even for the data generated using FRM or FEM. In summary aggregations, the AQS generally substitutes one half the method detection level (MDL) for reported concentration readings less than or equal MDL. That protocol was not utilized in this national aggregation; data were used 'as reported' to AQS. Only a small number of Pb-TSP measurements for 2003-2005 were flagged for exceptional events (e.g., structural fires, chemical spills, sandblasting); none of the exceptional event flag-flagged data, however, were concurred (i.e., approved) by the associated EPA Regional Office. Data flags were ignored in this analysis.

2.3.2.3.1 Screening Criteria

Measurements of Pb-TSP with 24-hour sample collection duration were reported to AQS for more than 350 monitors for the years 2003 to 2005. 189 of those monitors met the following screening criteria and were used in this national characterization. The completeness criteria employed for this national characterization were: 1) a minimum of 10 observations per quarter, 2) for at least one full year (all 4 quarters), and 3) at least 9 months with 4 observations each[5]; all three criteria had to be met for inclusion. 209 monitors met the three-pronged criteria; of these 209 monitors, 20 were collocated with another complete monitor. Only one monitor from each collocated pair (i.e., from each site location) was kept in the analysis, specifically the one with highest 3-year maximum quarterly mean. Thus, data from 189 monitors at 189 distinct locations were actually used; 109 of these monitors/sites had 3 complete years, 36 monitors/sites had two complete years, and 44 monitors/sites had only one compete year. Complete quarters that were not part of a complete year were used. Likewise, all complete months were used, even if they did not correspond to the complete years. The 189 sites have an average of about 10 complete quarters and 28 complete months. The 189 utilized monitors are listed along with various summary and demographic data in Appendix 2B, Tables 2B-1 and 2B-2.

2.3.2.3.2 Urban Sites

The 189 monitors are located in 86 counties, in 23 states. 140 of the 189 sites were deemed 'urban' and aggregated as such. Sites were labeled 'urban' if they located within a defined urbanized area or urban cluster (per 2000 Census geographic definitions). All of the 'urban' designated sites were located in a Core Based Statistical Area (CBSA) per 2003 CBSA geographic definitions. CBSA is a collective term for both metropolitan and micropolitan statistical areas. A metro area contains a core urban area of 50,000 or more population, and a micro area contains an urban core of at least 10,000 (but less than 50,000) population. Each metro or micro area consists of one or more whole counties and includes the counties containing the core urban area, as well as any adjacent counties that have a high degree of social and economic integration with the urban core. The monitors in the analysis map to 65 unique CBSA's. Only 10 of the 189 monitors are not located within a CBSA. CBSA's do not always exclusively encompass wholes or parts of urbanized areas and/or urbanized clusters. 39 of the 189 Pb monitoring sites are located in a CBSA but are not classified as 'urban'. Although 'urban' locations (i.e., parts of urbanized areas or urban clusters) are found in counties not defined as (or part of) a CBSA, all of the 140 urban sites in this characterization are located in a

[5] Quarterly means calculated with less than ten observations, annual means calculated with only three quarters, and monthly means derived with less than four observations were also considered valid if that mean value exceeded the level of the current standard (i.e., 1.5 $\mu g/m^3$ for quarterly mean).

CBSA. 91 of the 140 urban sites are located in CBSA's with one million or greater population. Note that the 65 CBSA's containing the Pb-TSP monitoring sites are generally among the largest in the nation (with respect to total population). Almost 75 percent of the Pb-TSP CBSA's are larger (in population) than the highest-population 75 percent of all U.S. CBSA's. With respect to total CBSA population, the five overall largest CBSA's and 18 of the largest 25 contain at least one Pb-TSP monitor.

2.3.2.3.3 Source-oriented Sites

Monitoring sites were classified as being "source oriented" with regard to sources of Pb emissions if: 1) they met a graduated (or sliding scale of) cumulative emission ton per year by distance criterion, or 2) they were classified as source oriented in previous EPA analysis. Sixty of the 189 Pb-TSP sites met at least one of these criteria. Of the 60 total source-oriented sites, 40 met the first criterion and 51 met the second.

The graduated cumulative emission ton per year to distance criterion (criterion #1) utilized the 2002 (version 3) national emission inventory (NEI) for Pb point sources and Pb area nonpoint sources. The Pb point source emissions were assigned to the specific facility point locations (longitude/latitude coordinates), and the area nonpoint inventory was allocated to Census tracts and based on an assumed uniform distribution across those extents. To meet the graduated 'source-oriented' criterion, a Pb monitoring site had to be within at least one multiple of 0.1 miles (checking up to 1 mile away) for a corresponding multiple of 0.1 tpy of total point and nonpoint emissions (e.g., Within 0.1 mile of a cumulative 0.1 tpy, within 0.2 miles of a cumulative 0.2 tpy, within 0.3 miles of a cumulative 0.3 tpy, …, or within 1.0 miles of a cumulative 1.0 tpy). The area nonpoint contribution to the comparison cumulative inventory was based on the composite emission densities of the Census tract in which a site was located and all other tracts with population centroids within a mile of the monitoring site.

The sites 'classified as source oriented in previous EPA analysis' (criterion #2) were identified via a reference list that was last updated in 2003; this list has been utilized in recent EPA Trends Report analysis. The list encompasses 114 sites. Many of the monitoring sites on this list did not have data that met the data completeness criteria for 2003–2005 because they have permanently discontinued Pb monitoring, most ostensibly because the associated nearby Pb emission source(s) has implemented controls, closed operations, and/or reduced production. Some ambient monitoring sites continue monitoring even after significant reported reductions in nearby new Pb emissions. Sites were not screened out of the source-oriented classification in those instances. In addition to including such sites in the source-oriented category, these sites were separately reviewed to see if they still had higher concentrations than nonsource sites because of previously emitted Pb becoming resuspended into the air and/or possible emission

estimate errors. These sites are termed, "previous source-oriented sites" in relevant figures and tables.

There are only nine sites that were categorized as "previous" source-oriented in this national analysis. The particular circumstances related to the emission sources associated with these nine monitoring sites vary considerably. In some instances the emission sources have been closed for more than a decade and the facility locations have undergone remediation. For other sources, production and clean-up status was not fully ascertained. In the case of one emission source (that has numerous nearby monitoring sites), production was reportedly halted at the end of 2003 and no significant clean-up activity has yet been undertaken. For the monitoring sites associated with this source, two sets of statistics were generated (or attempted). Statistics representing the entire 3-year period were calculated and used everywhere applicable except for the "previous" category, and statistics representing the post-production period (2004-2005) were generated and used for the "previous" classification. Note that some of these monitoring sites met the data completeness criteria for the 3-year period (2003-2005) but not for the 2-year period (2004-2005). Because of the small number of sites included in the "previous" source oriented classification and the uncertainty in the emission source status, results for this category should be viewed with caution.

2.3.2.3.4 Population Associations

Two population statistics were summarized with the Pb concentration data, the "total population" within 1 mile of the site (a.k.a., a "radial mile") and the "under age 5 population" within 1 mile of the site. Populations assigned to sites were based on Census block group population densities, specifically the density of the block group in which the site was located and (if relevant) the density of other block groups with population centroids within 1 mile of the site. The average population density across these blocks (expressed in square miles) was multiplied by pi (3.142) to obtain a radial mile population (i.e., the number of people living within a one mile radius of the monitoring site). Population data and block group definitions utilized are from the 2000 Census.

The median size of populations associated with the Pb-TSP monitors in this analysis is about 6,200 and the corresponding under age 5 median population is around 420. These median populations are slightly smaller than the overall U.S. block group median radial mile populations (19 percent smaller for total and 7 percent smaller for under age 5). Appendix 2B, Table 2B-1 shows the assigned site-level populations; CBSA information for each site is also shown. Based on the radial mile population association (described above) approximately 1.73 million people (0.125 million under the age of 5) are in proximity of a 2003-2005 Pb-TSP monitor included in this analysis.

2.3.2.3.5 Statistical Metrics

Four basic statistics were computed for the 2003-2005 Pb-TSP concentration data: annual means, maximum quarterly means, maximum monthly means, and second maximum monthly means. These metrics were calculated at the site level. They were calculated both for the overall 3-year period (2003-2005) and for each of the three individual years (2003, 2004, and 2005). The former set of statistics (representing the overall 3-year period) were the general focus of the analysis, and unless otherwise stated, figures, maps, and text should be assumed to be that type. Note that the 3-year annual mean statistic is actually the average of the annual means for the complete years; thus it is the average of three annual means, the average of two annual means, or the only available single complete annual mean. Annual means were computed from quarterly means. The 3-year maximum quarterly mean statistic represents the highest quarterly mean of the complete ones (sites have from four to 12 complete quarters), and the 3-year maximum monthly mean represents the highest monthly mean of the complete ones (each site has from nine to 36 complete months). The 3-year second maximum monthly mean represents the highest second highest monthly mean of the complete ones. Two additional 3-year metrics were also calculated but, like the individual year statistics for the four basic metrics, utilized sparingly. These two metrics are 1) the average of the three overall highest monthly means for the 3-year period (year nonspecific), and 2) the average of the annual maximum monthly means.

Population weighted means were also calculated for the four basic metrics for various aggregation levels. The site-level means were weighted by total population. To compute the population weighted measures, 1) the mean for each site in a specific category was multiplied by its associated population (i.e., within a mile radius), 2) these products (of #1) and the associated populations were summed, and 3) the sum of the products of #1 were divided by the population sums. Theoretically, these population weighted means show the average outdoor concentration exposure for each individual within a mile of a monitoring site. That supposition, of course, assumes that concentrations reported at the monitor are uniform over the entire radial mile.

2.3.2.4 Current Concentrations

In the following subsections, analyses are presented for the different categorizations of Pb-TSP monitoring sites described above. These categories include “all Pb-TSP sites meeting screening criteria”, and the following subsets: sites in urban areas, sites in urban areas of population greater than 1 million, sites that are source-oriented, sites that are not known to be source-oriented, and sites that were previously source-oriented.

The site-level Pb-TSP concentrations for all computed statistics are shown in Appendix 2B, Tables 2B-1 and 2B-2. The distributions of sites for the four basic (3-year) statistics (annual mean, maximum quarterly mean, maximum monthly mean, and second maximum monthly

mean) and the two additional 3-year statistics (average of three overall highest monthly means and average of 3 annual maximum monthly means) are shown in Figure 2-8; the boxes depict inter-quartile ranges and medians, whiskers depict the 5^{th} and 95^{th} percentiles, and asterisks identify composite averages. Additional points on the distributions for these statistics are given in Appendix 2B, Table 2B-3. For example, the national composite average annual mean was 0.09 $\mu g/m^3$, and the corresponding median annual mean was 0.02 $\mu g/m^3$. The national composite average maximum quarterly mean was 0.17 $\mu g/m^3$ and the corresponding median maximum quarterly mean was 0.03 $\mu g/m^3$. The national composite average maximum monthly mean was 0.31 $\mu g/m^3$ and the median maximum monthly mean was 0.04 $\mu g/m^3$. The national composite average second maximum monthly mean was 0.21 μg/m3 and the median value was 0.03 $\mu g/m^3$. The national composite average of the mean of the three overall highest monthly averages was 0.31 $\mu g/m^3$ and the median value was 0.04 $\mu g/m^3$. The national composite average of the mean of the annual highest monthly means was 0.21 $\mu g/m^3$ and the median value was 0.03 $\mu g/m^3$.

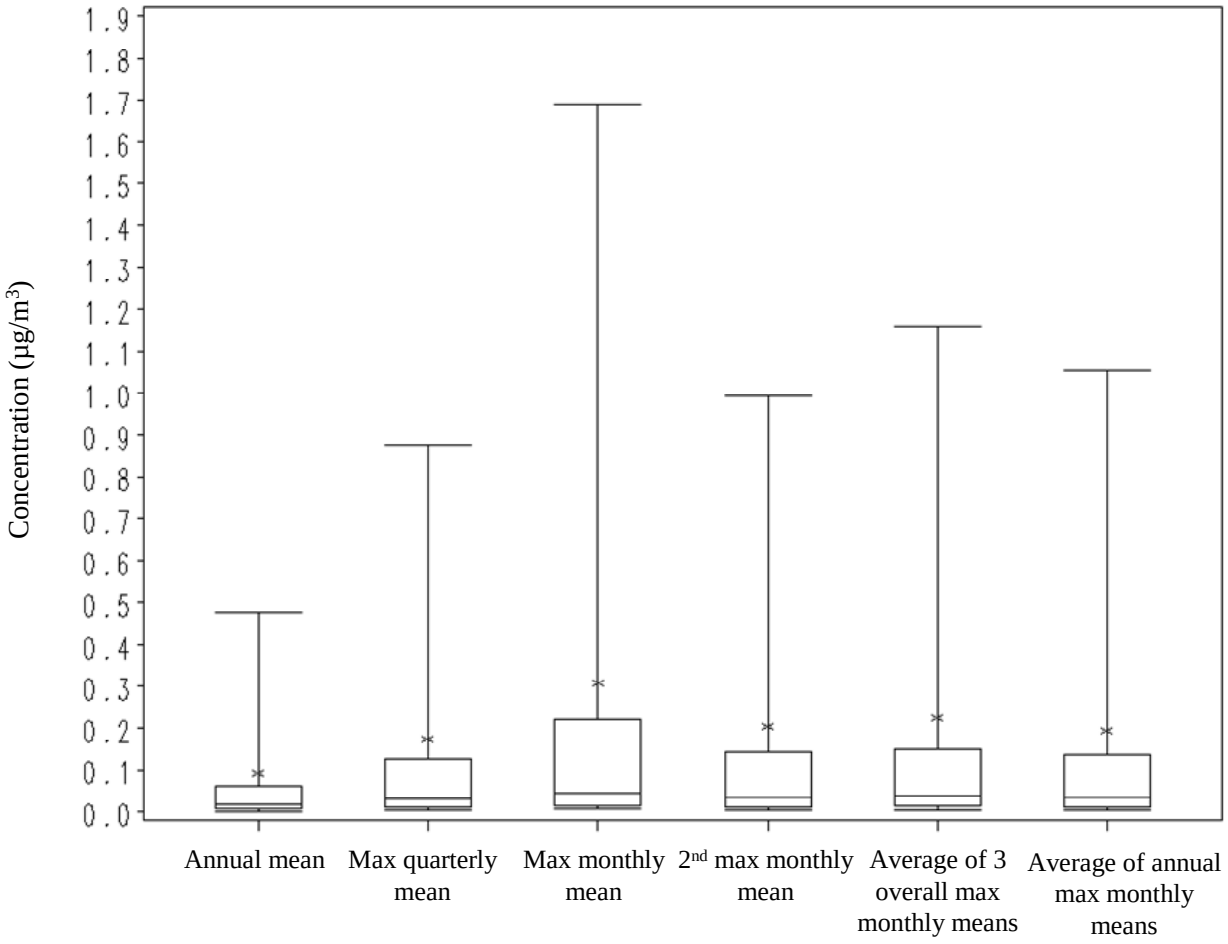


Figure 2-8. Distribution of Pb-TSP concentrations (represented by 6 different statistics) at the 189 Pb-TSP monitoring sites, 2003-2005.

Figure 2-9 shows cumulative percentages of total monitored populations associated with each of the four Pb metrics for various levels [> 0.02 µg/m^3, > 0.05 µg/m^3, > 0.20 µg/m^3, > 0.50 µg/m^3, and > 1.54 µg/m^3]. Note that site statistics were rounded to 2 decimal places before comparing to stated levels. The phrase "monitored populations" refers to the number of people residing in proximity to these 189 monitors as described in Section 2.3.2.3.4. The site-level values for the four statistical metrics (annual average, maximum quarterly mean, maximum quarterly mean, and second maximum monthly mean) are mapped in Figures 2-19 through 2-13. As seen when comparing these figures, the geographic locations of the high (and low) concentration values for all three metrics are generally the same. In fact, there are significant correlations among all four 3-year (2003-2005) summary metrics; see Appendix 2B, Table 2B-4.

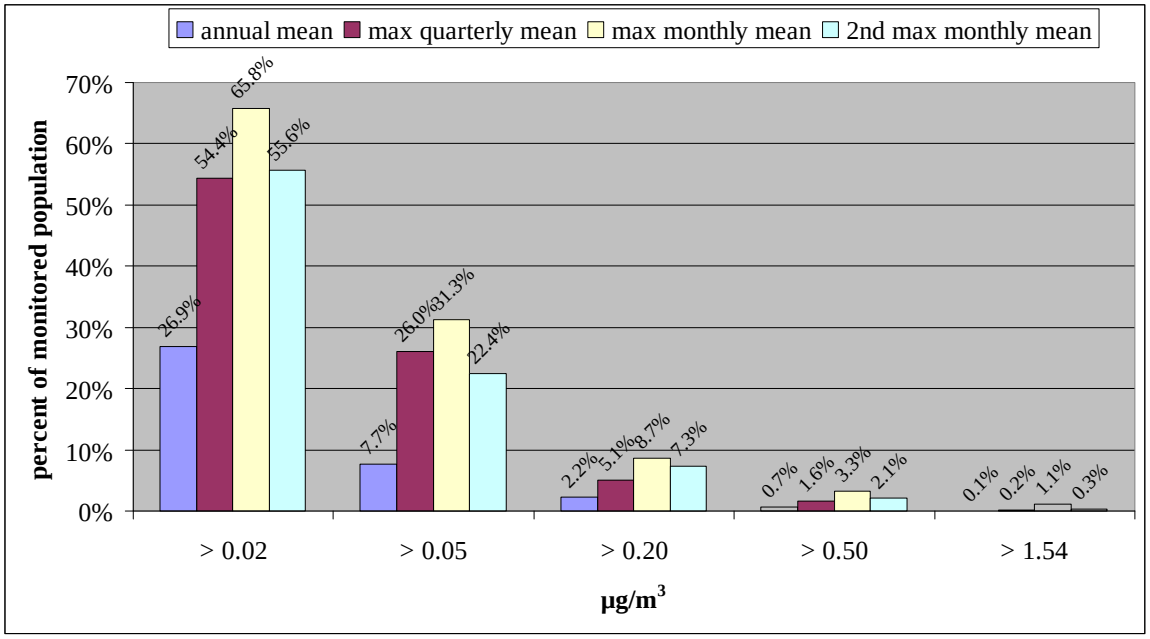


Figure 2-9. Percentages of Pb-TSP monitored populations residing in areas exceeding various concentrations (for 4 different statistics), 2003-2005.

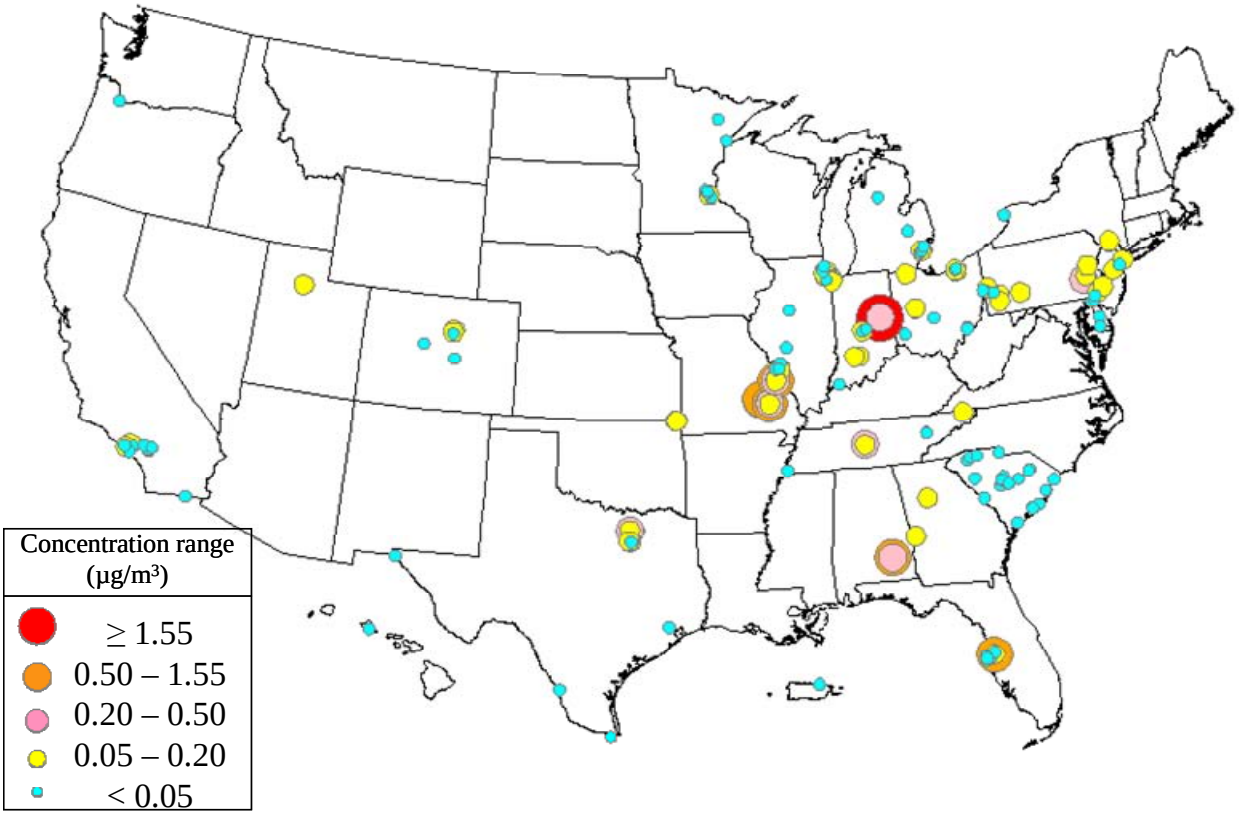


Figure 2-10. Pb-TSP annual means (for all sites), 2003-2005.

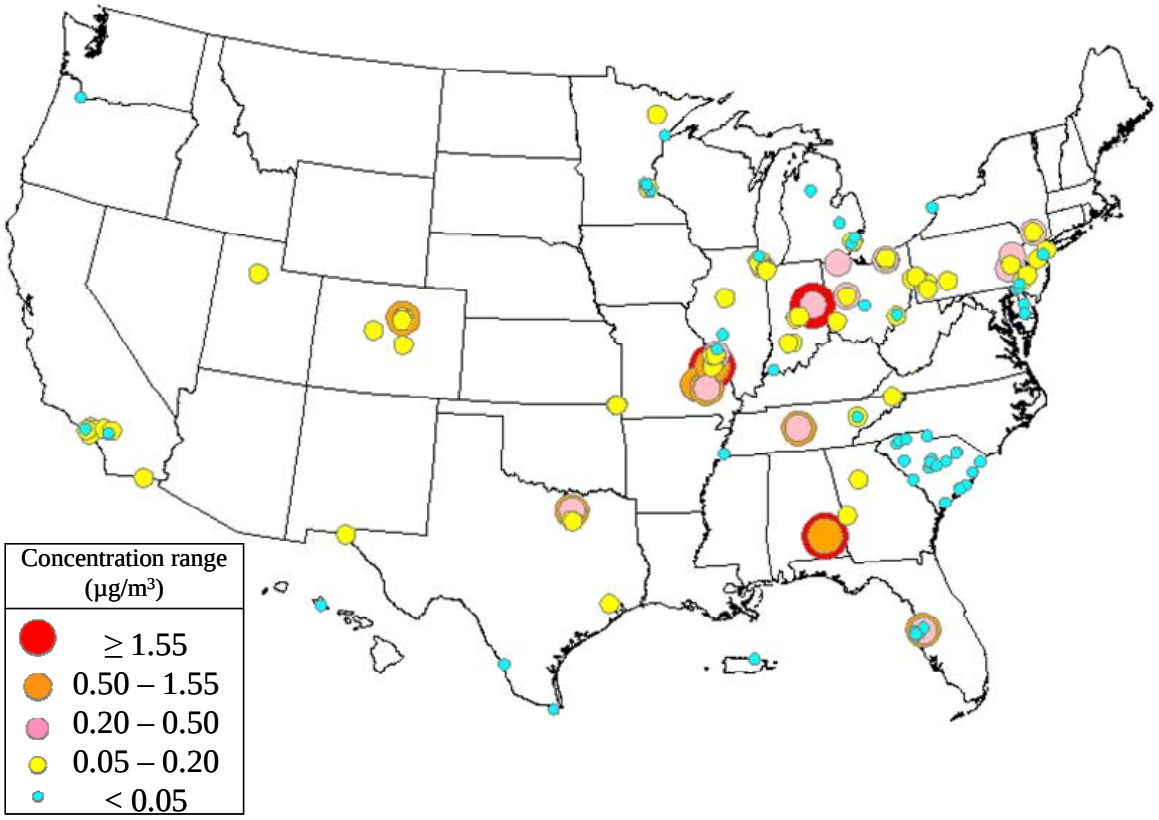


Figure 2-11. Pb-TSP maximum quarterly means (for all sites), 2003-2005.

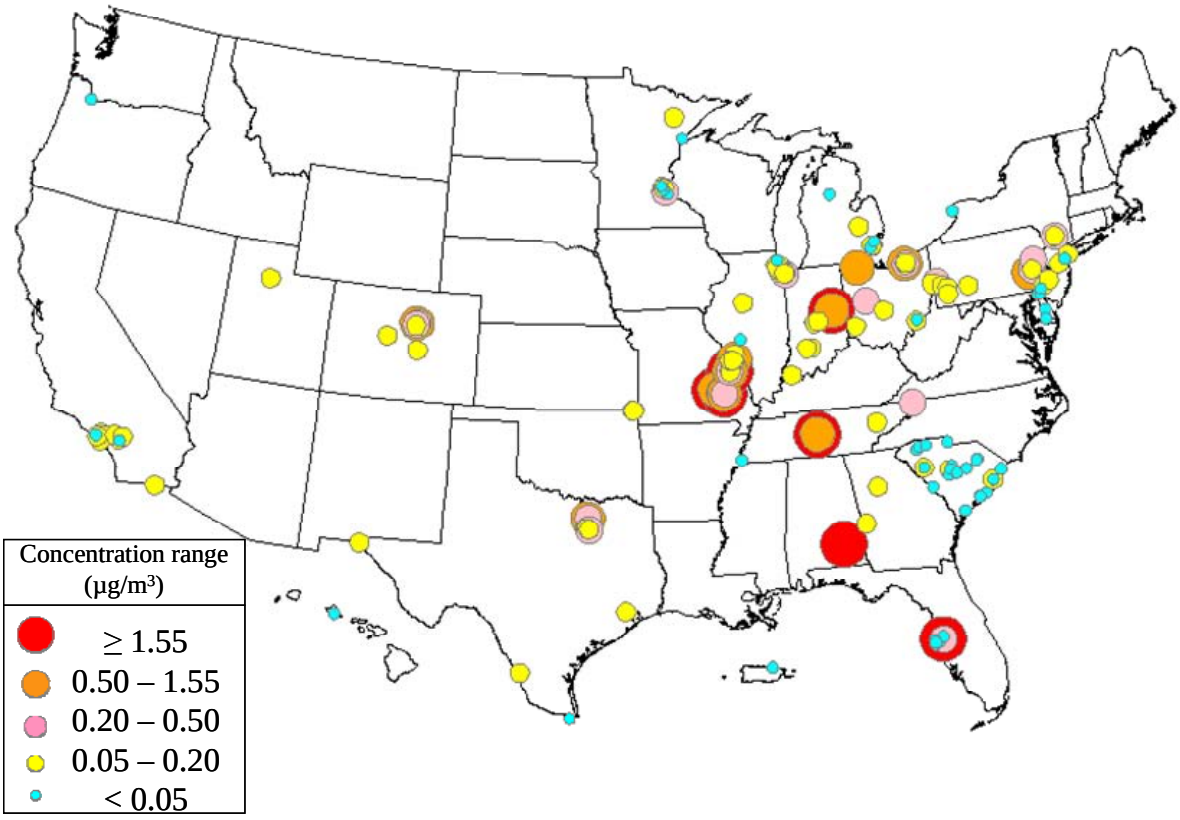


Figure 2-12. Maximum monthly Pb-TSP means (all sites), 2003-2005.

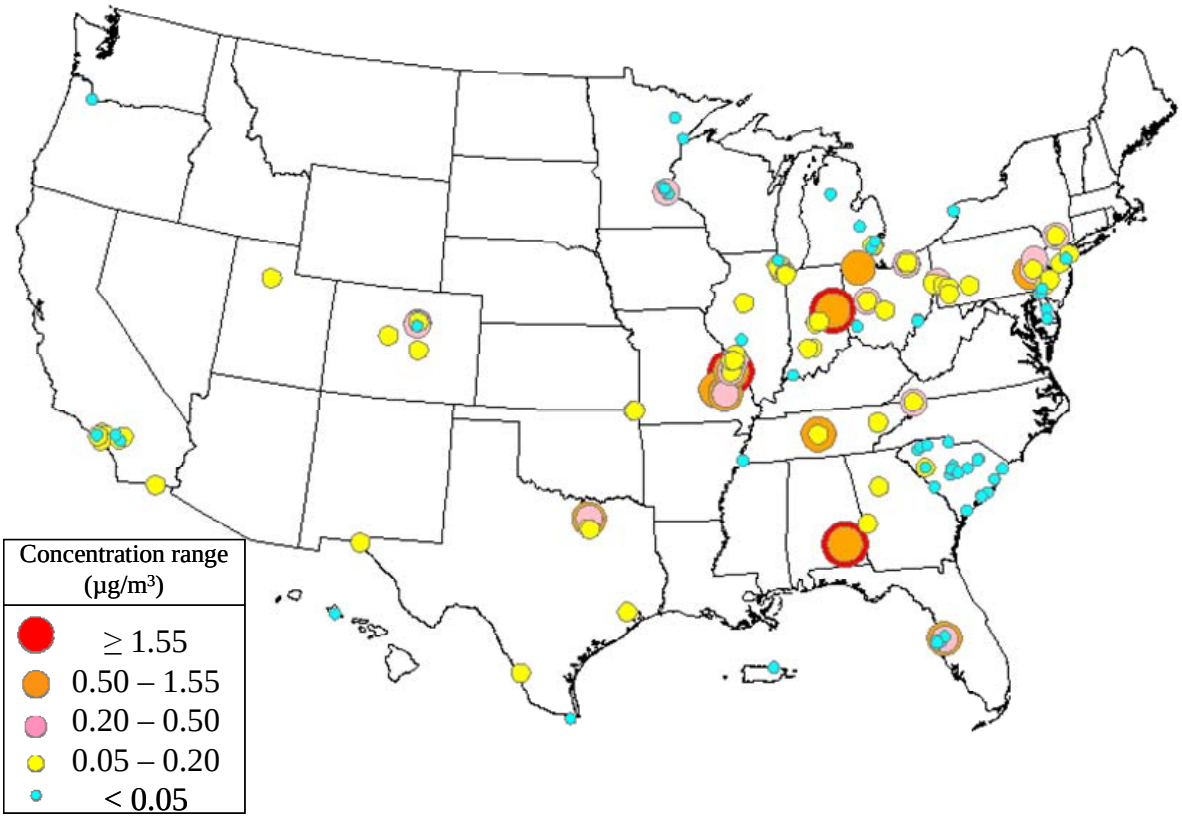


Figure 2-13. Second maximum monthly Pb-TSP means (all sites), 2003-200

The site-level ratios of 1) maximum quarterly mean to annual mean, 2) maximum monthly mean to annual mean, and second maximum monthly mean to annual mean are presented in Appendix 2B, Table 2B-5. For all TSP-Pb sites included in the analysis, the national median for the ratio of site-level maximum quarterly average to site-level annual mean was about 1.8; the national median for the ratio of site-level maximum monthly mean to site-level annual mean was about 2.8; and the national median for the ratio of site-level second maximum monthly mean to site-level annual mean was about 2.1.

2.3.2.4.1 Source-oriented Sites

As seen in the previously discussed Figure 2-8, the national ("all sites") means are substantially higher than the national medians for all four statistical metrics (annual mean, maximum quarterly mean, maximum monthly mean, and second maximum monthly mean). This is due to a small number of monitors with significantly higher levels. These monitors with higher concentrations are almost exclusively associated with industrial point sources.

Eliminating the source-oriented monitors from the national aggregations lowers most of the corresponding distribution statistics and makes the means more comparable to the medians.

The distributions of the site-level metrics for the source-oriented sites, the non-source-oriented sites, and the "previous" source-oriented sites, are presented in Figures 2-14, 2-15, and 2-16, respectively. For comparison purposes, Figures 2-17 through 2-20 present the categorical data distributions for each of the four statistical metrics on the same scales. In all of these figures, the boxes depict inter-quartile ranges and medians, whiskers depict the 5^{th} and 95^{th} percentiles, and asterisks identify composite averages. Additional points on the distributions of these statistical metrics for these three categories of monitoring sites are given in Appendix 2B, Table 2B-3. The medians, means, and population-weighted means of the site-level values of the three statistical metrics are presented in Figure 2-21 for the source-oriented and other groupings of monitoring sites.

Per Figure 2-18, the median maximum quarterly mean for source-oriented sites (0.25 $\mu g/m^3$) is about 14 times greater than the same statistic for non-source-oriented sites (0.02 $\mu g/m^3$); in fact, the median (50^{th} percentile) maximum quarterly mean for non-source-oriented sites is approximately the same value as the 5^{th} percentile for source-oriented sites. Almost 95 percent of all monitors identified as being source-oriented had a maximum quarterly average of 0.02 $\mu g/m^3$ or more, and over 25 percent had maximum quarterly average of 0.50 $\mu g/m^3$ or more.

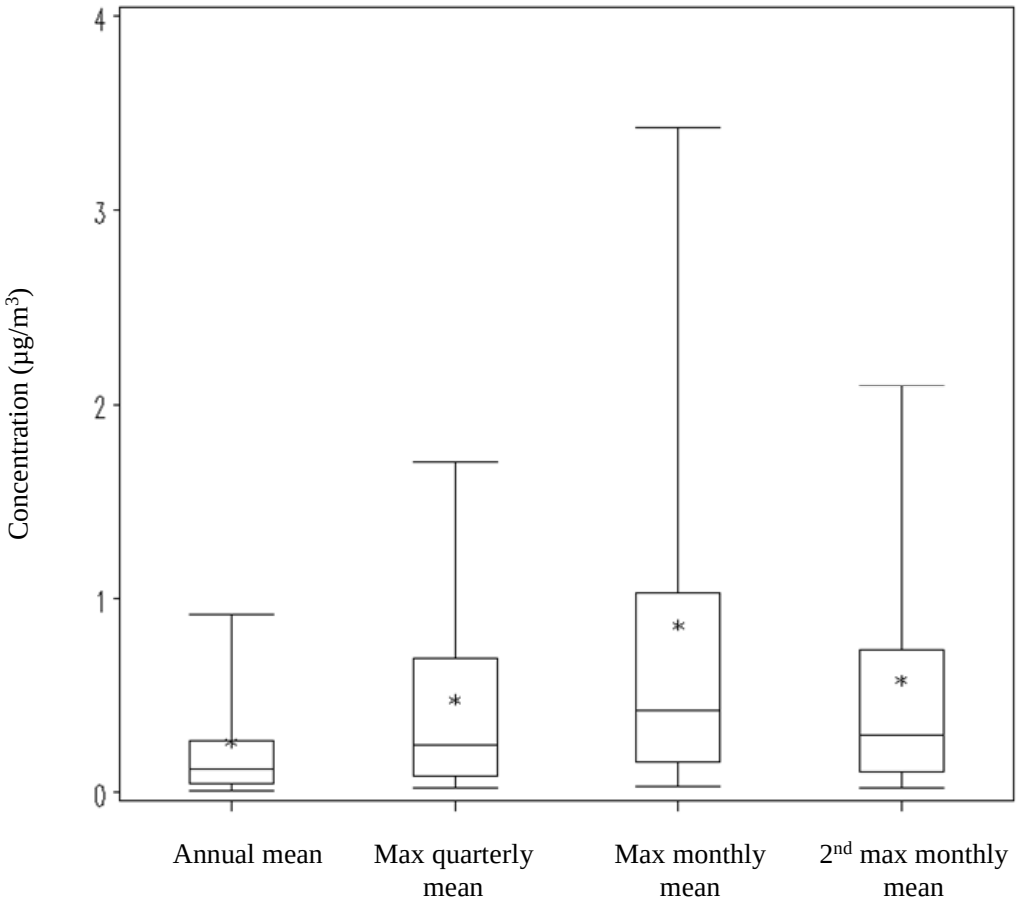


Figure 2-14. Distribution of Pb-TSP concentrations (represented by 4 different statistics) at the source-oriented monitoring sites, 2003-2005.

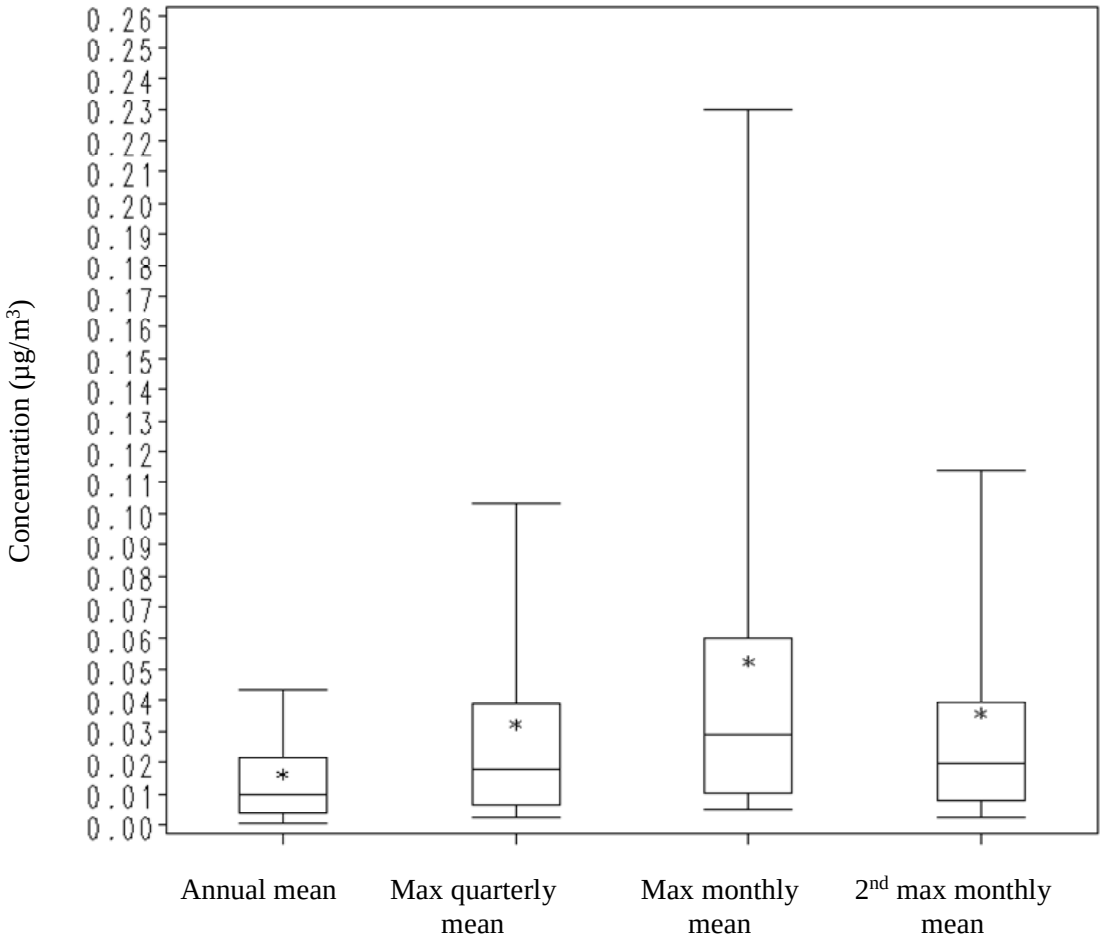


Figure 2-15. Distribution of Pb-TSP concentrations (represented by 4 different statistics) at the non-source-oriented monitoring sites, 2003-2005.

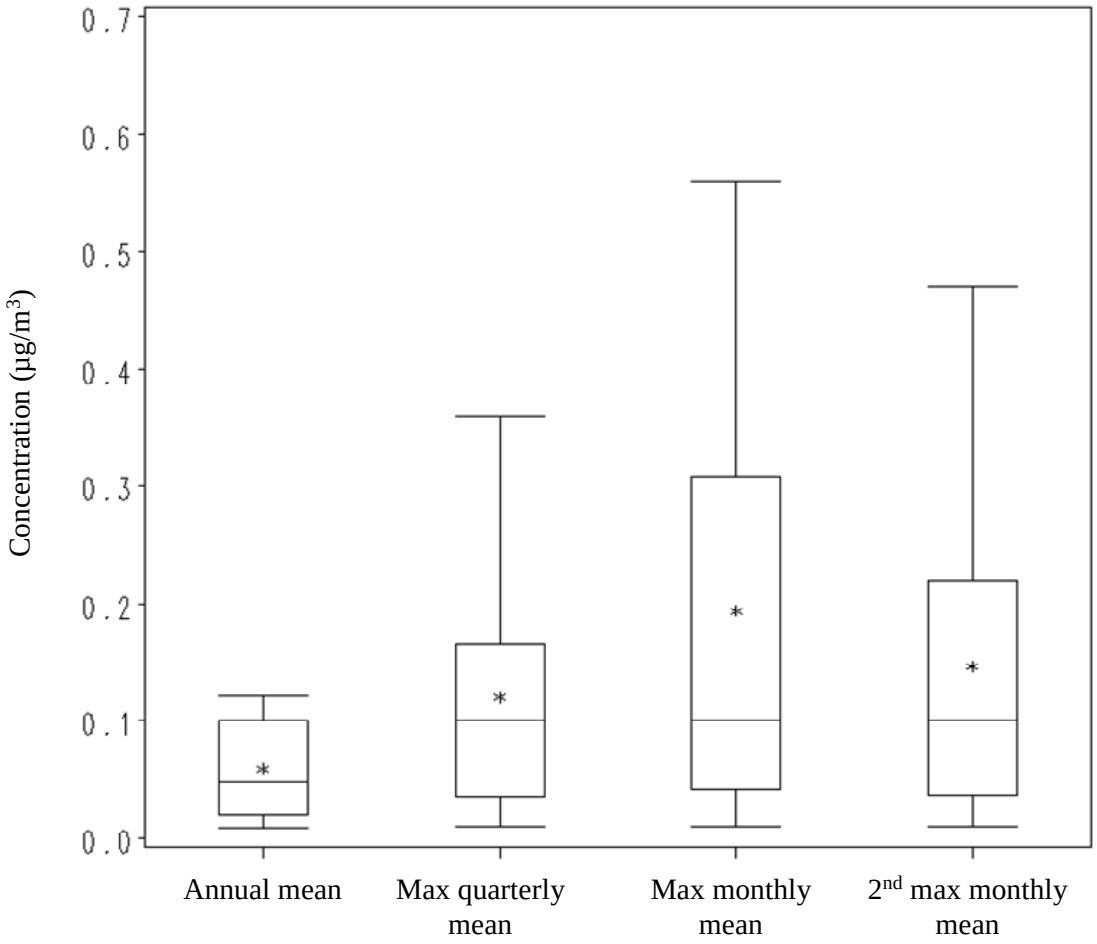


Figure 2-16. Distribution of Pb-TSP concentrations (represented by 4 different statistics) at the nine monitoring sites near previous large emission sources, 2003-2005.

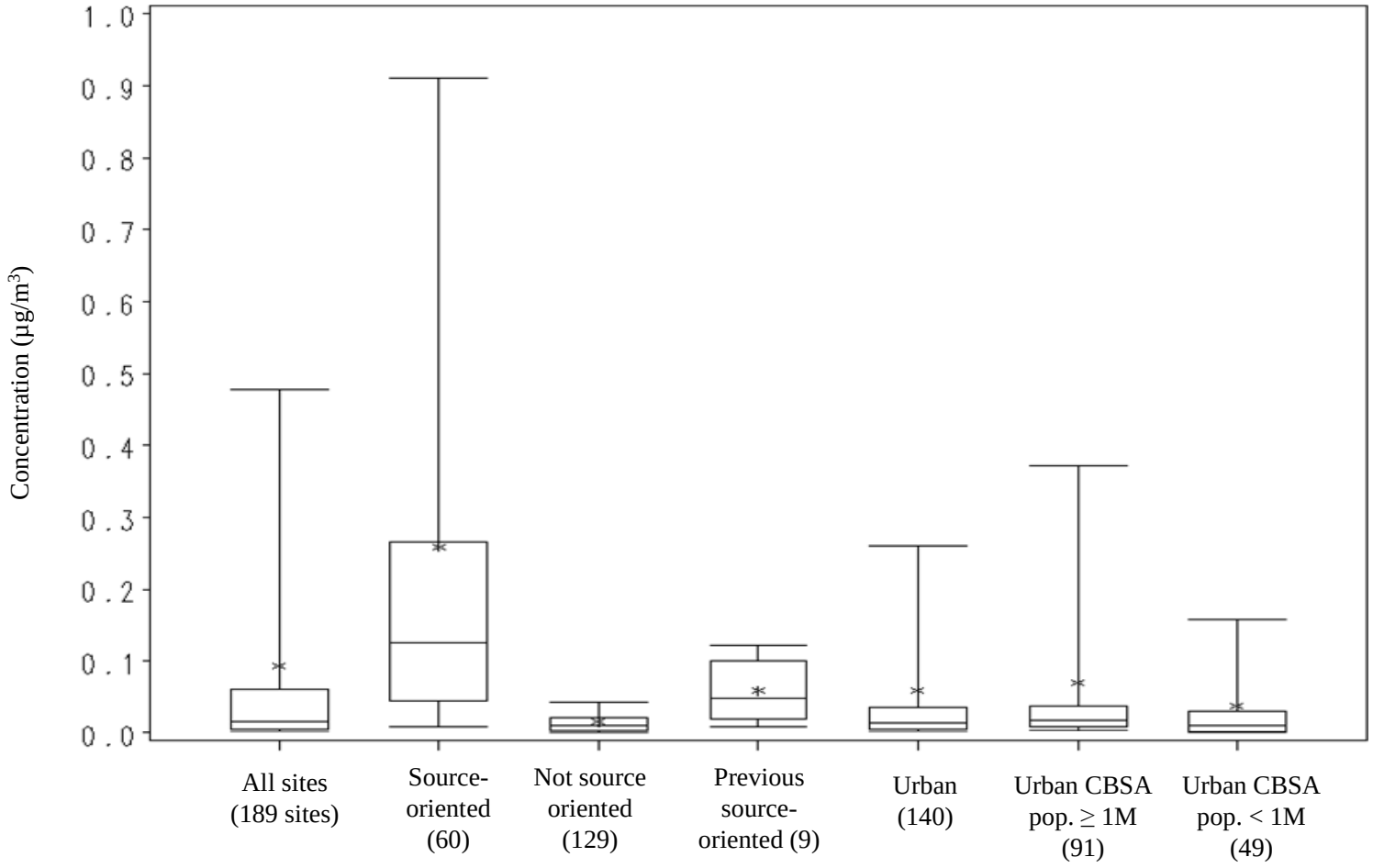


Figure 2-17. Distribution of Pb-TSP annual mean concentrations at different categories of sites, 2003-2005.

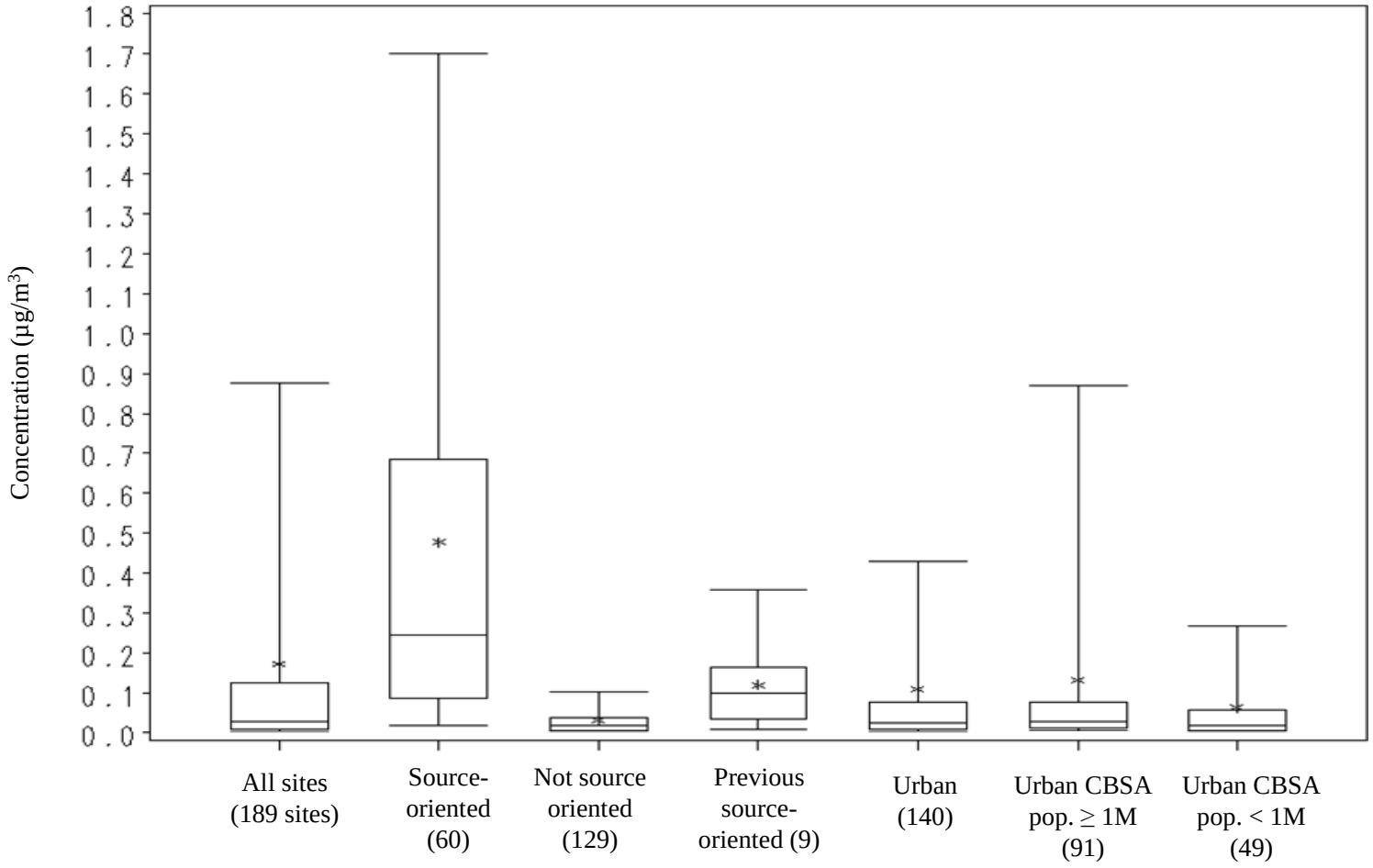


Figure 2-18. Distribution of Pb-TSP maximum quarterly mean concentrations at different categories of sites, 2003-2005.

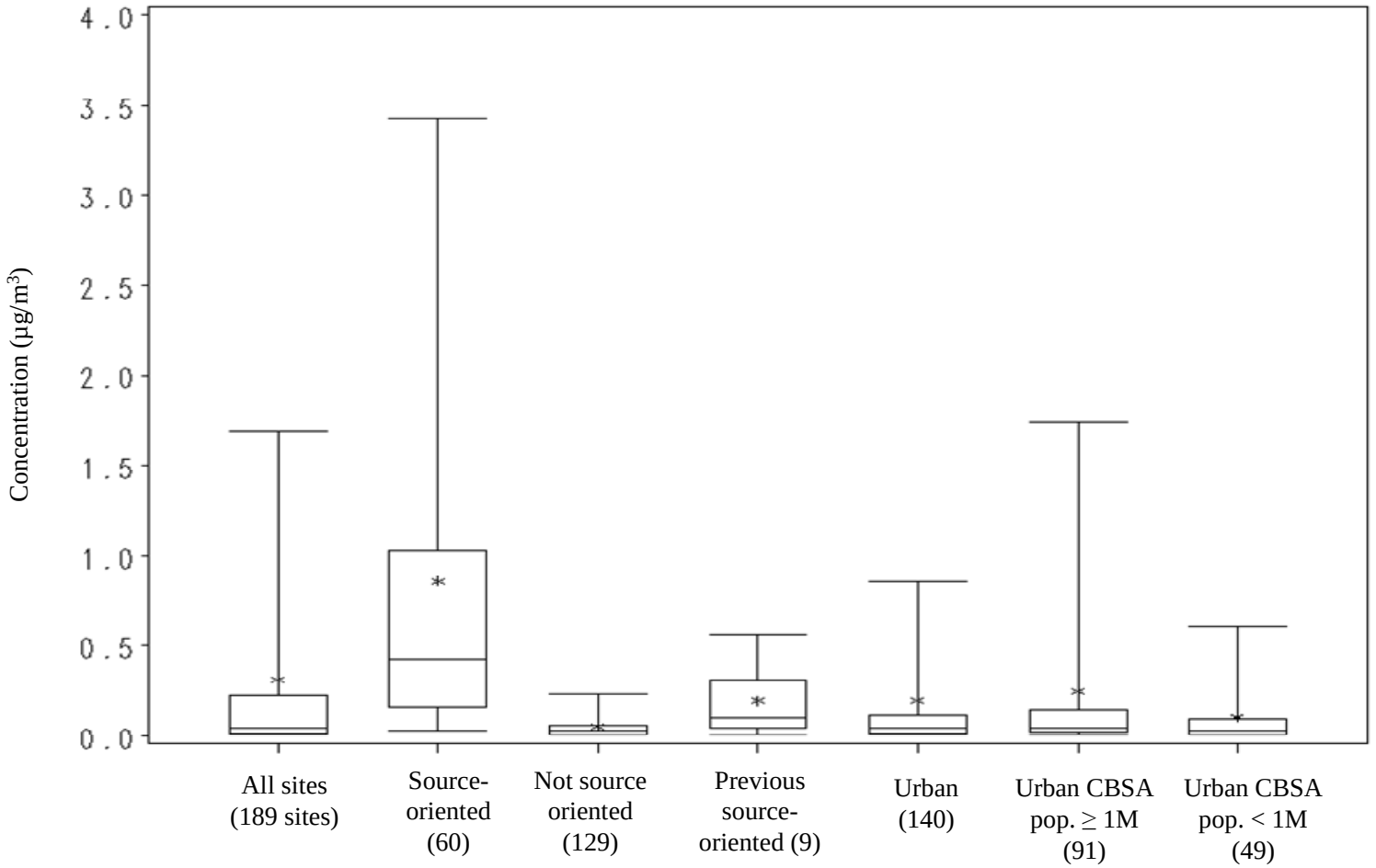


Figure 2-19. Distribution of Pb-TSP maximum monthly mean concentrations at different categories of sites, 2003-2005.

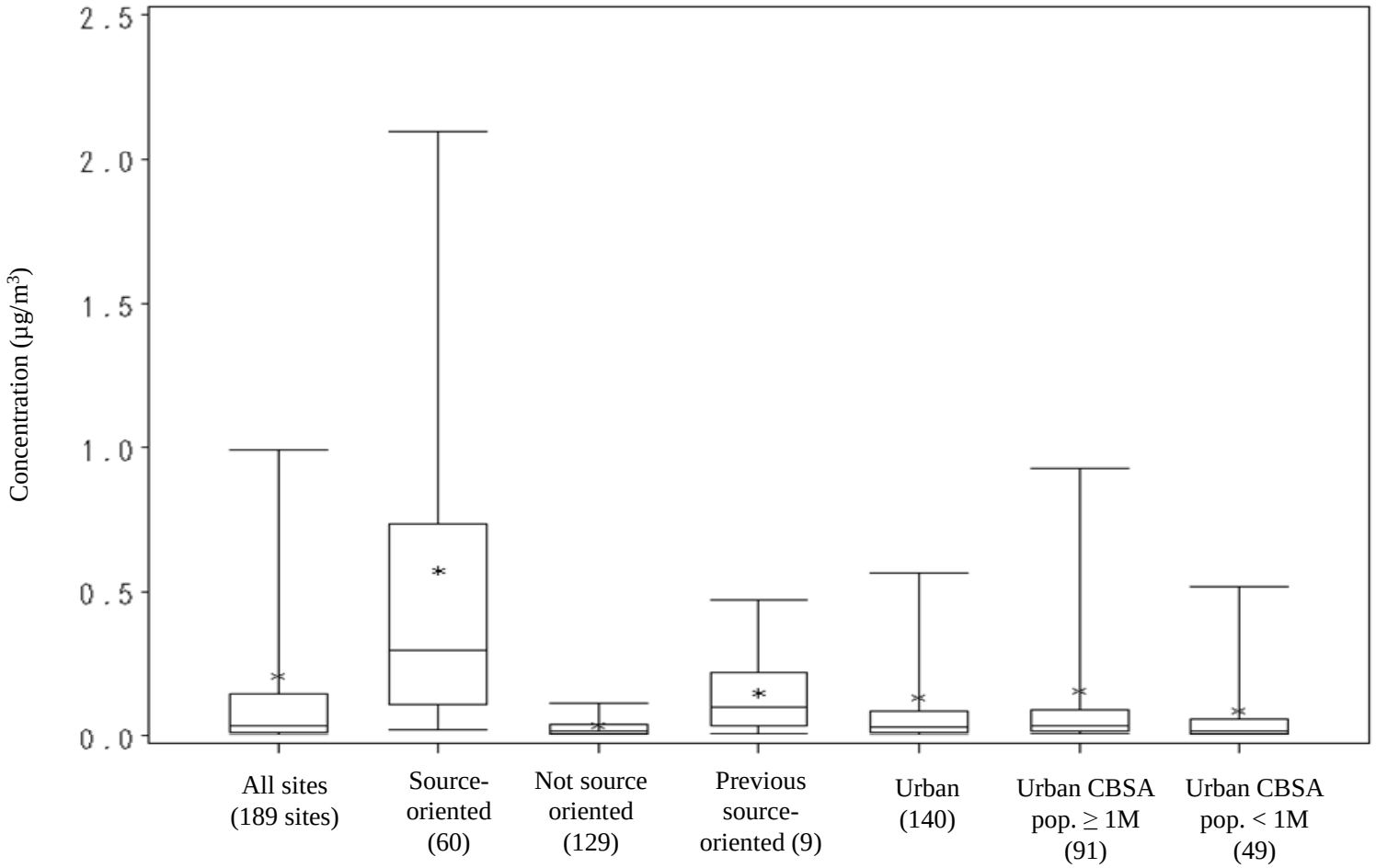


Figure 2-20. Distribution of Pb-TSP second maximum monthly mean concentrations at different categories of sites, 2003-2005.

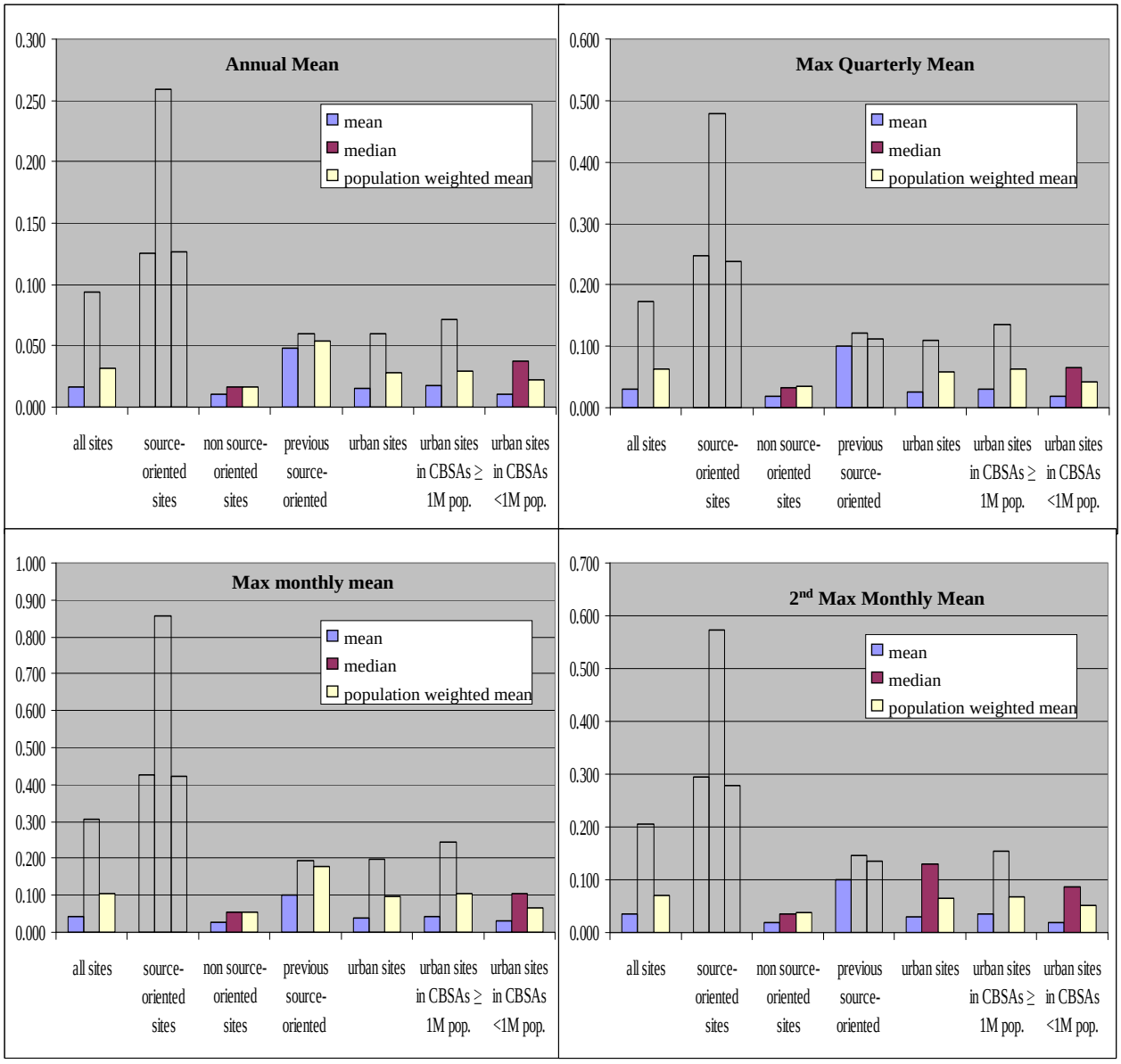


Figure 2-21. Medians, means, and population-weighted means for 4 site-level statistics. (All y-axes are in concentration units of µg/m^3).

Although 60 Pb-TSP monitoring sites met the source oriented classification criteria, that number does not correspond to the number of represented or "covered" sources of significant emissions. Recall that the emissions sliding scale was based on the aggregate emissions within one mile of the site (see Section 2.3.2.3.3). Thus, instead of having only one significant source within a specified range, a site tagged as source oriented could actually have several nearby moderate-sized emission sources and/or many nearby small sources. However, the majority of the source-oriented sites in this national analysis do have just one nearby significant emission source. Furthermore, many of these significant emission sources have multiple Pb-TSP monitors

in the vicinity. For example, the Herculaneum primary Pb smelter has 7 nearby Pb-TSP monitoring sites that are included in this national characterization (as well as others that operated during 2003-2005 but that did not meet the screening criteria). Thus, the 60 source-oriented sites really represent fewer than 60 significant emission sources. For the 60 source-oriented sites, there are only 37 unique closest emission sources (i.e., NEI site ID's). The 60 source-oriented sites are located in 29 different counties.

Although the "previous" source-oriented category contains only a limited number of sites (nine) with varied and undetermined circumstances, the distribution statistics for that category (for all three metrics) are generally much higher than the non-source-oriented levels; for example, the "previous" median maximum quarterly mean of 0.10 $\mu g/m^3$ is more than five times higher than the comparable non-source-oriented level of 0.02 $\mu g/m^3$.

2.3.2.4.2 Urban Sites

The distributions of the site-level values for the four statistical metrics for the set of 140 sites classified as "urban" are presented in Figure 2-22. The distributions for the subset of sites (n = 91) located in a CBSA with one million or more population are presented in Figure 2-23, and for the subset of sites (n=49) located in a CBSA with less than a million population, in Figure 2-24. In these figures, the boxes depict inter-quartile ranges and medians, whiskers depict the 5^{th} and 95^{th} percentiles, and asterisks identify composite averages. Additional points on the distributions for these statistics for these three groupings of monitoring sites are given in Appendix 2B, Table 2B-3.

Previously mentioned Figures 2-17 through 2-20 plot on uniform scales the four statistical metrics for these three categories of urban sites. The median and mean values for all three concentration metrics are lower for sites in less populated CBSA's than they are for sites in high population CBSA's. Figure 2-25 shows cumulative percentages of urban monitored populations ("total) associated with each of the three Pb metrics for various concentration ranges [> 0.02 $\mu g/m^3$, > 0.05 $\mu g/m^3$, > 0.20 $\mu g/m^3$, > 0.50 $\mu g/m^3$, and > 1.54 $\mu g/m^3$]. The phrase "monitored populations" refers to the number of people residing in proximity to monitors as described in Section 2.3.2.3.4. Figure 2-25, for urban monitored populations, resembles Figure 2-9 (for all monitored populations) because the large majority of the monitored population resides in urban areas.

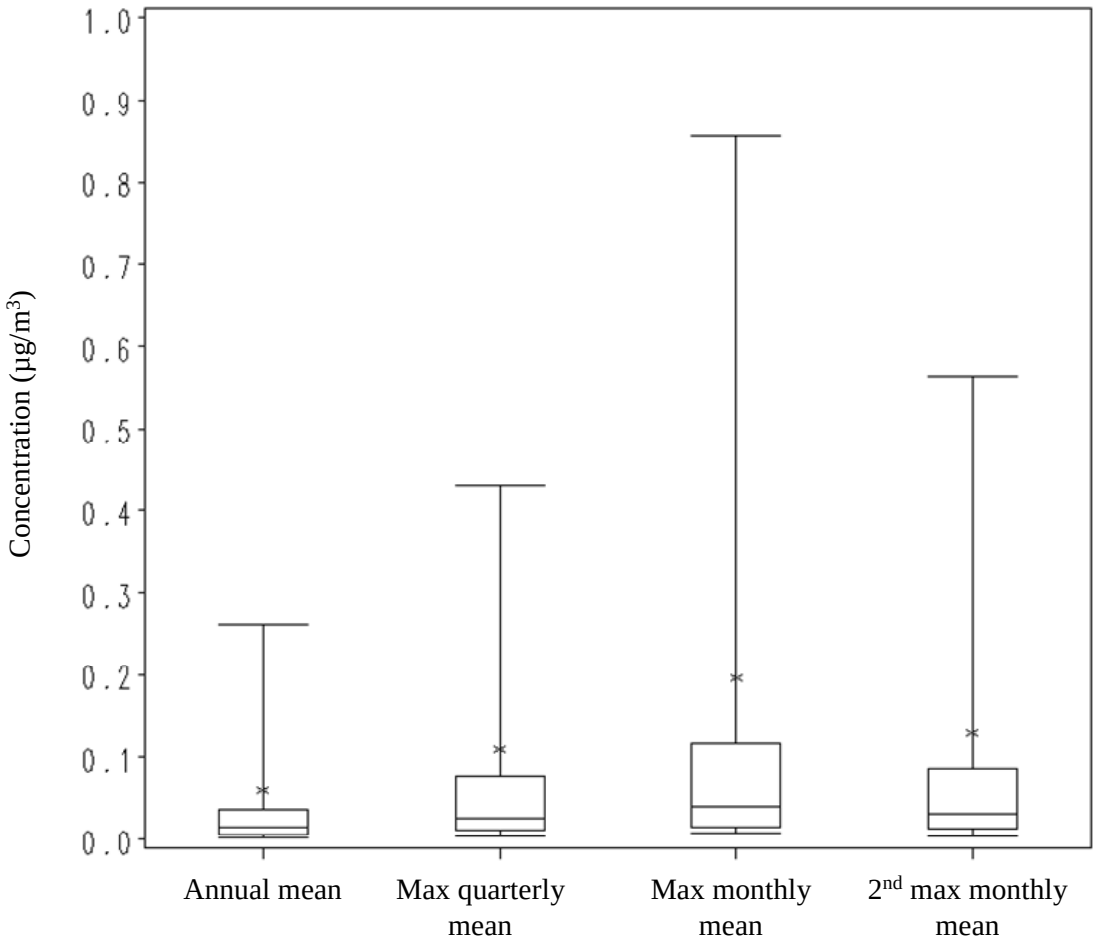


Figure 2-22. Distribution of Pb-TSP concentrations (represented by 4 different statistics) at the 140 urban monitoring sites, 2003-2005.

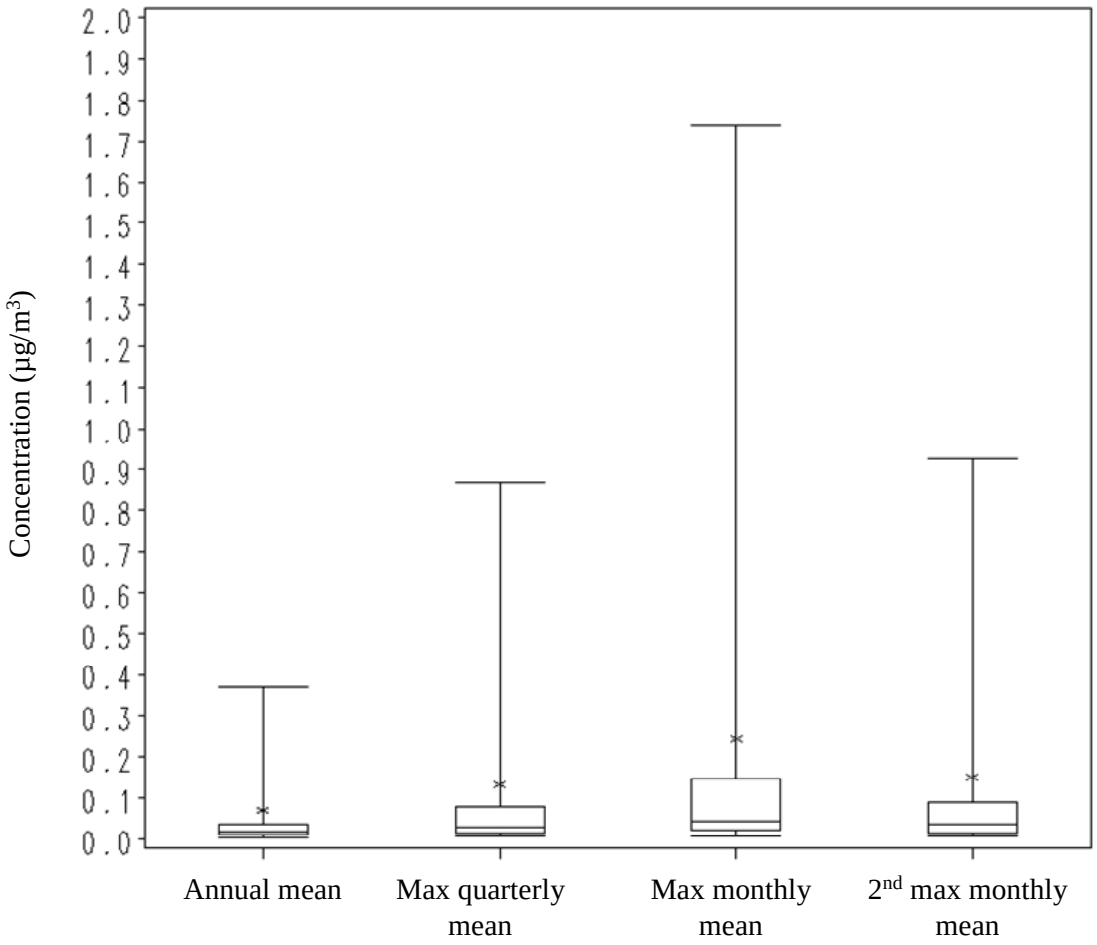


Figure 2-23. Distribution of Pb-TSP concentrations (represented by 4 different statistics) at the 91 urban monitoring sites located in metropolitan areas (CBSAs) with 1 million or more population, 2003-2005.

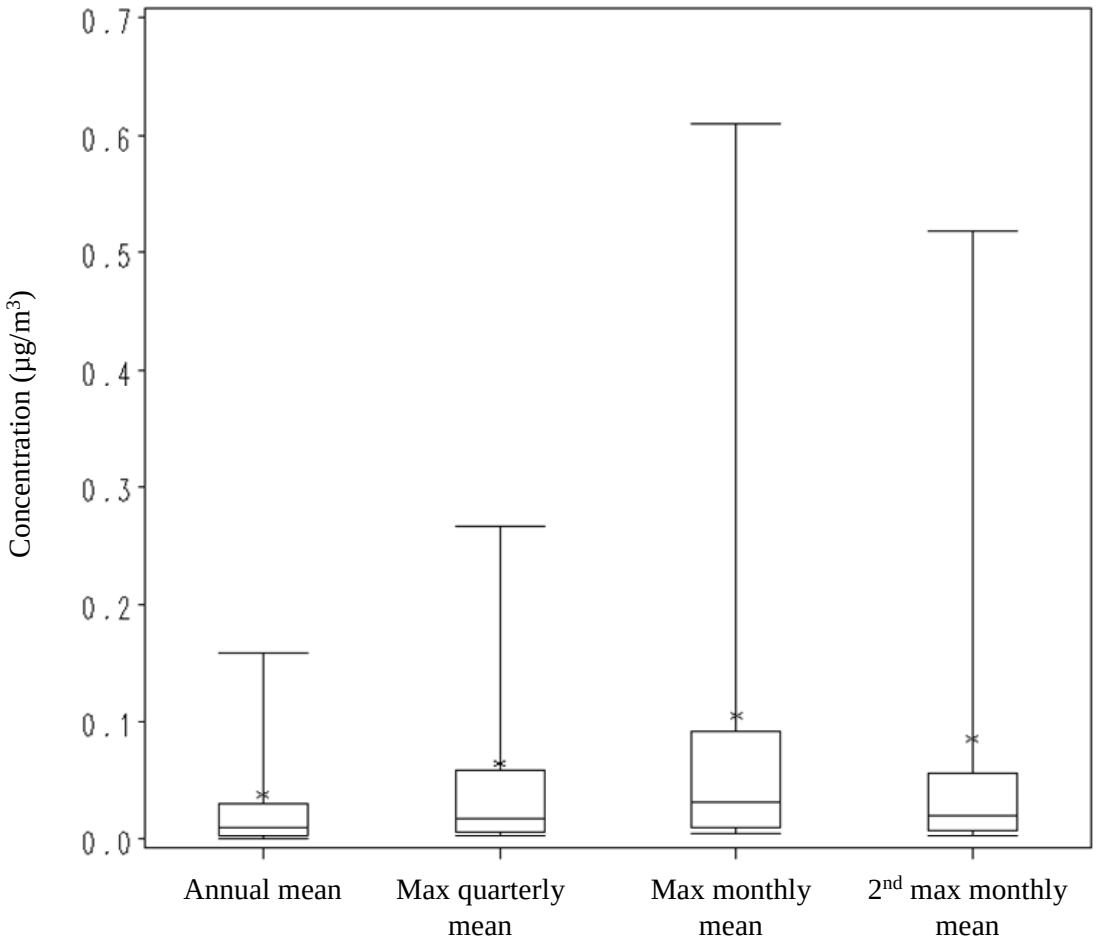


Figure 2-24. Distribution of Pb-TSP concentrations (represented by 4 different statistics) at the 49 urban monitoring sites located in CBSA's with less than 1 million population, 2003-2005.

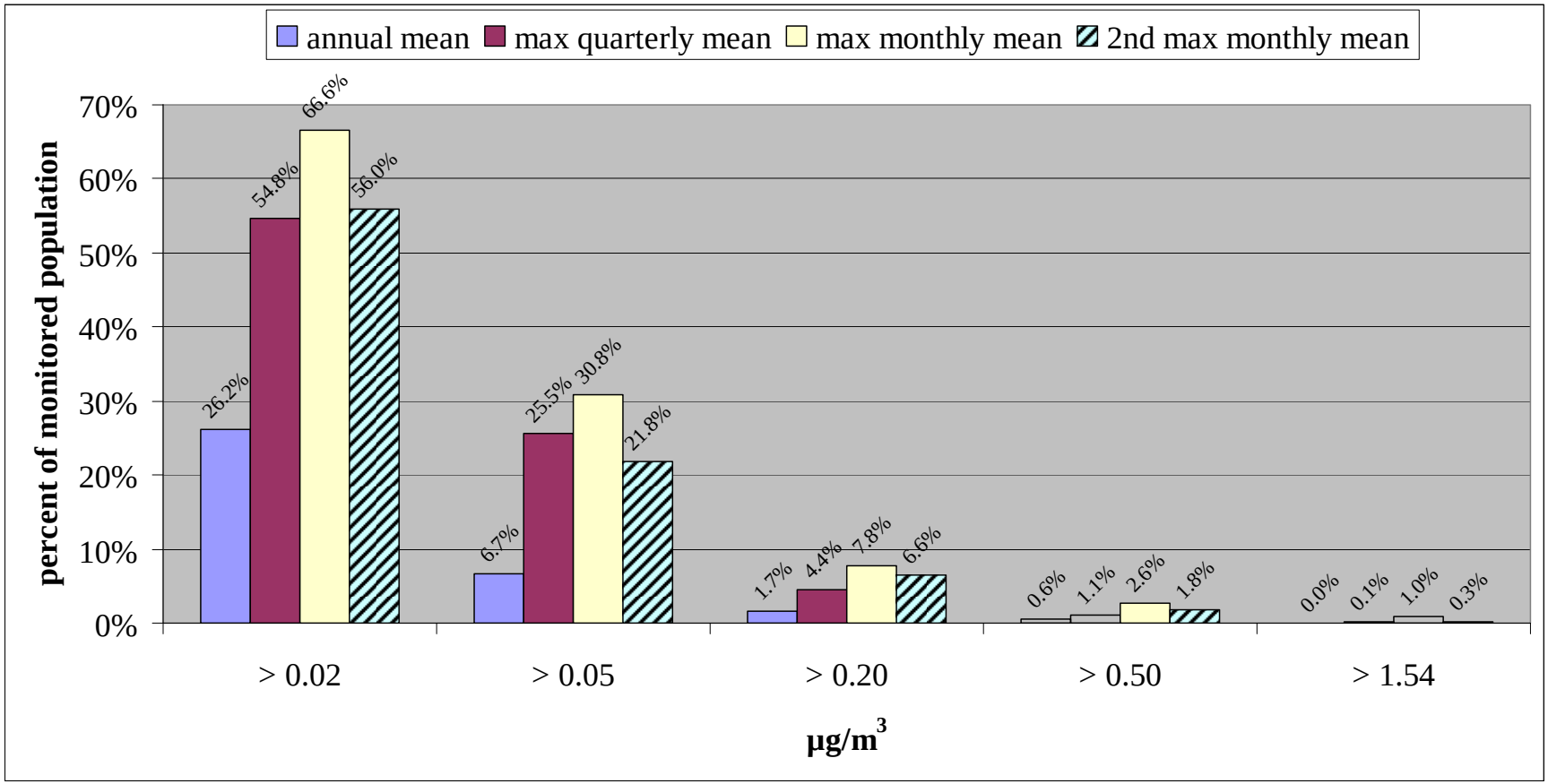


Figure 2-25. Percentages of Pb-TSP urban monitored populations residing in areas (represented by 4 different statistics) exceeding various levels. (Note: Site statistics were rounded to 2 decimal places before comparing to stated levels.)

2.3.2.5 Variability

Some seasonal variability related to meteorology is common for air Pb concentrations. The extent to which seasonal variability associated with meteorological conditions is present for a given area depends on precipitation trends, wind direction patterns, and mixing height fluctuations. For monitors situated near Pb point sources, aspects related to the facilities' operations also contribute to temporal variability. These same factors, weather and emissions (location and magnitude), also contribute to spatial variability, as do other features such as topography. Many monitors that are not source oriented exhibit no discernable seasonal pattern because their concentration levels are so low; over 57 percent of the 129 non-source-oriented sites had over 50 percent of their 2003-2005 raw concentration data at levels equal to or less than the associated MDL. Note that some data reporting agencies erroneously submit substituted values (e.g., zero or half the minimum detection limit) for data less than or equal the MDL instead of the requested actual quantifiable value.

Temporal variability in Pb-TSP concentrations, especially near source locations, is better characterized by short-term averaging times (e.g., monthly) than longer-term averaging times (e.g., yearly or quarterly). This is demonstrated in Tables 2-6 and 2-7. These tables show the number of TSP monitors, in the "all sites" database and the urban site subset, that exceeded various concentration levels between 0.02 and 1.5 $\mu g/m^3$ with averaging times or forms of maximum quarterly, maximum monthly, and second maximum monthly For example, with a stated level equal to the current standard of 1.5 $\mu g/m^3$ (actually 1.54 $\mu g/m^3$ per rounding protocol) and a 3-year evaluation window (i.e., the first table subset), 3 sites in 3 counties (1 urban site) exceeded on a quarterly averaging basis and 11 sites in 6 counties (5 urban sites in 2 counties) exceeded on a maximum monthly basis. At the lowest level examined, 0.02 $\mu g/m^3$, 107 sites in 47 counties (75 urban sites in 39 counties) exceeded that level on a maximum quarterly average basis and 127 sites in 59 counties (94 urban sites in 50 counties) exceeded that level on a maximum monthly average basis.

The four additional table subsets in Tables 2-6 and 2-7 are results for 1-year evaluations; each of the three component years (2003, 2004, 2005) are shown individually plus an average of the three years is provided. Almost all of the 3-years results are greater than the 1-year results (mathematically they could not be less), indicating year-to-year variability. For example, at the 1.55 $\mu g/m^3$ concentration level with a maximum quarterly average statistic, 3 sites exceeded using the 3-year window but only one site per year (actual and on average) exceeded with the 1-year window. At the 0.02 $\mu g/m^3$ concentration level and same statistic, 107 sites exceeded using the 3-year window but only 84 sites on average exceeded with a 1-year window.

Table 2-6. Comparison of number of sites that exceed various Pb-TSP levels using different averaging times or forms, 2003-2005

3-year statistics, 2003-2005												
Level	Maximum Quarterly Mean				Maximum Monthly Mean				2nd Maximum Monthly Mean			
	All Sites (189 in 86 counties) *		Urban Sites (140 in 73 counties) *		All Sites (189 in 86 counties) *		Urban Sites (140 in 73 counties) *		All Sites (189 in 86 counties) *		Urban Sites (140 in 73 counties) *	
	Sites	Counties	Sites	Counties	Sites	Counties	Sites	Counties	Sites	Counties	Sites	Counties
0.02	107	47	75	39	127	59	94	50	110	50	78	42
0.05	74	35	47	29	85	40	55	33	71	34	44	28
0.10	49	21	26	15	64	31	38	25	54	24	28	17
0.20	35	15	17	10	49	21	26	16	42	19	22	13
0.30	25	11	10	6	37	16	17	11	29	12	12	7
0.40	19	8	7	3	30	12	14	8	26	11	11	6
0.50	18	8	6	2	27	12	11	7	20	9	9	5
0.60	17	7	6	2	26	11	10	6	16	7	6	2
0.70	13	6	5	2	22	10	9	5	15	7	5	2
0.80	11	6	5	2	19	9	7	3	15	7	5	2
0.90	9	6	4	2	19	9	7	3	13	5	5	2
1.00	7	5	4	2	15	7	6	2	9	5	4	2
1.54	3	3	1	1	11	6	5	2	4	3	2	1
1-year statistics, 2003												
Level	Maximum Quarterly Mean				Maximum Monthly Mean				2nd Maximum Monthly Mean			
	All Sites (175 in 83 counties) *		Urban Sites (131 in 70 counties) *		All Sites (177 in 83 counties) *		Urban Sites (133 in 71 counties) *		All Sites (176 in 83 counties) *		Urban Sites (132 in 71 counties) *	
	Sites	Counties	Sites	Counties	Sites	Counties	Sites	Counties	Sites	Counties	Sites	Counties
0.02	90	41	63	35	103	46	76	40	89	42	62	36
0.05	60	27	35	21	73	34	48	28	56	26	31	20
0.10	39	15	19	9	50	23	28	17	40	15	19	9
0.20	28	12	12	7	36	15	17	10	29	11	13	8
0.30	20	10	7	5	29	12	13	8	24	9	9	5
0.40	15	7	4	2	25	11	11	6	20	9	7	4
0.50	15	7	4	2	20	10	7	5	15	7	4	2
0.60	12	7	2	2	18	8	5	3	14	7	4	2
0.70	8	6	2	2	16	8	5	3	12	6	3	2
0.80	6	5	1	1	15	8	4	3	9	5	1	1
0.90	4	3	1	1	14	7	4	3	7	4	1	1
1.00	2	2	1	1	11	6	3	2	4	3	1	1
1.54	1	1	0	0	5	4	1	1	2	2	1	1
1-year statistics, 2004												
Level	Maximum Quarterly Mean				Maximum Monthly Mean				2nd Maximum Monthly Mean			
	All Sites (166 in 77 counties) *		Urban Sites (127 in 68 counties) *		All Sites (169 in 77 counties) *		Urban Sites (130 in 68 counties) *		All Sites (168 in 77 counties) *		Urban Sites (129 in 68 counties) *	
	Sites	Counties	Sites	Counties	Sites	Counties	Sites	Counties	Sites	Counties	Sites	Counties
0.02	82	39	56	32	100	50	73	42	90	44	64	37
0.05	53	26	31	20	63	30	39	24	54	26	32	20
0.10	37	18	22	12	46	23	27	17	37	17	21	11
0.20	21	11	12	6	30	16	18	10	22	10	13	6
0.30	14	7	8	4	21	11	11	6	14	7	8	4
0.40	13	6	7	3	15	8	8	4	14	7	8	4
0.50	12	6	6	2	15	8	8	4	12	6	6	2
0.60	11	5	6	2	14	7	7	3	12	6	6	2
0.70	10	5	5	2	13	6	7	3	10	5	5	2
0.80	8	4	5	2	11	5	6	2	10	5	5	2
0.90	6	4	4	2	11	5	6	2	10	5	5	2
1.00	6	4	4	2	10	5	6	2	6	4	4	2
1.54	1	1	0	0	7	4	5	2	3	2	2	1

Table 2-7. Comparison of number of sites that exceed various Pb-TSP levels using different averaging times or forms, 2003-2005 – continued.

1-year statistics, 2005												
Level	Maximum Quarterly Mean				Maximum Monthly Mean				2nd Maximum Monthly Mean			
	All Sites (157 in 76 counties) *		Urban Sites (122 in 67 counties) *		All Sites (157 in 76 counties) *		Urban Sites (122 in 67 counties) *		All Sites (157 in 76 counties) *		Urban Sites (122 in 67 counties) *	
	Sites	Counties	Sites	Counties	Sites	Counties	Sites	Counties	Sites	Counties	Sites	Counties
0.02	80	39	57	33	93	45	70	39	83	42	60	36
0.05	52	27	35	23	60	31	40	26	53	27	36	22
0.10	35	17	23	13	46	23	30	19	34	17	22	12
0.20	19	10	12	7	32	16	21	13	26	15	17	11
0.30	15	8	8	4	22	12	14	9	16	8	8	4
0.40	10	6	5	3	18	9	11	6	13	7	7	4
0.50	8	6	3	2	16	9	9	5	8	5	4	2
0.60	7	5	3	2	14	9	7	4	8	5	4	2
0.70	5	4	3	2	11	7	5	3	8	5	4	2
0.80	5	4	3	2	8	6	3	2	6	4	3	2
0.90	5	4	3	2	8	6	3	2	5	4	3	2
1.00	5	4	3	2	6	5	3	2	5	4	3	2
1.54	1	1	1	1	3	3	1	1	1	1	1	1
Average of 3-year statistics, 2003-2005												
Level	Maximum Quarterly Mean				Maximum Monthly Mean				2nd Maximum Monthly Mean			
	All Sites		Urban Sites		All Sites		Urban Sites		All Sites		Urban Sites	
	Sites	Counties	Sites	Counties	Sites	Counties	Sites	Counties	Sites	Counties	Sites	Counties
0.02	84.0	39.7	58.7	33.3	98.7	47.0	73.0	40.3	87.3	42.7	62.0	36.3
0.05	55.0	26.7	33.7	21.3	65.3	31.7	42.3	26.0	54.3	26.3	33.0	20.7
0.10	37.0	16.7	21.3	11.3	47.3	23.0	28.3	17.7	37.0	16.3	20.7	10.7
0.20	22.7	11.0	12.0	6.7	32.7	15.7	18.7	11.0	25.7	12.0	14.3	8.3
0.30	16.3	8.3	7.7	4.3	24.0	11.7	12.7	7.7	18.0	8.0	8.3	4.3
0.40	12.7	6.3	5.3	2.7	19.3	9.3	10.0	5.3	15.7	7.7	7.3	4.0
0.50	11.7	6.3	4.3	2.0	17.0	9.0	8.0	4.7	11.7	6.0	4.7	2.0
0.60	10.0	5.7	3.7	2.0	15.3	8.0	6.3	3.3	11.3	6.0	4.7	2.0
0.70	7.7	5.0	3.3	2.0	13.3	7.0	5.7	3.0	10.0	5.3	4.0	2.0
0.80	6.3	4.3	3.0	1.7	11.3	6.3	4.3	2.3	8.3	4.7	3.0	1.7
0.90	5.0	3.7	2.7	1.7	11.0	6.0	4.3	2.3	7.3	4.3	3.0	1.7
1.00	4.3	3.3	2.7	1.7	9.0	5.3	4.0	2.0	5.0	3.7	2.7	1.7
1.54	1.0	1.0	0.3	0.3	5.0	3.7	2.3	1.3	2.0	1.7	1.3	1.0

* Note that the total site counts and total county counts vary for the 3-year period and 1-periods. Only one valid ('complete') year was required for inclusion in the 3-year dataset but additional complete quarter and months (not part of the valid year(s)) were kept and considered for the max statistsics. The 1-year (max) data statistics were computed from available quarters and months with no additional completeness criteria imposed.. Thus, some sites have valid max monthly statistics but not valid max quarterly and/or max 2nd monthly statistic; because of this, the 1-year site and county counts varied slightly for the different metrics. However, most sites did have 75%+ data capture for all years represented.. [Percent of sites with 75%+ data capture for all sites: 2003=95%, 2004=91%, 20005=90%.. Oprecent of sites with 75% data capture for urban sites: 2003=95%, 2004=93%, 2005=91%.]

To further illustrate temporal variability and also spatial variability, Figure 2-26 graphs 2003-2005 Pb-TSP monthly averages for several sites in the Dallas, TX metropolitan area (CBSA). Three of the plotted sites (the ones using circle, square, and diamonds symbols) were classified as source-oriented; they are all located within one mile (generally north) of a facility that emits three tons of Pb per year. The other two sites (using the triangle symbols) are located about 25 miles south of the facility. The 3-year maximum monthly averages for the three source-oriented sites were 0.97, 0.80, and 0.48 µg/m^3; the 3-year maximum monthly averages for the two sites that were not source oriented sites were 0.23 and 0.10 µg/m^3. The 3-year maximum quarterly averages for the three source-oriented sites were 0.70, 0.35, and 0.21 µg/m^3; the 3-year quarterly averages for the two non-source-oriented sites were 0.08 and 0.06 µg/m^3. Thus, the

highest source-related values and lowest non-source- related values for those two statistical metrics varied by about a factor of ten to 12 (ten for maximum monthly average and 12 for maximum quarterly average). This situation is common to metropolitan areas with both source and nonsource sites; the ratios of highest area value to lowest area value for the maximum quarterly and maximum monthly statistics ranged from two to over 300 in the 14 CBSA's with Pb-TSP sites in both categories. The two non-source-oriented Dallas sites shown in Figure 2-26 recorded a large number of 24-hour Pb-TSP concentration values less than or equal to the corresponding minimum detection limit; these low values accounted for about 18 percent of their combined total number of values in 2003-2005. Even the high site, though, had some of these low reported values; about 3 percent of its 2003-2005 data were at or below detection limits.

Figure 2-26. Pb-TSP monthly means at five sites located in the Dallas, TX metropolitan area, 2003-2005.

The data in Figure 2-26 also illustrate some of the points made with Tables 2-6 and 2-7. For example, two sites exceed a monthly comparison level of 0.50 µg/m^3 using a 3-year

evaluation window, but only one site exceeds that level using a 1-year timeframe with year 2003 or 2004. And, although two sites exceeded a maximum monthly average level of 0.50 $\mu g/m^3$ using the 3-year timeframe only one site exceeded that level using a maximum quarterly metric with the same 3-year period. The high site (circle symbols) in the top plot provides good examples of the inconsistencies between maximum monthly averages and maximum quarterly averages. The highest concentration month for that site in 2003 was April with an average of 0.89 $\mu g/m^3$; however, the corresponding quarterly average is 0.48 $\mu g/m^3$ which is the only third highest of the year. Note that the quarter (second) with the highest monthly average (April) also contains a month with the lowest average (June). Thus, the low month offset the high month in the quarterly average and the high month was not within the high quarter. These situations are not atypical in the Pb-TSP data. In the 189 site 2003-2005 Pb-TSP database, the site level maximum month for the 3-year period occurred in the maximum quarter 66 percent of the time. All three of the largest months were synonymous with the largest quarter at only six percent of the sites.

2.3.3 Pb-PM_{10}

The NATTS network operated in 2003-2005 included 23 sites in mostly urban, but some rural, areas (Figure 2-27). These sites are operated by 21 state or local host agencies. All collect particulate matter as PM_{10} for toxic metals analysis, typically on a 1 in 6 day sampling schedule. Lead in the collected sample is generally quantified via the ICP/MS method. The standard operating procedure for metals by ICP/MS is available at: http://www.epa.gov/ttn/amtic/airtox.html. These NATTS sites are relatively new, with 2004 being the first year in which all were operating. The AQS can be accessed at http://www.epa.gov/ttn/airs/airsaqs/.

Figure 2-27. Pb-PM_{10} (NATTS) monitoring sites network.

2.3.3.1 Data Analysis Details

Lead-PM_{10} data collected in 2003-2005 (parameter code 82128, duration '7') were extracted from EPA's AQS on May 22, 2007. Most of the monitors reporting such data are in the NATTS network. The same screening criteria utilized for Pb-TSP were implemented for Pb-PM_{10} with one variation; because of the limited amount of available data, only three valid quarters were required (instead of all four) to make a valid year. Thus the criteria used were: 1) a minimum of 10 observations per quarter, 2) for at least three quarters of one calendar year, and 3) at least 9 months with 4 observations each; all three criteria had to be met for inclusion. Forty monitors met the three-part criteria. Of these 40 monitors, two were collocated with another complete monitor. Only one monitor from each collocated pair (i.e., from each site location) was kept in the analysis, specifically the one with highest maximum quarterly mean. Thus, data from 38 monitors at 38 distinct site locations were actually used. Seven of the 38 sites had complete data (i.e., 3 or 4 valid quarters) for each of the three years (2003-2005), 10 sites had only two years of complete data; and 21 sites had only one complete year of data. Complete quarters that were not part of a complete year were used. Likewise, all complete months were used, even if they did not correspond to the complete years. The 38 sites have an average of about 7 complete quarters and 19 complete months.

As with the Pb-TSP data processing, the PM_{10} data were used "as reported"; that is, ½ MDL substitutions were not made for reported concentrations less than or equal to MDL. Pb-PM_{10} sites were categorized similarly to the Pb-TSP sites. However, no Pb-PM_{10} sites fell into the source-oriented classification. 25 of the 38 Pb-PM_{10} sites were classified as urban; 20 of those 25 sites are located in CBSA's of 1 million or more population and the other 5 are located in smaller CBSA's. The 38 Pb-PM_{10} monitors are listed with various summary and demographic data in Appendix 2B, Table 2B-6.

Three statistical metrics were computed for the Pb-PM_{10} data: annual means, maximum quarterly means, and maximum monthly means. These metrics were calculated at the site level. They were calculated only for the overall 3-year period (2003-2005). Note that the 3-year annual mean statistic is actually the average of the annual means for the complete years; thus it is the average of three annual means, the average of two annual means, or the only available single complete annual mean. The 3-year maximum quarterly mean statistic represents the highest quarterly mean of the complete quarters (sites have from three to 12 complete quarters), and the 3-year maximum monthly mean represents the highest monthly mean of the complete months (each site has from nine to 36 complete months).

2.3.3.2 Current Concentrations

Monitoring site-level concentrations for each of the 3 statistical metrics (annual mean, maximum quarterly mean, and maximum monthly mean) are provided in Appendix 2B, Table 2B-6. Figure 2-28 shows the distributions of the annual means, maximum quarterly averages, and maximum monthly means for the 38 Pb-PM_{10} sites. The national composite average annual mean for Pb-PM_{10} was 0.006 $\mu g/m^3$ for the 3-year period, 2003-2005; the corresponding median annual mean was also 0.006 $\mu g/m^3$. The national composite average maximum quarterly mean was 0.012 $\mu g/m^3$ for 2003-2005 and the corresponding median maximum quarterly mean was 0.009 $\mu g/m^3$. The national composite average maximum monthly mean was 0.021 $\mu g/m^3$ and the median maximum monthly mean was 0.014 $\mu g/m^3$. Figure 2-29 shows distribution boxplots for the 25 urban sites and Figure 2-30 shows distribution boxplots for the 20 urban sites located in CBSA's with one million or more population. In these three figures (Figures 2-28 through 2-30), the boxes depict inter-quartile ranges and medians, whiskers depict the 5^{th} and 95^{th} percentiles, and asterisks identify composite averages. Additional points on the distribution for these statistics are given in Appendix 2B, Table 2B-7.

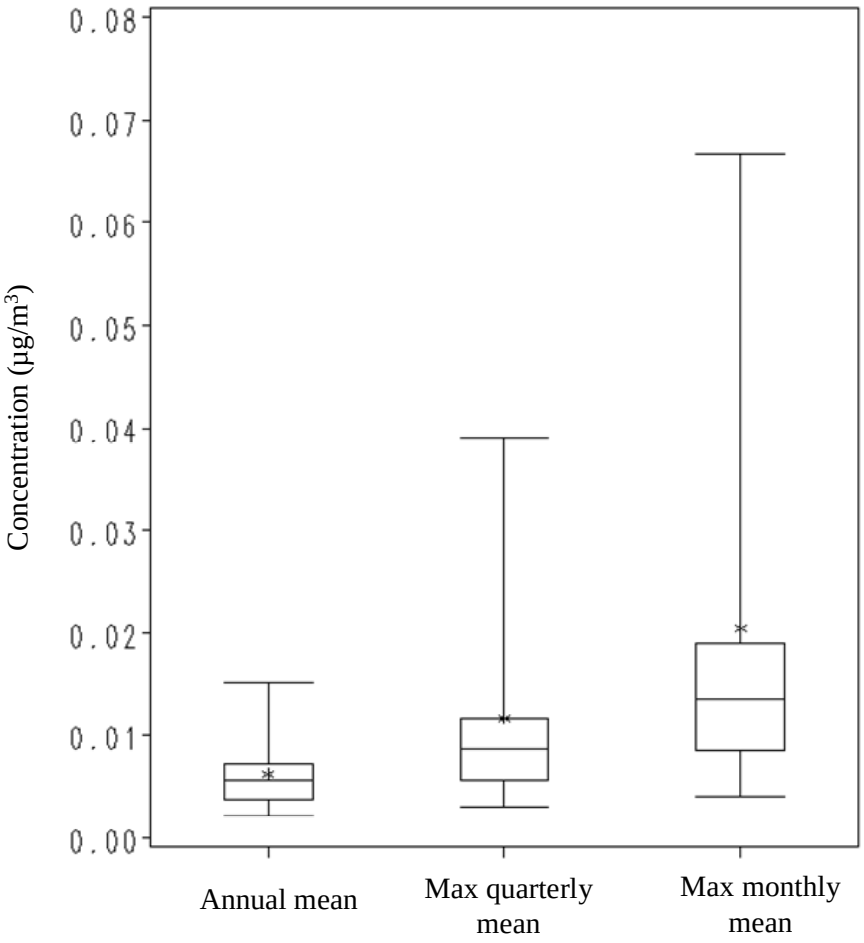


Figure 2-28. Distribution of Pb-PM_{10} concentrations (represented by 3 different statistics) at all 28 monitoring sites, 2003-2005.

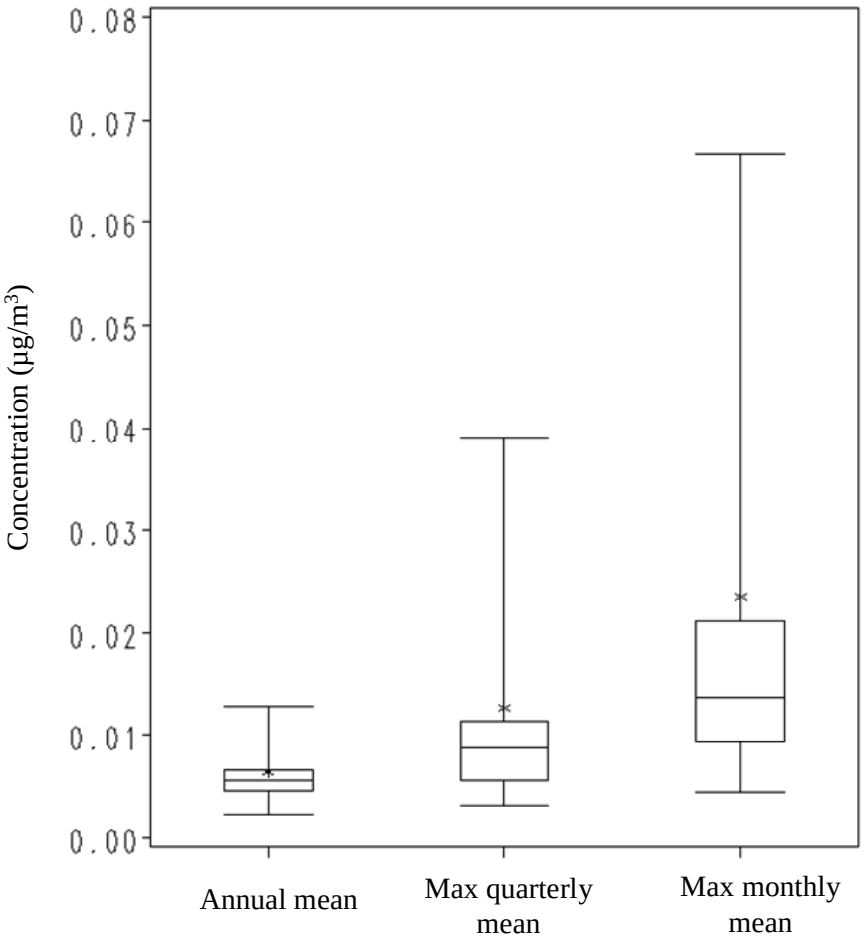


Figure 2-29. Distribution of Pb-PM_{10} concentrations (represented by 3 different statistics) at the 25 urban monitoring sites, 2003-2005.

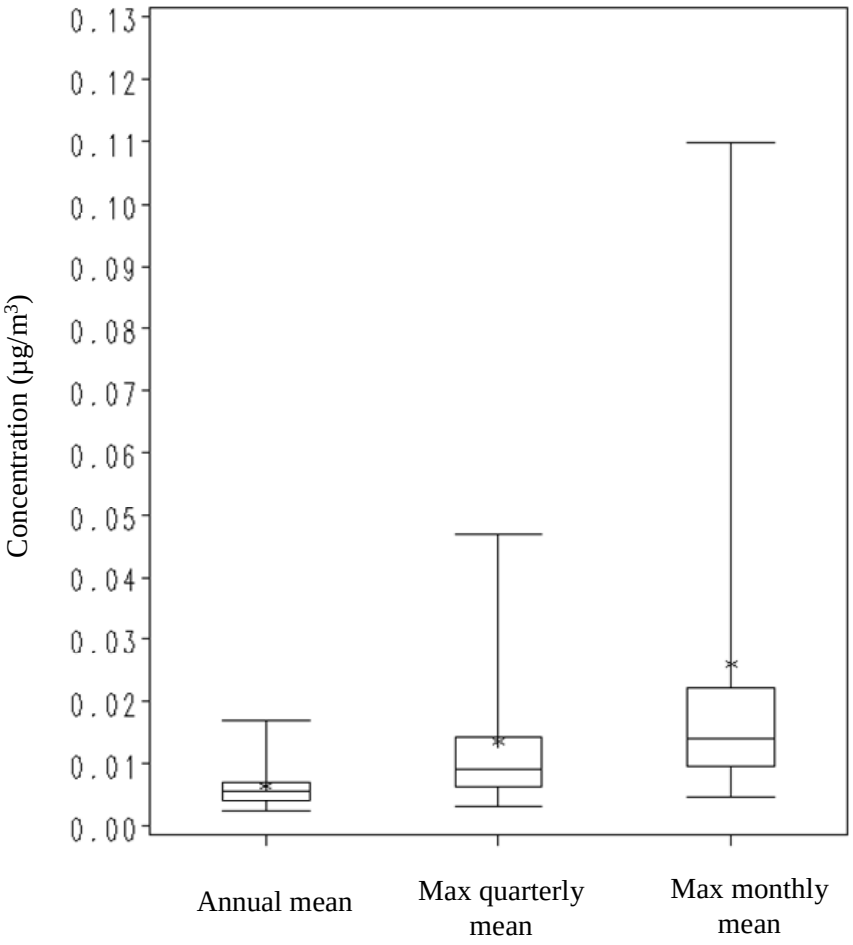


Figure 2-30. Distribution of Pb-PM_{10} concentrations (represented by 3 different statistics) at the urban monitoring sites located in CBSAs of ≥ 1 million population, 2003-2005.

Site-level annual means are mapped in Figure 2-31 and the corresponding maximum quarterly means are mapped in Figure 2-32.

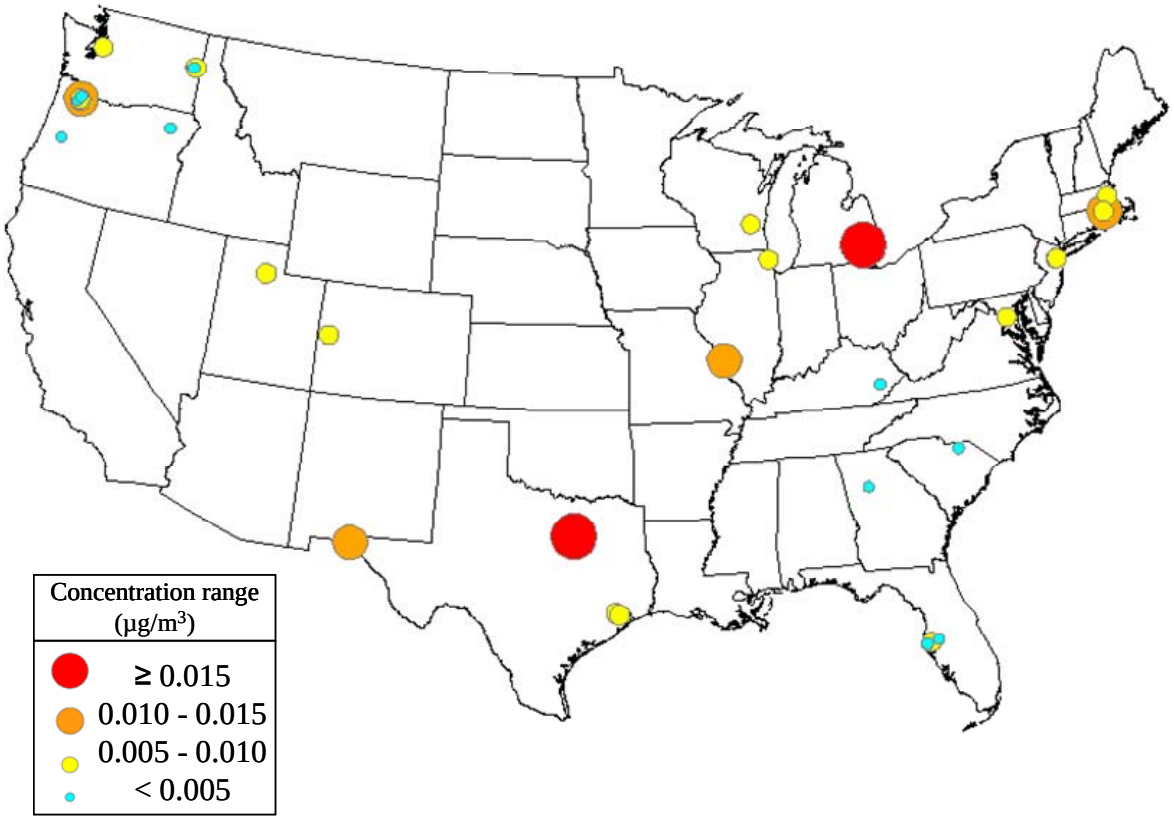


Figure 2-31. Pb-PM_{10} annual means (for all sites), 2003-2005.

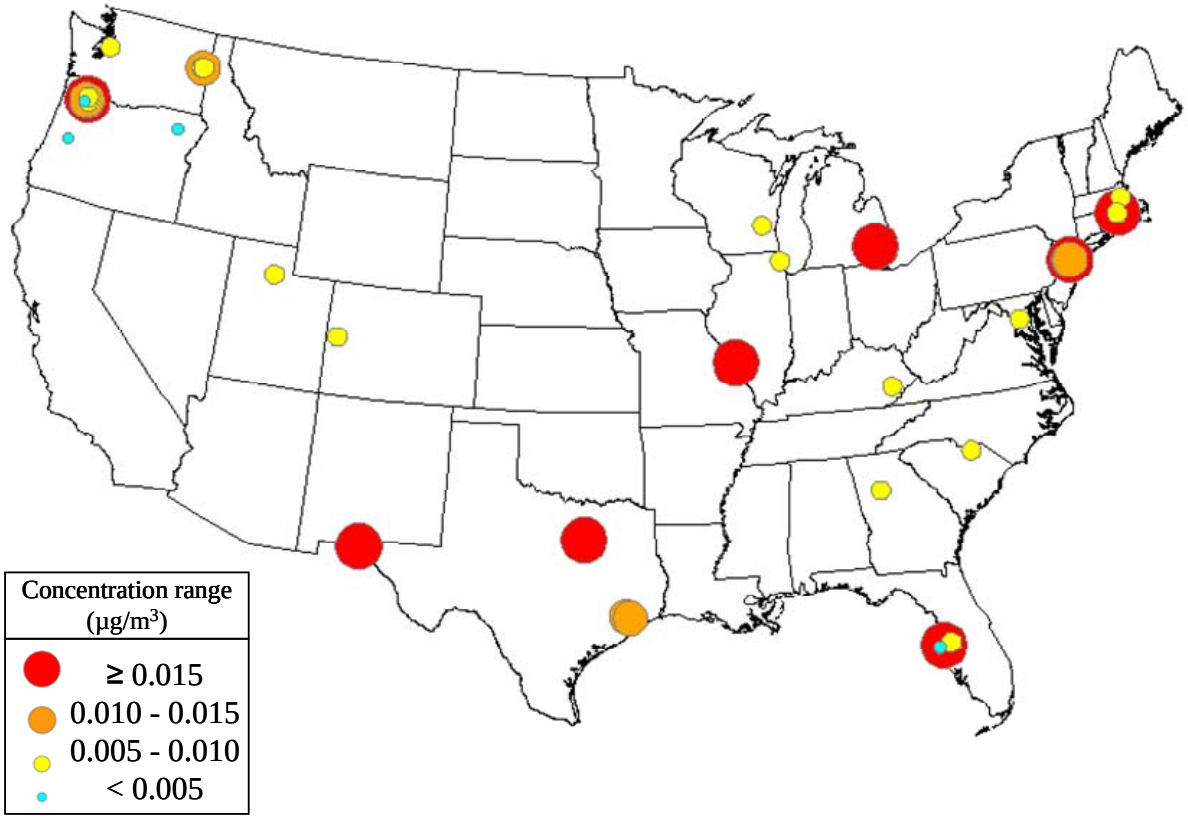


Figure 2-32. Pb-PM_{10} maximum quarterly means (for all sites), 2003-2005

2.3.4 Pb-$PM_{2.5}$

Two networks measure Pb in $PM_{2.5}$, the EPA CSN and the IMPROVE network. The CSN consists of 54 long-term trends sites (commonly referred to as the Speciation Trends Network or STN sites) and about 150 supplemental sites, all operated by state and local monitoring agencies. Most STN sites operate on a 1 in 3 day sampling schedule, while most supplemental sites operate on a 1 in 6 day sampling schedule. All sites in the CSN network determine the Pb concentrations in $PM_{2.5}$ samples and, as such, do not measure Pb in the size fraction >2.5 µm in diameter. Lead is quantified via the XRF method. The standard operating procedure for metals by XRF is available at: http://www.epa.gov/ttnamti1/files/ambient/pm25/spec/xrfsop.pdf. Data are managed through the AQS.

The IMPROVE network is administered by the National Park Service, largely with funding by EPA, on behalf of federal land management agencies and state air agencies that use the data to track trends in rural visibility. Lead in the $PM_{2.5}$ is quantified via the XRF method, as in the CSN. Data are managed and made accessible mainly through the VIEWS website (http://vista.cira.colostate.edu/views/), but also are available via the AQS. Samplers are operated by several different federal, state, and tribal host agencies on the same 1 in 3 day schedule as the STN.

The locations of the CSN are shown in Figure 2-33. Nearly all of the CSN sites are in urban areas, often at the location of highest known $PM_{2.5}$ concentrations. The first CSN sites generally began operation around 2000.

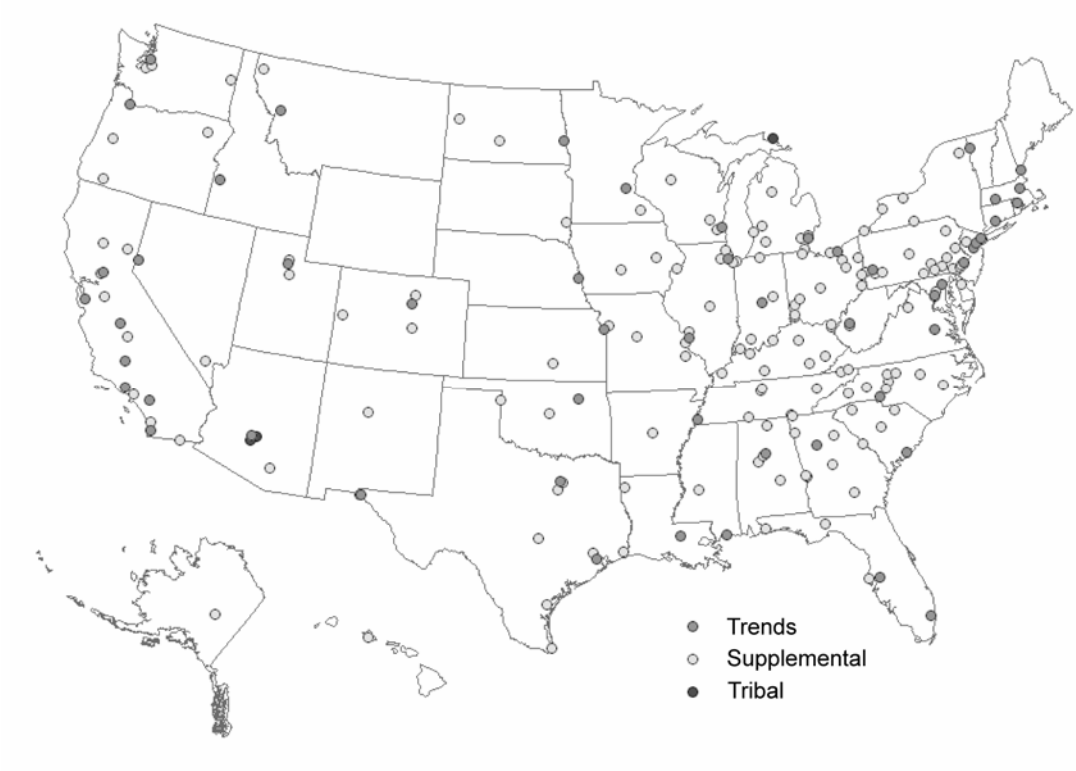


Figure 2-33. Pb-$PM_{2.5}$ (CSN) monitoring sites.

In the IMPROVE network, $PM_{2.5}$ monitors are placed in "Class I" areas (including National Parks and wilderness areas) and are mostly in rural locations (Figure 2-34). The oldest of these sites began operation in 1988, while many others began in the mid 1990s. There are 110 formally designated IMPROVE sites, which are located in or near national parks and other Class I visibility areas, virtually all of these being rural. Approximately 80 additional sites at various urban and rural locations, requested and funded by various parties, are also informally treated as part of the network.

Figure 2-34. Pb-$PM_{2.5}$ (IMPROVE) monitoring sites.

2.3.4.1 Data Analysis Details

2003-2005 Pb-$PM_{2.5}$ data (parameter code 88128, duration '7') were extracted from EPA's AQS on May 22, 2007. Data generated with IMPROVE collection/analysis methods were excluded from the central focus of this national characterization on the basis that most of the monitors utilizing those methods are located in rural or remote areas distant from both Pb sources and large populations. Most remaining data are associated with EPA's CSN program.

The same screening criteria utilized for Pb-PM_{10} were also implemented for Pb-$PM_{2.5}$: 1) a minimum of 10 observations per quarter, 2) for at least 3 quarters of one calendar year, and 3) at least 9 months with 4 observations each; all three criteria had to be met for inclusion. 278 monitors met the data completeness criteria. Of these 278 monitors, 7 were collocated with another complete monitor. Only one monitor from each collocated pair (i.e., from each site location) was kept in the analysis, specifically the one with highest maximum quarterly mean. Thus, data from 271 monitors at 271 distinct locations were actually used. 192 of the 271 sites had complete data (i.e., 3 or 4 valid quarters) for each of the three years (2003-2005), 40 sites had only two years of complete data; and 39 sites had only one complete year of data. Complete

quarters that were not part of a complete year were used. Likewise, all complete months were used, even if they did not correspond to the complete years. The 38 sites have an average of about 10 complete quarters and 29 complete months Pb-$PM_{2.5}$ data were used "as reported"; ½ MDL substitutions were not made for reported concentrations less than or equal MDL.

$PM_{2.5}$ sites were categorized similarly to the sites in the other size cuts. Only eight Pb-$PM_{2.5}$ sites were classified as source-oriented. 216 of the 271 Pb-$PM_{2.5}$ sites were classified as urban; 99 of those 216 sites are located in CBSAs of 1 million or more population and the other 117 are located in smaller CBSAs. The 271 Pb-$PM_{2.5}$ monitors are listed with various summary and demographic data in Appendix 2B, Table 2B-8.

2.3.4.2 Current Concentrations

The site-level Pb-$PM_{2.5}$ concentrations for each of the three statistics (annual mean, maximum quarterly mean, and maximum monthly mean) during the three-year period, 2003-2005, are shown in Appendix 2B, Table 2B-8. Figure 2-35 shows the distributions of the three statistical metrics for the 271 Pb-$PM_{2.5}$ sites; the boxes depict inter-quartile ranges and medians, whiskers depict the 5^{th} and 95^{th} percentiles, and asterisks identify composite averages. Additional points on the distribution for these statistics are given in Appendix 2B, Table 2B-9. The national composite average annual mean was 0.004 $\mu g/m^3$ for the 3-year period, 2003-2005; the corresponding median annual mean was 0.003 $\mu g/m^3$. The national composite average maximum quarterly mean was 0.008 $\mu g/m^3$ for 2003-2005 and the corresponding median maximum quarterly mean was 0.005 $\mu g/m^3$. The national composite average maximum monthly mean was 0.013 $\mu g/m^3$ and the median maximum monthly mean was 0.007 $\mu g/m^3$. As also shown in Appendix 2B, Table 2B-9, the median and mean site-level annual mean and maximum quarterly mean levels for source oriented sites were approximately double those for the non-source-oriented sites. Figure 2-36 maps the annual means for Pb-$PM_{2.5}$ sites.

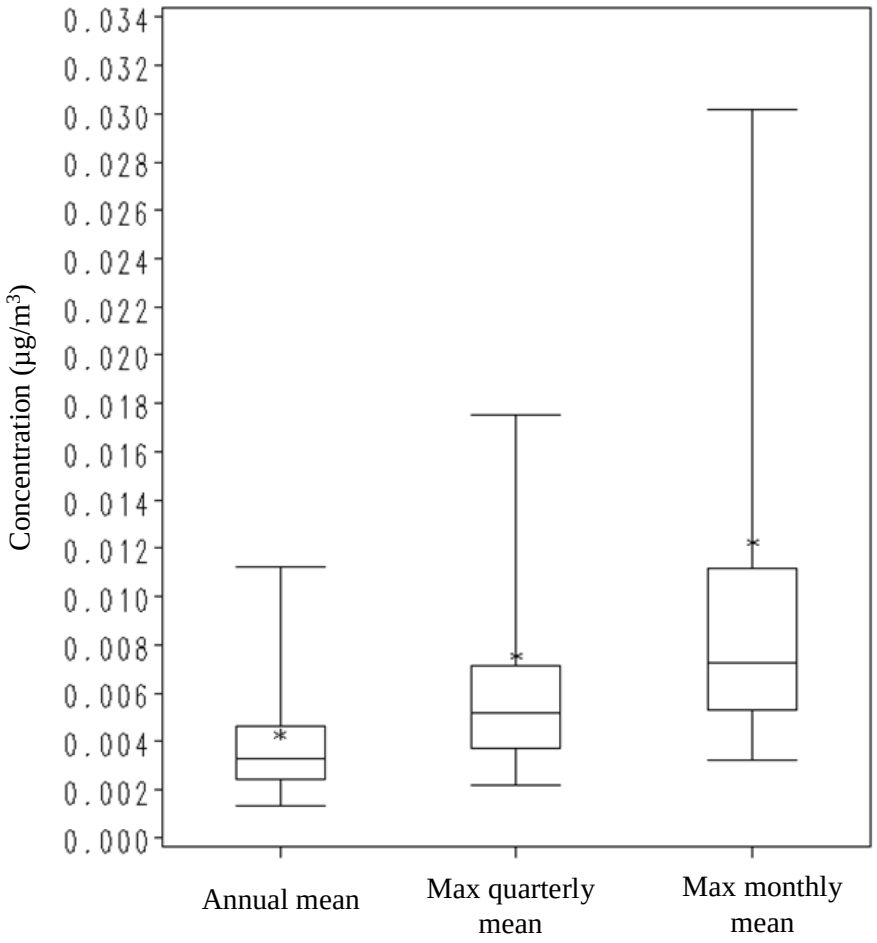


Figure 2-35. Distribution of Pb-$PM_{2.5}$ concentrations (represented by 3 different statistics) at all 271 monitoring sites, 2003-2005.

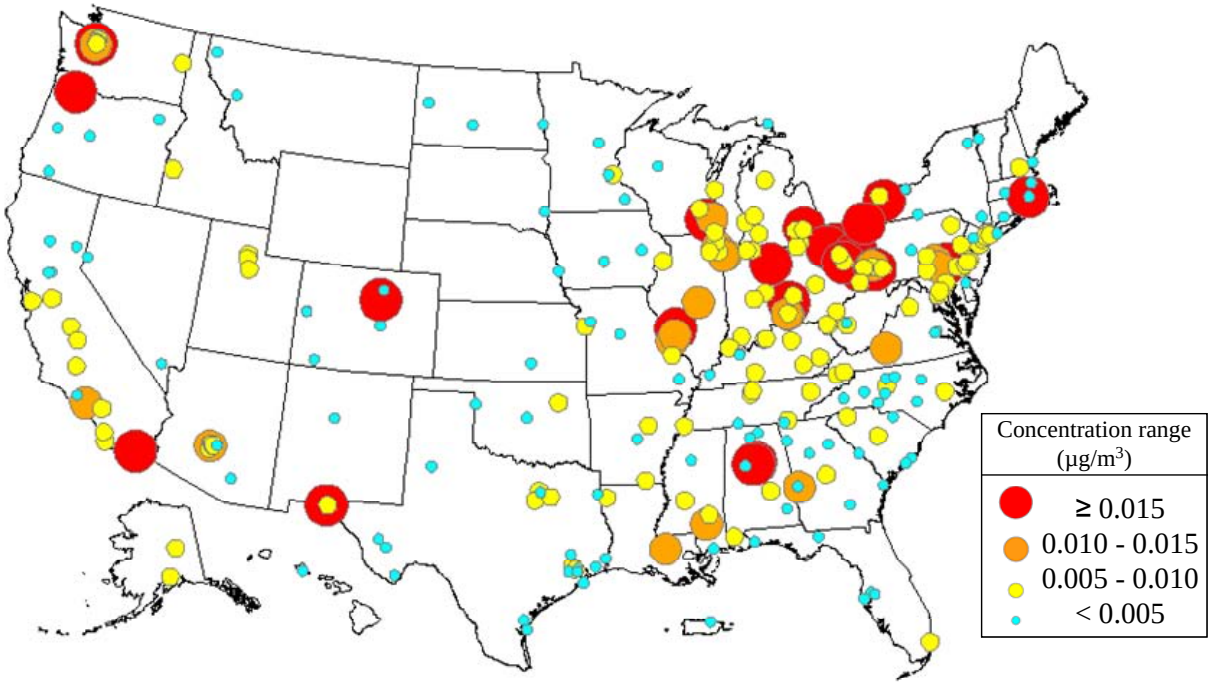


Figure 2-36. Pb-$PM_{2.5}$ annual means (for all sites), 2003-2005.

2.3.5 Relationships among Different Particle-sized Pb Concentrations

There are not many sites where Pb measurements are made in different PM size fractions at the same location and the same day (and where Pb values exceed minimum detection limits). Very few locations in the United States have measured all three PM size fractions at the same time. Table 2-8 shows information for sites with collocated Pb data size fraction data. The first table section shows sites with collocated Pb-TSP and Pb-PM_{10} measurements. For this particular analysis, data prior to 2003 (and in one case, data not from AQS) were utilized. In general, there is typically good correlation between Pb measurements in TSP and PM_{10}; the average site level correlation coefficient (r) is 0.79 for the 23 listed sites. Although site-level correlations ranged from 0.05 to 1.00, about two thirds of the sites (15) had correlations of 0.80 or better. All but one of the 23 sites was categorized as *not* source-oriented, and the low concentrations at those 22 sites seem to corroborate this classification. The lone known source-influenced site has much higher concentrations; it also has a high r (0.95). For the 23 sites listed, on the days collocated measurements were made, most of the measured Pb-TSP appears to fall in the PM_{10} fraction. On

average, daily Pb-PM_{10} concentrations were about 83 percent of the level of the Pb-TSP concentrations. All sites had an average daily ratio (Pb-PM_{10} / Pb-TSP) of 0.60 or greater; 19 of the 23 sites (83 percent) had an average ratio of 0.75 or more. The lone source site had an average ratio of 0.65. The next table section lists sites with collocated Pb-TSP and Pb-$PM_{2.5}$ during 2003-2005. The relationship between these two size fractions is not nearly as strong as for Pb-TSP and Pb-PM_{10}. At the 31 sites the average r was 0.49; only four of the 31 sites (~13 percent) have correlations of 0.80 or higher. Site-level correlations ranged from 0.04 to 0.96. On average, daily Pb-$PM_{2.5}$ concentrations were about 55 percent of the level of Pb-TSP concentrations; however, the percentage varied significantly by site. The last section of the table lists the sites with co-located Pb-PM_{10} and Pb-$PM_{2.5}$ during 2003-2005. It appears that the relationship between Pb-PM_{10} and Pb-$PM_{2.5}$ is stronger than that for Pb-TSP and Pb-$PM_{2.5}$ but not as strong as between Pb-TSP and Pb-PM_{10}. The average r for the 28 sites is 0.69; ten of the 26 sites (38 percent) have r's (correlation coefficients) of 0.80 or above. Site-level correlations ranged from 0.31 to 1.00. On average, daily Pb-$PM_{2.5}$ concentrations were about 79 percent of the level of Pb-PM_{10} concentrations; all but 3 sites had an average daily ratio (Pb-$PM_{2.5}$ / Pb-PM_{10}) of 0.50 or more. All of the above results should be viewed with some caution based on the limited number of sites with collocated data. Further, the relationships described can only be presumed to exist at sites with little influence from significant Pb sources. Lack of source-oriented PM_{10} data is a significant data gap in understanding size relationships where Pb exposures are of most potential concern.

Figure 2-37 summarizes (for all sites, not just the ones with collocated data) the annual means and medians for Pb in the various PM size fractions collected by different monitoring networks. Additionally, means and medians are presented for different site classifications. The top chart uses a scale that fits all shown categories, up to the maximum 95th percentile. The Pb-TSP monitor averages for the "source-oriented" subset (and other subsets that include those monitors; e.g., "TSP - all sites") dwarf the other categories. The bottom chart replots the data on a smaller concentration scale for enhanced resolution. Using the national averages (left bars), the Pb-TSP non-source-oriented annual means are about 2.6 times larger than the Pb-PM_{10} "all sites" averages (recall that no Pb-PM_{10} sites were classified as source-oriented); using the national medians (right bars), the ratio was closer to 1.8. Restated as a PM_{10}/TSP ratio it is 0.55, (i.e., 55 percent of TSP Pb is in the PM_{10} fraction); the collocated sites analysis discussed above has a median PM_{10}/TSP ratio of 0.85) The Pb-PM_{10} "all sites" averages are about 1.4 (using means) to 1.6 (using medians) times the Pb-$PM_{2.5}$ CSN urban averages. The Pb-$PM_{2.5}$ CSN urban averages are about 3.3 times the Pb-$PM_{2.5}$ IMPROVE averages.

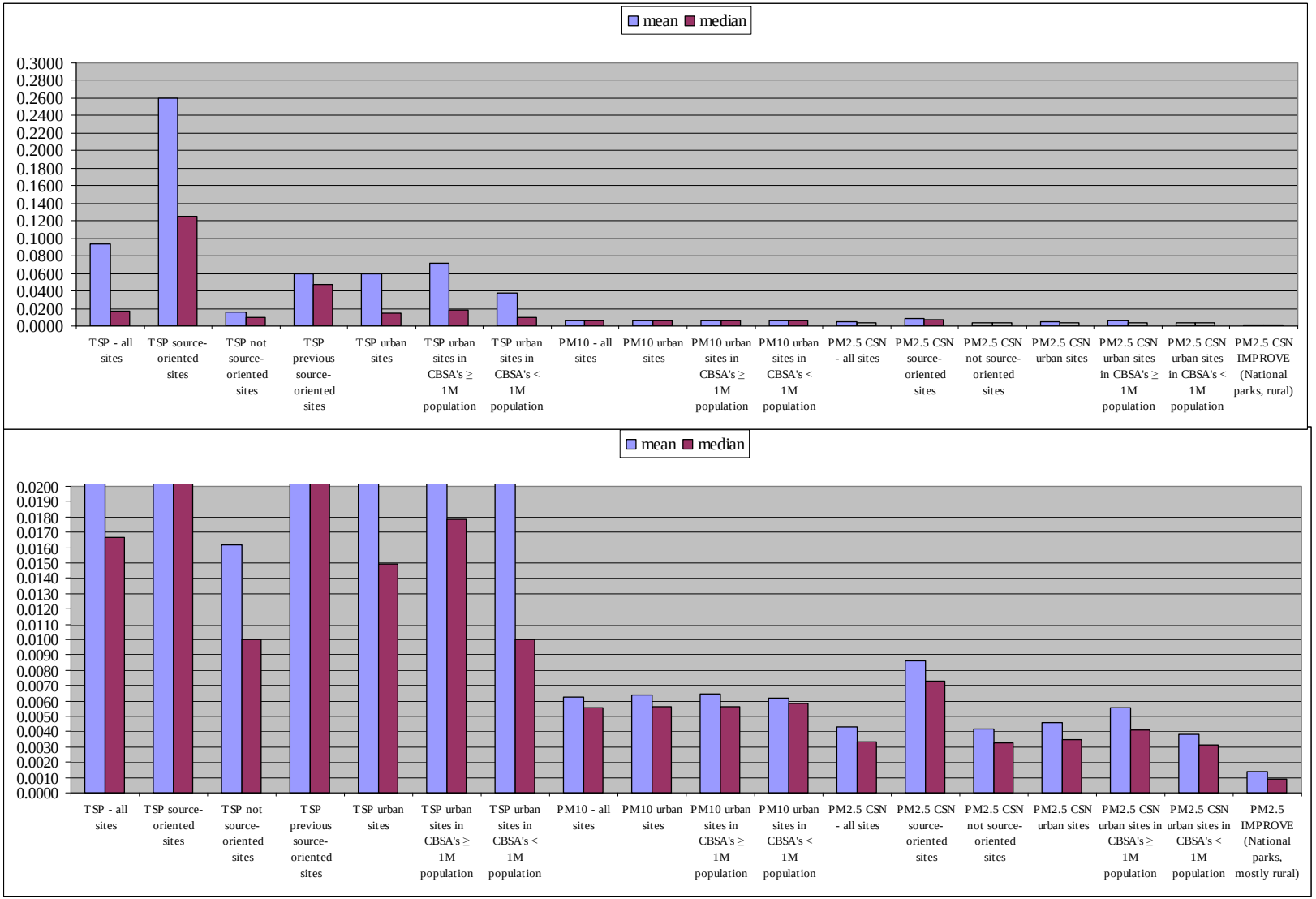


Figure 2-37. National mean and median monitor level Pb annual means for different size cut PM networks, 2003-2005.

Table 2-8. Monitoring sites with collocated Pb data in different size fractions

Site	Urban?	source-orinted?	years	number of collocated days	average (larger size cut)	average (smaller size cut)	minimum ratio of smaller size cut / larger size cut	maximum ratio of smaller size cut / larger size cut	average ratio of smaller size cut / larger size cut	correlation (r)
Collocated Pb-TSP and Pb-PM10										
060658001	1		1995 - 1996	54	0.031	0.018	0.24	1.17	0.60	0.44
060374002	1		1995 - 2000	129	0.041	0.022	0.14	1.64	0.60	0.76
270530053	1		1996 - 2001	13	0.017	0.008	0.33	0.89	0.61	0.93
Unknown		1	1988	22	2.245	1.121	0.17	1.86	0.65	0.97
060850004	1		1994 - 1999	23	0.025	0.018	0.38	1.57	0.77	0.72
261390009	1		2000 - 2001	26	0.013	0.009	0.46	1.07	0.78	0.94
201730009	1		1993 - 1997	18	0.019	0.015	0.38	1.53	0.81	0.50
060771002	1		1995 - 2000	53	0.021	0.017	0.39	1.84	0.81	0.82
202090015	1		1993 - 1997	118	0.028	0.021	0.29	2.79	0.82	0.65
201730007	1		1993 - 1997	18	0.018	0.013	0.33	1.27	0.82	0.08
202090020	1		1993 - 1997	107	0.092	0.059	0.04	11.92	0.85	0.99
060990002	1		1995 - 1998	17	0.024	0.015	0.40	1.19	0.85	0.99
260770905	1		1993 - 1996	78	0.017	0.014	0.55	1.55	0.85	0.99
201770007	1		1993 - 1997	19	0.017	0.014	0.50	1.33	0.87	0.67
060250005	1		1996 - 2001	205	0.031	0.027	0.27	1.55	0.87	0.95
295100085	1		2004 - 2004	26	0.021	0.017	0.59	1.13	0.89	0.98
060290014	1		1995 - 2000	32	0.020	0.019	0.65	1.44	0.92	0.98
261630033	1		2003 - 2006	167	0.031	0.028	0.33	2.69	0.92	0.90
060190008	1		1995 - 2001	32	0.020	0.018	0.60	1.33	0.92	0.94
201290003			1993 - 1998	14	0.020	0.017	0.55	1.45	0.93	0.91
201730008	1		1993 - 1997	16	0.015	0.015	0.50	2.06	0.98	0.05
490110001	1		2003 - 2003	19	0.024	0.025	0.96	1.10	1.03	1.00
201731012			1993 - 1997	22	0.022	0.022	0.63	3.33	1.05	0.96
Collocated Pb-TSP and Pb-PM2.5										
010730023	1	1	2005	14	0.0224	0.0154	0.00	2.04	0.77	0.59
010731009			2005	15	0.0042	0.0023	0.00	1.84	0.57	0.12
060250005	1		2003-2005	129	0.0159	0.0122	0.12	1.50	0.71	0.96
060371103	1		2003-2005	165	0.0228	0.0048	0.00	1.22	0.27	0.11
060658001	1		2003-2005	148	0.0138	0.0062	0.00	1.39	0.42	0.74
080010006	1		2003-2005	125	0.0323	0.0080	0.00	3.52	0.43	0.12
080410011	1		2003-2005	132	0.0176	0.0020	0.00	1.17	0.24	0.07
100032004	1		2003-2005	50	0.0100	0.0045	0.00	1.94	0.44	0.79
120571075	1		2003-2005	57	0.0038	0.0020	0.00	8.78	1.12	0.79
130690002			2003-2005	73	0.0021	0.0015	0.00	17.06	1.01	0.55
150032004	1		2003-2005	151	0.0015	0.0010	0.00	14.88	0.92	0.18
170314201	1		2003-2005	115	0.0113	0.0036	0.00	1.40	0.35	0.08
180970078	1		2003-2005	155	0.0097	0.0050	0.00	3.04	0.66	0.52
260810020	1		2005	29	0.0076	0.0042	0.00	1.43	0.53	0.59
261130001			2003-2005	157	0.0032	0.0023	0.00	6.54	0.78	0.59
261610008	1		2003-2005	69	0.0079	0.0042	0.00	1.82	0.52	0.51
261630001	1		2003-2005	172	0.0086	0.0042	0.00	1.48	0.46	0.59
261630033	1		2003-2005	152	0.0239	0.0118	0.00	2.20	0.48	0.71
270530963	1		2003	54	0.0106	0.0048	0.00	1.83	0.45	0.48
295100085	1		2004	53	0.0135	0.0077	0.00	7.40	0.94	0.32
350010023	1		2004	15	0.0029	0.0003	0.00	0.37	0.10	0.50
410390060	1		2003-2004	56	0.0027	0.0018	0.00	9.32	1.07	0.26
410510246	1		2003	57	0.0082	0.0058	0.00	3.11	0.74	0.69
420450002	1		2003-2005	167	0.0374	0.0042	0.00	0.75	0.11	0.04
450790019	1		2003-2005	54	0.0154	0.0096	0.00	1.81	0.58	0.79
470370023	1		2003-2004	48	0.0052	0.0041	0.00	8.12	0.79	0.40
482011034	1		2003-2005	147	0.0087	0.0027	0.00	1.73	0.32	0.42
490110001	1		2003-2005	23	0.0291	0.0036	0.00	0.29	0.11	0.95
490110004	1		2003	13	0.0107	0.0017	0.00	0.66	0.22	0.06
550270007			2004-2005	42	0.0067	0.0044	0.00	1.45	0.58	0.93
261630001 *			2003-2004	67	0.0083	0.0040	0.11	0.74	0.47	0.83
Collocated Pb-PM10 and Pb-PM2.5 (CSN)										
490110001	1		2003	20	0.0341	0.0041	0.00	0.28	0.11	0.95
250250042	1		2003-2005	108	0.0063	0.0026	0.00	2.15	0.46	0.52
482011039	1		2003-2005	161	0.0057	0.0020	0.00	2.34	0.49	0.43
080770017	1		2004-2005	108	0.0047	0.0023	0.00	3.24	0.51	0.31
261630033	1		2003-2005	148	0.0208	0.0113	0.00	1.92	0.54	0.89
490110004	1		2003	134	0.0060	0.0032	0.00	2.96	0.56	0.62
440070022	1		2003-2005	151	0.0110	0.0098	0.00	3.65	0.58	1.00
330150014	1		2003-2005	55	0.0033	0.0022	0.00	2.76	0.60	0.79
530330080	1		2003-2005	159	0.0047	0.0033	0.00	3.75	0.72	0.86
550270007			2005	48	0.0057	0.0044	0.00	2.43	0.74	0.93
330110020	1		2003-2005	59	0.0046	0.0033	0.00	2.79	0.77	0.54
410610119			2004	95	0.0017	0.0011	0.00	6.86	0.77	0.46
482011039 *			2004	35	0.0025	0.0017	0.27	2.23	0.78	0.81
410390060	1		2004	75	0.0024	0.0014	0.00	7.31	0.78	0.63
530330080 *			2003-2004	99	0.0046	0.0034	0.25	3.60	0.78	0.87
410510246	1		2003	120	0.0096	0.0084	0.00	4.04	0.83	0.99
110010043	1		2004-2005	85	0.0049	0.0039	0.00	10.43	0.86	0.39
450250001			2004	111	0.0029	0.0022	0.00	4.63	0.88	0.52
530630016	1		2005	54	0.0058	0.0037	0.00	4.23	0.88	0.77
295100085	1		2003-2005	148	0.0130	0.0096	0.00	7.30	0.89	0.55
170314201	1		2005	56	0.0059	0.0047	0.00	13.13	1.00	0.63
130890002	1		2003-2005	158	0.0026	0.0028	0.00	3.86	1.02	0.95
130890002 *			2004	34	0.0038	0.0049	0.49	1.81	1.09	0.99
120573002			2004-2005	113	0.0036	0.0025	0.00	19.63	1.16	0.48
211930003			2003-2005	165	0.0039	0.0042	0.00	33.45	1.42	0.58
121030026	1		2004-2005	70	0.0025	0.0023	0.00	13.13	1.42	0.61

As described in the CD, several special time-limited studies have investigated Pb concentrations in different PM size fractions (CD, p. 3-13). For example, average Pb concentrations reported in a rural area in the southeastern U.S. were 6.11 ng/m^3 in $PM_{2.5}$ and 15.04 ng/m^3 in TSP samples, with the average total mass concentration of 9.5 µg/m^3 and 19.1 µg/m^3 for $PM_{2.5}$ and TSP, respectively (Goforth and Christoforou, 2006); thus, Pb constituted a similar very small proportion of particles in each size fraction. Another study included two areas in the Los Angeles basin (Singh et al, 2002). In Downey, a site where refineries and traffic contribute heavily to particle concentrations, Pb was proportionally greater in the fine and ultrafine fractions of PM_{10}. In Riverside, which is considered a receptor site for particles transported from the Los Angeles basin and also has agricultural sources, Pb was proportionally greater in the coarse fraction of PM_{10}. In Boston, MA, Pb concentrations of 326 ng/m^3 and 75.6 ng/m^3 were reported from $PM_{2.5}$ and $PM_{10-2.5}$ (Thurston and Spengler, 1985). Overall, these findings suggest that for locations primarily impacted by combustion sources, Pb concentrations appear to be higher in the fine fraction of particles. However, at locations impacted by noncombustion sources (e.g., agriculture), Pb contained in the larger particles can be of significantly higher concentrations than those for the fine particles.

2.3.6 Summary

Ambient air Pb concentrations are measured by four monitoring networks in the United States, all funded in whole or in part by EPA. These networks - the Pb NAAQS compliance network, the $PM_{2.5}$ STN, the $PM_{2.5}$ IMPROVE network, and the PM_{10} network – provide Pb measurements for 3 different sizes of PM, and the $PM_{2.5}$ size is measured separately in urban and remote locations.

Airborne concentrations of Pb-TSP in the United States have fallen dramatically over the last 30 years due largely to the phase-out of leaded gasoline additives. Despite this decline, there have still been a small number of areas that have not met the current Pb NAAQS over the past few years. The sources of Pb in these areas are stationary sources (e.g. primary and/or secondary smelters). There appears to be significant ‘under-monitoring’ near large Pb emission point sources. Except for the monitors in a limited number of areas, Pb-TSP averages are quite low with respect to the current NAAQS; the median monitor level maximum quarterly average for 2003-2005 is about fifty times lower than the 1.5 µg/m^3 NAAQS level. However, when current concentrations are compared to alternative NAAQS thresholds (e.g., 0.05 and 0.20 µg/m^3), the number of locations that exceed those levels, and their associated populations, are much higher. For example, over 25 percent of the monitored Pb-TSP population exceeded the 0.05 µg/m^3 max quarterly level in 2003-2005 and 5 percent exceeded the 0.20 level.

Some monthly variability is common for ambient Pb concentrations. The current form of the standard (quarterly average) attempts to account for seasonal variability. As suggested during the last review, a shorter averaging period (monthly) would better capture short-term increases in Pb concentrations (USEPA 1990). Although there have only been 3 sites that violated the 1.5 $\mu g/m^3$ max quarterly average NAAQS during the 2003 – 2005 period, 11 sites violated that level with respect to a maximum monthly average.

There are not many sites that collect ambient Pb data in all three size ranges. Analyses of the available limited collocated Pb data for different size particles indicate that TSP-sized Pb and PM_{10}-sized Pb are fairly well correlated, however, almost all of the study sites were presumably not source-oriented. Additional source-oriented Pb-PM_{10} monitoring, collocated with Pb-TSP, would benefit additional evaluations. If further analyses also corroborate a fairly strong TSP-PM_{10} Pb relationship for source-oriented sites, Pb-PM_{10} measurements may be useful as a Pb-TSP surrogate.

2.4 AIR QUALITY MODELING

2.4.1 National Air Toxics Assessment

As part of the Agency's national air toxics assessment (NATA) activities, a national scale assessment of hazardous air pollutants including Pb compounds has been performed twice over the past few years (USEPA 2006b, 2002c, 2001a). These two assessments included the use of the NEI for the years 1996 and 1999, respectively, with atmospheric dispersion modeling to predict associated annual average Pb air concentrations across the country. A national scale assessment is not yet available based on the 2002 NEI. A number of limitations are associated with the 1996 and 1999 ambient concentration estimates (see Section 2.4.1.2) and the underlying emissions estimates (e.g., see Section 2.2.5). While the associated limitations handicap a reliance on the absolute magnitude of these estimates, they may prove informative with regard to relative patterns of concentrations across the country, and are presented in that light.

2.4.1.1 Methods

To develop national-scale estimates of annual average ambient Pb concentrations, EPA used the Assessment System for Population Exposure Nationwide (ASPEN) model. ASPEN uses a Gaussian model formulation and climatological data to estimate long-term average pollutant concentrations. The ASPEN model takes into account important determinants of pollutant concentrations, such as: rate of release, location of release, the height from which the pollutants are released, wind speeds and directions from the meteorological stations nearest to the release, breakdown of the pollutants in the atmosphere after being released (i.e., reactive decay), settling of pollutants out of the atmosphere (i.e., deposition), and transformation of one

pollutant into another (i.e., secondary formation). ASPEN concentration estimates do not account for day-of-week or seasonal variations in emissions (USEPA, 2001a).

For each source, the model calculates ground-level concentrations as a function of radial distance and direction from the source at a set of receptors laid out in a radial grid pattern. For each grid receptor, concentrations are calculated for each of a standard set of stability class/wind speed/wind direction combinations. These concentrations are averaged together using the annual frequency of occurrence of each combination (i.e., the climatology) as weightings to obtain annual average concentrations (USEPA, 2001a). For the 1999 NATA assessment, meteorological data for 1999 were used and the frequency distributions were also stratified by time of day into eight 3-hour time blocks. This along with similar emission rate stratification helps to preserve any characteristic diurnal patterns that might be important in subsequent estimation of population exposure. The resulting output of ASPEN is a grid of annual average concentration estimates for each source/pollutant combination by time block (USEPA, 2001a).

Annual average concentration estimates for grid receptors surrounding each emission source are spatially interpolated to the census tract centroids within the 50 kilometers impact zone, and contributions from all modeled sources are summed to give cumulative ambient concentrations in each census tract. By accounting for all identified source categories (including background concentrations, which are added to the ASPEN-calculated concentrations), the sum of the concentration increments yields an estimate of the overall Pb concentration within each census tract. For many pollutants modeled, total concentrations include a "background" component which includes concentrations due to natural sources, sources not in the emissions inventory, and long-range transport (USEPA, 2001a). In the case of Pb, however, a background concentration value of zero was used.

2.4.1.2 Findings and Limitations

Historical studies show that Gaussian dispersion models, such as ASPEN, typically agree with monitoring data within a factor of 2 most of the time. In the case of Pb in the NATA assessment, model estimates at monitor locations were generally lower than the monitor averages for Pb, suggesting that the modeling system (i.e., emissions estimates, spatial allocation estimates, dispersion modeling) may be systematically underestimating ambient concentrations. This may be particularly true for Pb as metals tend to deposit rapidly with distance from the source according to their particle size and weight. The model-to-monitor analysis is described in detail at http://www.epa.gov/ttn/atw/nata1999/99compare.html. The modeling system underestimation may also be due in part to a lack of accounting for resuspension of previously emitted and deposited particles (these resuspended particles may be observed by the monitors, but they are not accounted for in the emissions inventory, and thus would not contribute to the

model estimate). For more details on the limitations of the 1999 NATA national scale assessment, see http://www.epa.gov/ttn/atw/nata1999/limitations.html.

Because higher Pb concentrations are associated with localized sources, which are not well-characterized by this modeling approach, national scale assessments such as this can only provide answers to questions about emissions, ambient air concentrations, exposures and risks across broad geographic areas (such as counties, states and the country) for that period. They are also based on assumptions and methods that limit the range of questions that can be answered reliably such as identifying Pb exposures and risks for specific individuals, or identifying exposures and risks in small geographic regions such as a specific census tract.

Given the limitations of this analysis with regard to estimating Pb concentrations nationally (see above), specific absolute ambient concentration estimates for Pb compounds generated by this analysis are not presented here. The general pattern of results, presented elsewhere (USEPA, 2006b), is consistent with the following conclusions: 1) there are Pb concentrations projected in remote areas; 2) there are distinct geographical variations in ambient Pb concentrations; concentrations in rural areas are generally much lower than in urban areas; and, 3) there are areas with high Pb concentrations associated with localized sources with high emissions. These results also support the general conclusion that more detailed source and site specific analyses are needed when addressing Pb impacts.

2.4.1.3 Summary

The NATA national scale assessment estimates based on 1999 NEI reflect the quantity and distribution of Pb emissions, with the highest estimates associated with point sources. For example, the census tract with the highest estimated Pb concentration is located in the county with the highest Pb emissions estimate in the 1999 NEI, and the second highest census tract is located in a county with a now-closed major Pb smelter. Limitations of the assessment, however, contribute to uncertainty and potential underestimation of Pb concentrations.

2.4.2 Community Multiscale Air Quality Model

The Community Multiscale Air Quality (CMAQ) model is a three-dimensional grid-based Eulerian air quality model designed to estimate the formation and fate of oxidant precursors, primary and secondary particulate matter (including Pb) concentrations and deposition over regional and urban spatial scales, such as over the contiguous U.S. (Byun and Ching, 1999; Byun and Schere, 2006).

The key inputs to the CMAQ model include emissions from anthropogenic and biogenic sources, meteorological data, and initial and boundary conditions. The CMAQ meteorological input files were derived from simulations of the Pennsylvania State University / National Center for Atmospheric Research Mesoscale Model (Grell et al., 1994). This model, commonly referred

to as MM5, is a limited-area, nonhydrostatic, terrain-following system that solves for the full set of physical and thermodynamic equations which govern atmospheric motions. The lateral boundary and initial species concentrations were obtained from a three-dimensional global atmospheric chemistry model, GEOS-CHEM (Yantosca, 2004; Moon and Byun, 2004).

To assess the potential usefulness of CMAQ simulations for this review, a preliminary national scale air quality modeling analysis was performed using version 4.6 of the CMAQ modeling system that simulates urban and regional air quality. Aerosol phase HAPs track toxic components within particulate matter and are treated as chemically inert but undergo the same microphysical processes and deposition rates determined within the aerosol module (Binkowski and Roselle, 2003). Lead is treated as aerosol phase HAP within CMAQ. Simulations were performed for the period January 1 through December 31, 2002. The computational grid used 148 by 112 grid cells, with horizontal dimensions equal of 36 km on each side, to cover the continental United States. Vertically, the model domain spanned from the surface to about 15 km and divided the distance into 14 layers based on sigma pressure coordinates.

The 2002 CMAQ model simulation used meteorological data for that year produced by the MM5 model that were processed through the Meteorology-Chemical Interface Processor (MCIP), version 3.1. The Sparse Matrix Operator Kernal Emissions (SMOKE) version 2.0 (http://cf.unc.edu/cep/empd/products/smoke/index.cfm) was used to produce model-ready emissions inputs for the CMAQ simulation based on the 2002 National Emission Inventory (NEI) version 3 (http://www.epa.gov/ttn/chief/net/2002inventory.html) For CMAQ, the biogenic emissions were computed based on the 2002 meteorology data using the Biogenic Emission Inventory System (BEIS) version 3.13 model from SMOKE. The BEIS3.13 model computes gridded, hourly, model-species emissions for combination with the anthropogenic emissions to put into CMAQ. Emissions are calculated for the U.S., Mexico, and Canada and accounts for CO, VOC, and NO_X emissions from vegetation and soils. The meteorology data on which the biogenic emissions depend are the same as the meteorology data input to the CMAQ model.

CMAQ outputs include Pb-$PM_{2.5}$, Pb-PM_{10} and atmospheric deposition. To evaluate predictions, we obtained observations of metals in $PM_{2.5}$ from the US EPA's Air Quality System database (AQS). Observations are on a national scale and have an averaging period equal to 24 hours. Sampling frequencies range from several days to a week (see Section 2.3 for more discussion on Pb monitoring). Model performance varies by season but in general, predicted concentrations of lead underestimate observed concentrations (negative biases) and have a weak ability to match the time dependency of observations (low correlation coefficients). Lead predictions match observations within 50% at all locations. Comparison between predictions and observations is difficult when the observation equals zero and the prediction is above or near

the detection limit. In this case, the comparison gives an incorrectly high value of model biases because the actual observation lies below the detection limit by an uncertain amount especially at rural locations such as desert, forests and agricultural areas.

There are several reasons for the differences between modeled and monitored values including emissions and meteorological factors. One reason is that sources of emissions from Canada and Mexico were not included in the modeling. Lead associated with resuspension of historic Pb emissions (CD, Section 2.3.3) is also not included. Additionally, Pb emissions arise from both fuel combustion and aerosol suspension driven by mechanical action such as winds and disturbance of lead-bearing soil by construction activities. These types of sources driven by wind speed will have significant effects in areas that had large historic emissions yet are not included in the current emission inventory and thus are not input into the model. Issues related to aerosol suspension of Pb by mechanical action are equally important for both size classes, Pb-$PM_{2.5}$ and Pb-PM_{10}.

Model resolution at 36 km^2 grids affects emission processes because grid cells use rates that are composites of many sources. This method removes how individual sources affect concentrations based on their time dependent emissions and location relative to monitoring sites which are influenced by nearby sources. Composite sources produce larger errors in grid cells where individual sources have large changes in emissions. The error often likely occurs over populated areas such as residential areas in urban locations. Observed concentrations reflect unpredictable activity in adjacent automotive traffic, construction and businesses.

Besides the errors in the meteorological and emissions inputs, the CMAQ model does not include an aerosol mode that represents ultrafine particles (diameters < 50 nm). The aerosol mode is emitted by combustion and industrial sources such as diesel engines, boilers, metal foundries and plating or produced by gas to particle conversion near emission sources. Thus CMAQ can underpredict at urban and some suburban locations where sources of ultrafine particles are more numerous because coagulation is too slow to grow ultrafine particles into fine particles. Issues related to ultrafine lead emissions pertain to the $PM_{2.5}$ fraction since ultrafines would not be expected to grow into particles larger than $PM_{2.5}$. PM_{10} emissions are usually dominated by particles formed by mechanical action and not from combustion processes. These comparisons are expected to improve in the future from better Pb emissions inventories (suspension, biomass burning, and anthropogenic fuel composition) and better CMAQ science (smaller grid resolution, better boundary conditions, and better algorithms).

2.5 POLICY-RELEVANT BACKGROUND IN AIR

Some amount of Pb in the air derives from background sources, such as volcanoes, sea salt, and windborne soil particles from areas free of anthropogenic activity (CD, Section 2.2.1).

The impact of these sources on current air concentrations is expected to be quite low (relative to current concentrations) and has been estimated to fall within the range from 0.00002 $\mu g/m^3$ and 0.00007 $\mu g/m^3$ based on mass balance calculations (CD, Section 3.1 and USEPA 1986, Section 7.2.1.1.3). The midpoint in this range, 0.00005 $\mu g/m^3$, has been used in the past to represent the contribution of naturally occurring air Pb to total human exposure (USEPA 1986, Section 7.2.1.1.3). The data available to derive such an estimate are limited and such a value might be expected to vary geographically with the natural distribution of Pb. Comparing this to reported air Pb measurements is complicated by limitations of the common analytical methods and by inconsistent reporting practices. This value is one half the lowest reported nonzero value in AQS. For the purposes of the risk assessment described in Chapter 4, the value of 0.00005 $\mu g/m^3$ was selected as representative of policy-relevant background Pb in air. Unlike for other criteria pollutants, the role of this value for Pb is limited. In considering risk contributions from policy-relevant background, the contributions from exposures to nonair media are such that any credible estimate of policy-relevant background in air is likely insignificant in comparison.

2.6 ATMOSPHERIC DEPOSITION

As described in Section 2.1.2, deposition is the path by which Pb particles are removed from the atmosphere and transferred to other environmental media through deposition, and, as recognized in Chapters 4 and 6, deposited Pb, plays a major role in human and ecological exposures. There are several approaches described in the literature for estimating atmospheric deposition, or transfer of Pb from the atmosphere to soil or water bodies. These include measurements of Pb in rainfall (wet deposition) and on collection surfaces during dry periods (dry deposition); dry deposition has also been estimated via measurements of airborne Pb particles coupled with estimates of deposition velocity (see CD, Section 2.3.2). Studies that measure Pb in sediment or soil cores, coupled with isotope dating methods (see CD, Sections 2.2.1 and 8.1.2), provide observations informative of atmospheric deposition rates and trends. As there are currently no nationwide Pb atmospheric deposition monitoring programs, the information in this section is drawn from a variety of sources as discussed in the CD (CD, Sections 2.3, 8.2.2 and AX7.1.2.3).

2.6.1 Temporal Trends

The available atmospheric studies of dry, wet and bulk deposition of Pb indicate a pronounced downward trend in Pb deposition in the U.S. during the 1980s to early 1990s, likely reflecting the reduction in atmospheric levels during that time period (CD, Section 2.3.2). As an example, Pirrone and others (1995) estimated an order of magnitude reduction in dry deposition from 1982 to 1991 in Detroit, Michigan (CD, Section 2.3.2). Measurements of Pb in rainfall in

Lewes, Delaware (a small town at mouth of Delaware Bay) have fallen from approximately 3 μg/L in the early 1980s to less than 1 μg/L by 1989 (CD, pp. 2-60 and AX7-35; Scudlark et al., 1994). Sediment core studies provide evidence of the larger historical pattern (CD, Section 2.3.1). For example, Jackson and others (2004) reported that deposition to the Okefenokee Swamp, Georgia, USA peaked during the period from 1940s through 1970s, followed by a period of steady decline into the 1990s (CD, Section 2.3.1).

2.6.2 Deposition Flux Estimates since the Last Review

Contemporary rates of total Pb loadings to terrestrial ecosystems are estimated at approximately 1 to 2 mg/m^{2}year (CD, p. AX7-36). In association with the Great Lakes Water Quality Agreement between the United States and Canada, a deposition monitoring network was established to estimate regional atmospheric inputs to the Great Lakes (Voldner and Eisenreich, 1989). Based on measurements from that network, total Pb deposition to three of the Great Lakes (Lakes Superior, Michigan and Erie) in the early 1990s was estimated to be on the order of 1.5 -2 mg/m^2-year (CD, pp. 2-57 and 2-60; Sweet et al., 1998).

For Lakes Superior and Michigan, dry deposition estimates were greater than those for wet deposition by a factor of 1.5 to 2, while dry deposition to Lake Erie was estimated to be less than 80% of wet deposition (CD, pp. 2-57 and 2-60; Sweet et al., 1998). In the mid-Atlantic region during the 1990s, dry deposition was estimated to be equal to or lower than wet deposition, contributing ≤50% of total deposition (CD, Section 2.3.2; Scudlark et al., 2005). Reports of wet deposition for this region during the 1990s range from nearly 400 to just over 600 μg/m^2-year (CD, Section 2.3.2).

2.7 OUTDOOR DUST AND SOIL

Lead in outdoor dust and soil may be derived from a range of sources including current and historical air emissions sources, as well as miscellaneous nonair sources (e.g., land disposal of wastes and subsequent weathering). Both media may play a substantial role in human and ecological exposures. With regard to human exposures, contaminated soil can be a potential source of Pb exposure, particularly for children (CD, Section 3.2). Another source of children's exposure, as discussed in the CD (Sections 3.2 and 4.4), is house dust, which may be derived from Pb in outdoor dust and soil as well as from ambient air Pb.

2.7.1 Outdoor Dust

Outdoor dust refers to particles deposited on outdoor surfaces. Lead in outdoor dust has been associated with active point sources as well as well as older urban areas. For example, a 50% reduction in dust Pb levels accompanied a 75% reduction in airborne Pb concentrations associated with replacement of a smelting facility in Canada (CD, pp. 3-23 to 3-24).

Additionally, Caravanos and others (2006b) have described Pb in dust (particulate matter) deposited on surfaces in New York City. Lead levels have been found to be higher in dust on or near roadways, or in older urban areas as compared to newer or rural areas (CD, Sections 3.2.3 and 3.2.4; Caravanos et al 2006a,b). As with surface soil, contact with outdoor dust may contribute to incidental ingestion of environmental contaminants including Pb. Additionally, as stated in the CD (p. 2-62), the "resuspension of soil-bound Pb particles and contaminated road dust can be a significant source of airborne Pb". Resuspension, thus, provides a pathway for Pb transport into residences and its contribution to Pb in house dust. As mentioned in Section 2.1.2, particles containing Pb may be resuspended into the air by a range of processes including wind and vehicular traffic, as well as other mechanical processes including pedestrian traffic, agricultural operations, and construction.

2.7.2 Soil

A reservoir of 0.5 to 4 g/m^2 gasoline additive-derived Pb is estimated to exist in U.S. soils (CD, p. AX7-36), with most contained in the upper soil horizons (O + A horizons). Studies have indicated that industrial Pb can be strongly sequestered by organic matter and by secondary minerals such as clays and oxides of Al, Fe, and Mn, (CD, pp. AX7-24 to AX7-39). Accordingly, migration (e.g, to groundwater) and biological uptake of Pb in ecosystems is considered to be relatively low, with variability of Pb mobility in different systems influenced by factors including elevation and climate, vegetation type, acidity, and soil composition (CD, Sections 2.3.5 and AX7.1.2.3). Generally then, forest floors are considered to currently act as net sinks for Pb, and burial or movement of Pb over time down into lower soil/sediment layers also tends to sequester it away from more biologically active parts of the watershed, unless later disturbed or redistributed (CD, p. AX7-36). In areas of exposed soil, however, there is potential for interaction with airborne Pb (as discussed in Sections 2.7.1 and 2.1.2).

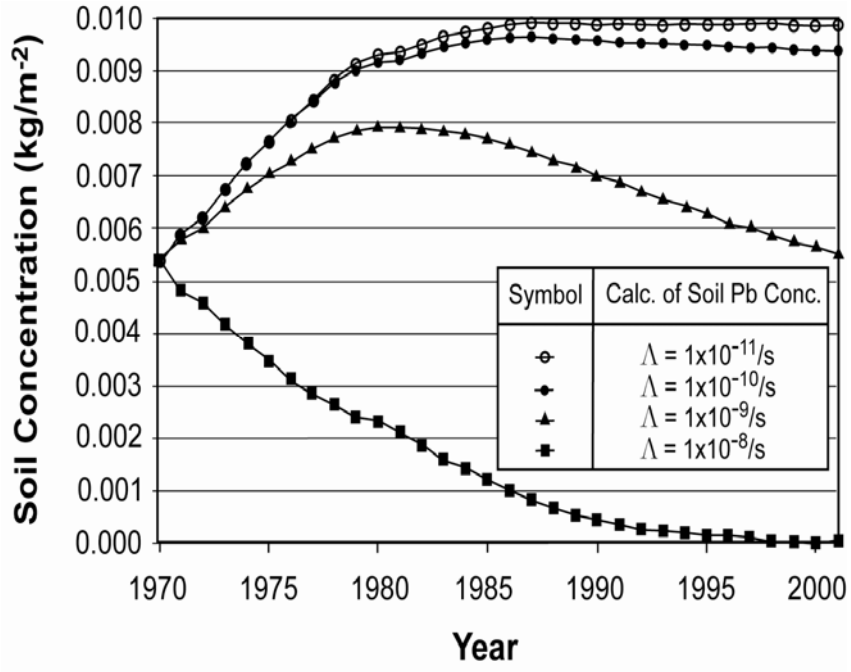
As discussed below (Section 2.7.2.1), findings to date indicate that those systems less influenced by current point sources are still responding to reduced Pb deposition rates associated with reduced atmospheric emissions of Pb, including those associated with the phase-out of leaded gasoline (see Section 2.3.2.3). Situations near point sources and those involving historically deposited Pb near roadways are less well characterized. Section 2.7.2.2 summarizes estimates of soil Pb concentrations since the time of the last review.

2.7.2.1 Temporal Trends

Variability among soil system characteristics that influence Pb mobility contributes to differences in current and projected temporal trends in soil concentrations (e.g., CD, pp. 3-18 to 3-19, Sections 3.2.1-3.2.2, and pp. AX7-33 to AX7-34).

Studies of forest soils have concluded that the time for soils to respond to reduced Pb deposition rates (e.g., associated with Pb gasoline phase-out) is shorter than previously believed. For example, Miller and Friedland (1994) projected that a 37% reduction in Pb concentration in northern hardwood and subalpine forest soils would occur within 17 years and 77 years, respectively. Kaste and Friedland (2003) traced atmospherically deposited Pb within forest soils in Vermont and found similar response times of 60 and 150 years for the two forest soils, respectively. They also concluded that the penetration of atmospherically delivered Pb in soils is currently limited to the upper 20 cm and that the heterogeneous distribution of Pb in soils would seem to indicate that the release of Pb to groundwater will be dispersed thereby reducing the likelihood of a large pulse to groundwater. This study and those of Wang and Benoit (1997), Johnson et al. (1995), and Zhang (2003) conclude that forest surface soils do not act as sinks under current deposition rates for Pb and that a gradual migration into mineral soils is occurring, making the possibility of a large pulse to groundwater in the future from past Pb pollution unlikely. Studies of the role of acidification in Pb mobility in sandy soils (e.g., NJ pine barrens), however, suggest a greater risk of mobilization of Pb and organic matter into these mineral soils, with subsequent inputs to associated stream waters (CD, p. AX7-91).

Studies in urban areas of southern California, where Pb has accumulated from past sources, suggests an environment in which Pb may remain at the soil surface (and other surfaces), contributing to air concentrations via resuspension for the near-term (CD, pp. 2-65 to 2-67 and 3-18 to 3-19). Figure 2-38 illustrates how the temporal trend in surface soil concentrations at a location may be influenced by the rate of resuspension. Harris and Davidson (2005) suggested that typical long-term values for resuspension rate fall in the range of 10^{-11} to 10^{-7} per second, based on wind speeds, with the range of 10^{-11} to 10^{-10} proposed as a range appropriate to California's south coast air basin. Under these assumptions, the model predicted that the occurrence of resuspension at this rate would lead to little to no reduction in soil Pb concentration in southern California over the next few hundred years (CD, pp. 2-65 to 2-67 and 3-18 to 3-20).



Source: Reprinted from Harris and Davidson (2005). Units for resuspension rate (Λ) are per second (/s).

Figure 2-38. Modeled soil concentrations of Pb in the South Coast Air Basin of California based on four resuspension rates (Λ).

Temporal trends in surface soils near established point sources are not well characterized. Information described in the CD for areas surrounding smelters after implementation of pollution controls, although showing declines in Pb concentrations in outdoor dustfall, street dust and indoor dustfall, has not indicated a noticeable decline in soil Pb concentrations (CD, pp. 3-23 to 3-24). Further, Pb concentrations in "clean" soil placed in areas influenced by current sources have been reported to exhibit increasing temporal trends (USEPA, 2006c). Concentrations of Pb in the very top layer of material (within the upper 1 inch of soil, analyzed using portable x-ray fluorescence) at locations less than a mile from a primary Pb smelter exhibited statistically significant increasing concentration over a four year period, with the average monthly change in Pb concentration ranging from 1 to 8 mg/kg (USEPA, 2001b, 2006c). Estimates of associated steady-state surface soil Pb concentrations or the expected longer-term temporal pattern for this situation have not been made.

2.7.2.2 Current Surface Soil Concentrations

Present concentrations of Pb in forest surface soils range from 40 to 100 mg/kg while natural background levels would be expected to be <1 mg/kg (CD, Section AX7.1.2.3). Urban and roadside soils and those in areas of long-term Pb emissions from point sources have much

higher concentrations of Pb, ranging up to hundreds to tens of thousands of mg/kg (CD, Section 3.2.1). For example, Pb surface soil concentrations near smelters have been found to range from thousands of mg/kg (dry weight) within approximately 100-250 meters, dropping to 200 mg/kg and below by distances of approximately 3-5 km (CD, Table 3-4). Soil Pb concentrations of 500-1100 mg/kg have been reported near U.S. mines that are no longer active (CD, Table 3-6).

2.8 SURFACE WATER AND SEDIMENT

The primary source of Pb in aquatic systems is atmospheric deposition. Lead is also carried into water bodies via wastewater effluent from municipalities and industry, stormwater runoff, erosion, and accidental discharges (CD, p. AX7-142). Most Pb occurring in aquatic systems is associated with particles, with the distribution between particle-bound and dissolved form being influenced by water chemistry as well as suspended sediment levels (CD, pp. AX7-117 to AX7-118; CD, Section AX7.2.2). The ratio of Pb in suspended solids to Pb in filtrate has been described to vary from 4:1 in rural streams to 27:1 in urban streams (CD, p. AX7-118).

Water columns have been described as “transient reservoirs” for pollutants (CD, p. 2-75). Once deposited to sediments, whether Pb is available for resuspension back into the water column with potential transport further down a watershed versus being buried into deeper sediments depends on the aquatic system. In open ocean waters (generally characterized by depth and distance from continental sources), resuspension to surface waters is unlikely. In more shallow systems, and additionally those influenced by land sources (e.g., stormwater runoff as well as point sources), resuspension may play a significant role in water column concentrations. For example, studies in San Francisco Bay, the southern arm of which as an average depth of 2 m, have indicated that Pb particles may be remobilized from surface sediments into the water column (CD, AX7-141).

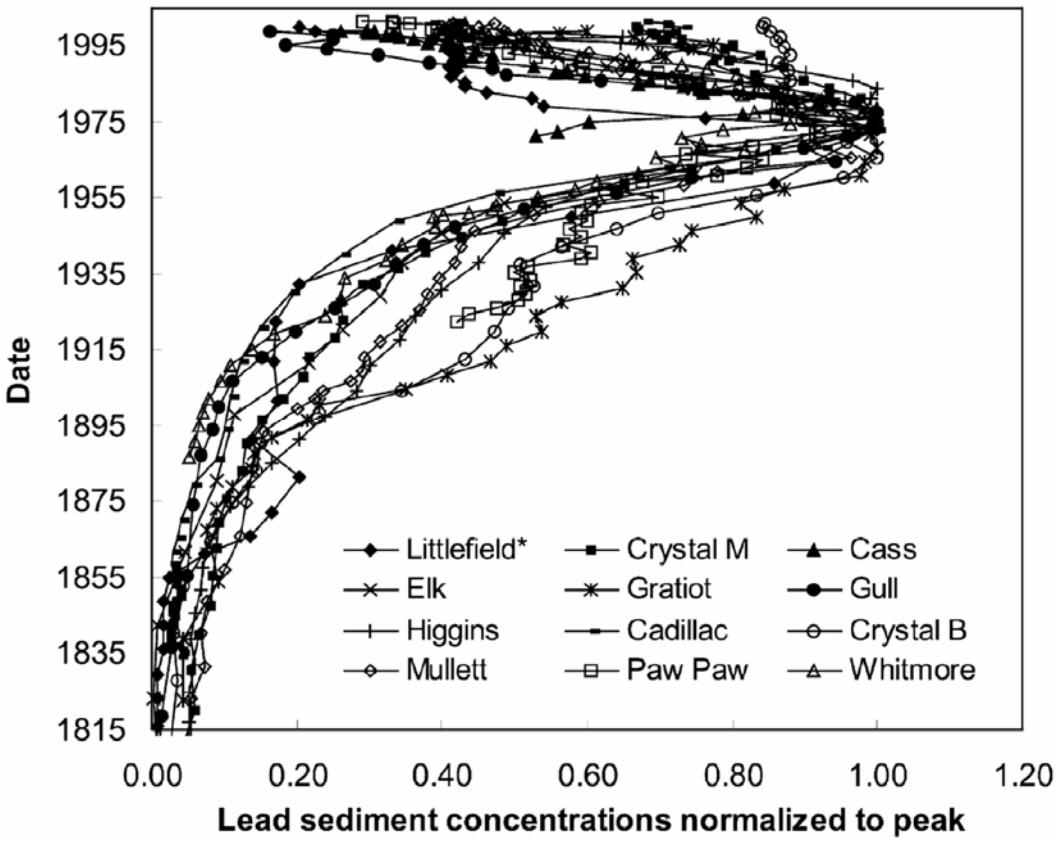
2.8.1 Temporal Trends

As discussed in the CD, many studies have investigated trends in Pb concentration in sediment and surface waters (CD, Section AX7.2.2), with declines documented in many systems and usually attributed to the phasing out of leaded gasoline.

Using sediment cores, temporal changes in Pb deposition and associated sediment Pb concentration have been documented. Sediment cores from the Okefenokee Swamp indicate that Pb concentrations were approximately 0.5 mg/kg prior to industrial development, reached a maximum of approximately 31 mg/kg from about 1935 to 1965, and following passage of the Clean Air Act in 1970 concentrations declined to about 18 mg/kg in 1990 (CD, p. AX7-141). Researchers investigating trends in metals concentrations (roughly from 1970-2001) in sediment cores from 35 reservoirs and lakes in urban and reference settings found that number of lakes

exhibiting decreasing trends in Pb concentration outnumbered increasing trends (83% versus 6%). Mass accumulation rates of Pb in cores, adjusted for background concentrations, decreased from the 1970s to the 1990s, with a median change of 246%. The largest decreases were found in lakes located in dense urban watersheds, although anthropogenic mass accumulation rates in dense urban lakes remained elevated over those in lakes in undeveloped watersheds, indicating that urban fluvial source signals can overwhelm those from regional atmospheric sources (CD, p. AX7-141; Mahler et al, 2006).

Figure 2-39 presents data on Pb concentrations in lake sediments from 12 lakes in the Great Lakes area. Consistent with other studies, this study showed a peak in Pb concentrations consistent with peak use of leaded gasoline in the U.S. in the mid 70's and declining concentrations in most lake sediments through the mid 1990's.



Source: Yohn et al. (2004).

Figure 2-39. Pb concentrations in sediment samples in 12 Michigan lakes. The concentrations are normalized by the peak Pb concentration in each lake; peak Pb concentrations ranged from approximately 50 to 300 mg/kg.

2.8.2 Current Concentrations

An analysis of data from the United States Geological Survey (USGS) National Water-Quality Assessment (NAWQA) program is described in the CD. The NAWQA data set encompasses data, collected over the past 15 years, on Pb concentrations in flowing surface waters, bed sediment, and animal tissue for more than 50 river basins and aquifers throughout the country (CD, Section AX7.2.2.3). Based on analysis of these data, the mean dissolved Pb concentration in ambient surface waters of the U.S. is estimated to be 0.66 μg/L (range 0.04 to 30 μg/L), as compared to a mean of 0.52 μg/L (range 0.04 to 8.4 μg/L) for the "natural" locations. The term "ambient" was used by NAWQA to describe the combined contribution of natural and anthropogenic sources, and a separate set of samples was identified for natural locations (e.g., "forest", "rangeland", and "reference" sites). The mean concentration of Pb in "ambient" bulk sediment (<63 microns, grain size) is 120 μg/g dry weight (range 0.5 to 12,000 μg/g), as compared to a mean of 109 μg/g dry weight (range 0.5 to 12,000 μg/g) for "natural" locations.

Geographic distribution of Pb concentrations in surface waters and sediments in this data set are presented in Figures 2-40 and 2-41 (CD, Figures AX7-2.2.7 and AX7-2.2.9). Areas with high surface water Pb concentrations were observed in Washington, Idaho, Utah, Colorado, Arkansas, and Missouri, with the maximum measured Pb concentration occurring at a site in Idaho with a land use classified as mining (CD, p. AX7-131). As was seen with surface water Pb concentrations, the highest measured sediment Pb concentrations were found in Idaho, Utah, and Colorado. And also similar to the surface water findings, seven of the top 10 sediment Pb concentrations recorded were measured at sites classified as mining land use (CD, p. AX7-133). As described in the CD, dissolved surface water concentrations reported for lakes have been generally much lower than the NAWQA values for lotic waters (CD, AX7-138).

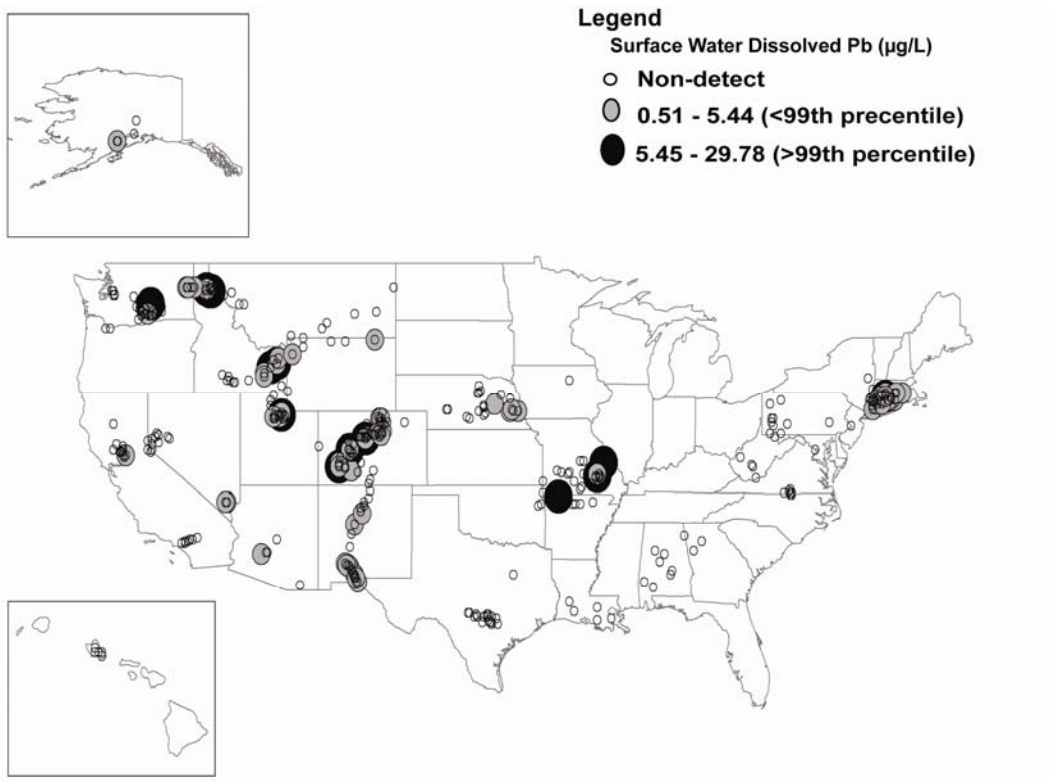


Figure 2-40. Spatial distribution of dissolved lead in surface water (N = 3445). [CD, Figure AX7-2.2.7.]

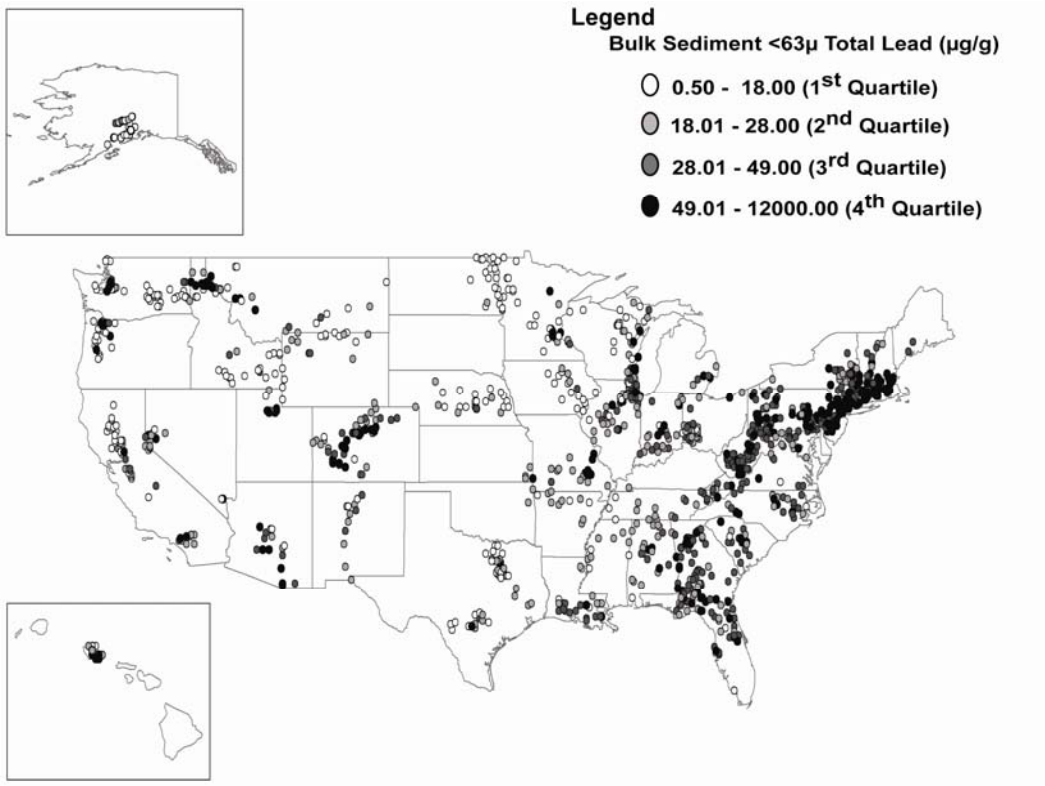


Figure 2-41. Spatial distribution of total lead in bulk sediment <63 µm (N = 1466). [CD, Figure AX7-2.2.9]

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3 POLICY-RELEVANT ASSESSMENT OF HEALTH EFFECTS EVIDENCE

3.1 INTRODUCTION

This chapter assesses key policy-relevant information on the known and potential health effects associated with exposure to ambient lead (Pb). The presentation here summarizes the qualitative assessment of health evidence contained in the CD, as a basis for the evidence-based assessment of primary standards for Pb presented in Chapter 5 of this document. The focus is on health endpoints associated with the range of exposures considered to be most relevant to current exposure levels. This presentation also gives particular attention to those endpoints for which there is quantitative health evidence available in this review that provides a foundation for the quantitative health risk assessment discussed in Chapter 4 and used in the risk-based assessment of primary standards for Pb presented in Chapter 5.

The presentation in this chapter recognizes several key aspects of the health evidence for Pb. First, because exposure to atmospheric Pb particles occurs not only via direct inhalation of airborne particles, but also via ingestion of deposited particles (e.g., associated with soil and dust), the exposure being assessed is multimedia and multi-pathway in nature, occurring via both the inhalation and ingestion routes. In fact, ingestion of indoor dust has been recognized as a significant Pb exposure pathway, particularly for young children (CD, p. 3-27 to 3-28). Second, the exposure index or dose metric most commonly used and associated with health effects information is an internal biomarker (i.e., blood Pb). Additionally, the exposure duration of interest (i.e., that influencing internal dose pertinent to health effects of interest) may span months to potentially years, as does the time scale of the environmental processes influencing Pb deposition and fate. Lastly, the nature of the evidence for the health effects of greatest interest for this review is epidemiological data strongly supported by toxicological data that provide biological plausibility and insights on mechanisms of action.

At the time of the last review, Pb was recognized to produce multiple effects in a variety of tissues and organ systems across a range of exposure levels, with blood Pb levels of 10-15 μg/dL being recognized as levels of concern for impaired neurobehavioral development in infants and children (USEPA, 1990). The current CD recognizes the existence of a wide array of Pb-induced deleterious effects, including several in children and/or adults that are induced by blood Pb levels extending well below 10 μg/dL, to below 5 μg/dL and possibly lower (CD, Section 8.4).

In recognition of the multi-pathway aspects of Pb, and use of an internal exposure metric in health risk assessment, Section 3.2 describes our understanding of the internal disposition or distribution of Pb, and the use of blood Pb as an internal exposure or dose metric. Section 3.3

discusses the nature of Pb-induced health effects, giving emphasis to those with the strongest evidence, particularly those associated with the range of current exposure levels. Potential impacts of Pb exposures on public health, including recognition of potentially susceptible or vulnerable subpopulations, are discussed in Section 3.4. Finally, Section 3.5 summarizes key policy-relevant conclusions about Pb-related health effects.

3.2 INTERNAL DISPOSITION – BLOOD LEAD AS DOSE METRIC

The health effects of Pb, discussed in the CD and summarized in Section 3.3 below, are remote from the portals of entry to the body (i.e., the respiratory system and gastrointestinal tract). Consequently, the internal disposition and distribution of Pb is an integral aspect of the relationship between exposure and effect. This section summarizes the current state of knowledge of Pb disposition pertaining to both inhalation and ingestion routes of exposure as described in the CD.

Inhaled Pb particles deposit in the different regions of the respiratory tract as a function of particle size (CD, pp. 4-3 to 4-4). Lead associated with smaller particles, which are predominantly deposited in the pulmonary region, may, depending on solubility, be absorbed into the general circulation or transported via phagocytic cells to the gastrointestinal tract (CD, pp. 4-3). Lead associated with larger particles, that are predominantly deposited in the head and conducting airways (e.g., nasal pharyngeal and tracheobronchial regions of respiratory tract), may be transported by mucociliary transport into the esophagus and swallowed, thus making its way to the gastrointestinal tract (CD, pp. 4-3 to 4-4), where it may be absorbed into the blood stream. Thus, Pb can reach the gastrointestinal tract either directional through the ingestion route or indirectly following inhalation.

The absorption efficiency of Pb from the gastrointestinal (GI) tract varies with particle size, as well as with the chemical form or matrix in which it is contained (CD, pp. 4-8 to 4-9). One line of evidence for this comes from research using animal models to estimate relative bioavailability (RBA) by comparing the absorbed fraction of ingested Pb for different test materials relative to that for a highly water-soluble form of Pb. Relative bioavailability of Pb from contaminated soils from different industrial sites (e.g., near Pb smelters, mines, etc), as assessed in such models, have been found to differ markedly, with RBA values ranging from 6 to 100% (CD, pp. 4-8 to 4-10; Casteel et al., 2006). As stated in the CD, “variations in size and mineral content of the Pb-bearing grains are the suspected cause of variations in the rate and extent of GI absorption of Pb” occurring in soil from different contaminated locations (CD, p. 4-9).

In addition to characteristics associated with the ingested Pb, GI absorption of Pb also varies with an individual’s physiology (e.g., maturity of the GI tract), and nutritional status (e.g.,

iron and calcium deficiency increases absorption), as well as the presence of food in the GI tract (CD, Section 4.2.1, pp. 4-5 to 4-8). With regard to GI tract maturity, estimates of Pb GI absorption reported in the past for young children (~40-50%) are higher than those reported for adults (CD, pp. 4-5 to 4-6). Several studies have reported that the presence of food in the GI tract reduces the absorption of water-soluble Pb (CD, p. 4-6). A contributing factor to this phenomenon is the presence of calcium, iron, and phosphate in the food, which depresses Pb absorption (CD, pp. 4-6 to 4-7). Animal studies have also indicated that Vitamin D, which regulates calcium absorption, enhances Pb absorption from the GI tract (CD, p. 4-7).

Once in the blood stream, where approximately 99% of the Pb associates with red blood cells, the Pb is quickly distributed throughout the body (e.g., within days) with the bone serving as a large, long-term storage compartment, and soft tissues (e.g., kidney, liver, brain, etc) serving as smaller compartments, in which Pb may be more mobile (CD, Sections 4.3.1.4 and 8.3.1.). Additionally, the epidemiologic evidence indicates that Pb freely crosses the placenta resulting in continued fetal exposure throughout pregnancy, and that exposure increases during the later half of pregnancy (CD, Section 6.6.2).

During childhood development, bone represents approximately 70% of a child's body burden of Pb, and this accumulation continues through adulthood, when more than 90% of the total Pb body burden is stored in the bone (CD, Section 4.2.2). Accordingly, levels of Pb in bone are indicative of a person's long-term, cumulative exposure to Pb. In contrast, blood Pb levels are usually indicative of recent exposures. Depending on exposure dynamics, however, blood Pb may – through its interaction with bone - be indicative of past exposure or of cumulative body burden (CD, Section 4.3.1.5).

Throughout life, Pb in the body is exchanged between blood and bone, and between blood and soft tissues (CD, Section 4.3.2), with variation in these exchanges reflecting "duration and intensity of the exposure, age and various physiological variables" (CD, p. 4-1). For example, resorption of bone (e.g., in pregnant or nursing women, and associated with osteoporosis in postmenopausal women or, to a lesser magnitude, in older men) results in a mobilization of Pb from bone into circulation (CD, Sections 4.3.2.4 and 4.3.2.5). Past exposures that contribute Pb to the bone, consequently, may influence current levels of Pb in blood. Where past exposures were elevated in comparison to recent exposures, this influence may complicate interpretations with regard to recent exposure (CD, Sections 4.3.1.4 to 4.3.1.6). That is, higher blood Pb concentrations may be indicative of higher cumulative exposures or of a recent elevation in exposure (CD, pp. 4-34 and 4-133).

In several recent studies investigating the relationship between Pb exposure and blood Pb in children (e.g., Lanphear and Roghmann 1997; Lanphear et al., 1998), blood Pb levels have been shown to reflect Pb exposures, with particular influence associated with exposures to Pb in

surface dust. Further, as stated in the CD "these and other studies of populations near active sources of air emissions (e.g., smelters, etc.), substantiate the effect of airborne Pb and resuspended soil Pb on interior dust and blood Pb" (CD, p. 8-22).

As mentioned earlier, blood Pb, although subject to influence by Pb levels in all organs and tissues, is generally described as reflecting recent exposures (CD, Section 4.3.1.4). Inhaled or ingested Pb quickly enters the blood, and Pb in the blood is available for exchange with the soft and skeletal tissues, conceptually viewed as the fast (half-life of ~28 days) and slow (half-life may be decades in adults) turnover pools, respectively (CD, Section 4.3.1.4). Simulations using biokinetic models indicate that blood Pb levels in adults achieve a new quasi-steady state within 75-100 days (approximately 3-4 times the blood elimination half-life) subsequent to abrupt increases in Pb uptake (CD, pp. 4-25 to 4-26). Similar models indicate a quicker response of blood Pb levels in children (CD, p. 4-27 and Figure 4-5). Additionally, response of the blood to reduction of a relatively brief Pb exposure appears to be faster than for an exposure of several years, with estimated half-lives of approximately 9 months as compared to 30 months for the longer exposure response (CD, pp. 4-25 to 4-26).

Blood Pb levels are extensively used as an index or biomarker of exposure by national and international health agencies, as well as in epidemiological (CD, Sections 4.3.1.3 and 8.3.2) and toxicological studies of Pb health effects and dose-response relationships (CD, Chapter 5). The prevalence of the use of blood Pb as an exposure index or biomarker is related to both the ease of blood sample collection (CD, p. 4-19; Section 4.3.1) and by findings of association with a variety of health effects (CD, Section 8.3.2). As noted above, blood Pb levels respond to elevations in exposure. Accordingly, the U.S. Centers for Disease Control and Prevention (CDC), and its predecessor agencies, have for many years used blood Pb level as a metric for identifying children at risk of adverse health effects and for specifying particular public health recommendations (CDC, 1991; CDC, 2005). In 1978, when the current Pb NAAQS was established, the CDC recognized a blood Pb level of 30 μg/dL as a level warranting individual intervention (CDC, 1991). In 1985, the CDC recognized a level of 25 μg/dL for individual child intervention, and in 1991, they recognized a level of 15 μg/dL for individual intervention and a level of 10 μg/dL for implementing community-wide prevention activities (CDC, 1991; CDC, 2005). In 2005, with consideration of a review of the evidence by their advisory committee, CDC revised their statement on *Preventing Lead Poisoning in Young Children*, specifically recognizing the evidence of adverse health effects in children with blood Pb levels below 10

μg/dL and the data demonstrating that no "safe" threshold for blood Pb had been identified, and emphasizing the importance of preventive measures (CDC, 2005).[1]

Since 1976, the CDC has been monitoring blood Pb levels nationally through the National Health and Nutrition Examination Survey (NHANES). This survey has documented the dramatic decline in mean blood Pb levels in the U.S. population that has occurred since the 1970s and that coincides with regulations regarding leaded fuels, leaded paint, and Pb-containing plumbing materials that have reduced Pb exposure among the general population (CD, Sections 4.3.1.3 and 8.3.3; Schwemberger et al., 2005). Although levels in the U.S. general population, including geometric mean levels in children aged 1-5, have declined, mean levels have been found to differ among children of different socioeconomic status (SES) and other demographic characteristics (CD, p. 4-21). The health effects associated with blood Pb levels are extensively discussed in the CD, while those of particular policy relevance for this review are summarized in subsequent subsections of this chapter.

Bone measurements, as a result of the generally slower Pb turnover in bone, are recognized as providing a better measure of cumulative Pb exposure (CD, Section 8.3.2). The bone pool of Pb in children, however, is thought to be much more labile than that in adults due to the more rapid turnover of bone mineral as a result of growth (CD, p. 4-27). As a result, changes in blood Pb concentration in children more closely parallel changes in total body burden (CD, pp. 4-20 and 4-27). This is in contrast to adults, whose bone has accumulated decades of Pb exposures (with past exposures often greater than current ones), and for whom the bone may be a significant source long after exposure has ended (CD, Section 4.3.2.5).

Given the association with exposure, particularly recent exposure, and the relative ease of collection, blood Pb levels are extensively used as an index or biomarker of exposure by national and international health agencies (CD, Section 4.3.1.5). Although recent methods are making bone Pb measurements easier to collect (CD, Section 4.3.2.2) and consequently, their use more widespread, epidemiological and toxicological studies of Pb health effects and dose-response relationships tend to be dominated by blood Pb as the exposure metric (CD, Sections 4.3.1.3, 8.3.2 and Chapter 5).

Accordingly, blood Pb level is the index of exposure or exposure metric in this risk assessment. The use of concentration-response functions that rely on blood Pb (e.g., rather than

[1] With the 2005 statement, CDC identified a variety of reasons, reflecting both scientific and practical considerations, for not lowering the 1991 level of concern, including a lack of effective clinical or public health interventions to reliably and consistently reduce blood Pb levels that are already below 10 μg/dL, the lack of a demonstrated threshold for adverse effects, and concerns for deflecting resources from children with higher blood Pb levels (CDC, 2005).

ambient Pb concentration) as the exposure metric reduces uncertainty in the causality aspects of Pb risk estimates. The relationship between specific sources and pathways of exposure and blood Pb level is needed, however, in order to identify the specific risk contributions associated with those sources and pathways of greatest interest to this assessment (i.e., those related to Pb emitted into the air). For example, the blood Pb-response relationships developed in epidemiological studies of Pb-exposed populations do not distinguish among different sources or pathways of Pb exposure (e.g., inhalation, ingestion of indoor dust, ingestion of dust containing leaded paint). In the exposure assessment for this review, models that estimate blood Pb levels associated with Pb exposure (e.g., CD, Section 4.4) are used to inform estimates of contributions to blood Pb arising from ambient air related Pb as compared to contributions from other sources.

3.3 NATURE OF EFFECTS

Lead has been demonstrated to exert "a broad array of deleterious effects on multiple organ systems via widely diverse mechanisms of action" (CD, p. 8-24 and Section 8.4.1). This array of health effects, the evidence for which is comprehensively described in the CD, includes

- Heme biosynthesis and related functions;
- Neurological development and function;
- Reproduction and physical development;
- Kidney function;
- Cardiovascular function; and,
- Immune function.

There is also some evidence of Pb carcinogenicity, primarily from animal studies, with limited human evidence of suggestive associations (CD, Sections 5.6.2, 6.7, and 8.4.10). [2]

This review is focused on those effects most pertinent to ambient exposures. Given the reductions in ambient Pb levels over the past 30 years, these effects are generally those associated with the lowest levels of Pb exposure. These are neurological, hematological and immune effects for children, and neurological, hematological, cardiovascular and renal effects for adults (See Tables 3-1 and 3-2), with neurological effects in children and cardiovascular effects in adults appearing to be of greatest public health concern (CD, p. 8-60). The toxicological and epidemiological information available since the time of the last review "includes assessment of new evidence substantiating risks of deleterious effects on certain health

[2] Lead has been classified as a probable human carcinogen by the International Agency for Research on Cancer, based mainly on sufficient animal evidence, and as reasonably anticipated to be a human carcinogen by the U.S. National Toxicology Program (CD, Section 6.7.2). U.S. EPA classified it in the past as a probable carcinogen (http://www.epa.gov/iris/subst/0277.htm).

endpoints being induced by distinctly lower than previously demonstrated Pb exposures indexed by blood Pb levels extending well below 10 μg/dL in children and/or adults" (CD, p. 8-25). Tables 3-1 and 3-2 (CD, Tables 8-5 and 8-6) indicate some health effects associated with blood Pb levels that extend below 5 ug/dL, and use the notation "(???)" to indicate that some studies have observed these effects at the lowest blood levels considered. That is, threshold levels for these effects cannot be discerned from the currently available studies.

The endpoints identified above and included in Tables 3-1 and 3-2 are important considerations for this review and are described briefly in sections below, while detailed discussion of the evidence is presented in the CD.

Table 3-1. Summary of Lowest Observed Effect Levels for Key Lead-Induced Health Effects in Children (reproduced from CD, Table 8-5).

Lowest Observed Effect Blood Lead Level	Neurological Effects	Hematological Effects	Immune Effects
30 µg/dL		Increased urinary δ-aminolevulinic acid	
15 µg/dL	Behavioral disturbances (e.g., inattention, delinquency) Altered electrophysiological responses	Erythrocyte protoporphyrin (EP) elevation	
10 µg/dL	Effects on neuromotor function CNS cognitive effects (e.g., IQ deficits)	Inhibition of δ-aminolevulinic acid dehydratase (ALAD) Pyrimidine-5′-nuclotidase (Py5N) activity inhibition	Effects on humoral (↑ serum IgE) and cell-mediated (↓ T-cell abundance) immunity
5 µg/dL	↓	↓	
	(???)	(???)	
0 µg/dL			

Note: Arrows depict cases where weight of overall evidence strongly substantiates likely occurrence of type of effect in association with blood-Pb concentrations in range of 5-10 µg/dL, or possibly lower, as implied by (???). Although no evident threshold has yet been clearly established for those effects, the existence of such effects at still lower blood-Pb levels cannot be ruled out based on available data.

Source: Adapted/updated from Table 1-17 of U.S. Environmental Protection Agency (1986a).

Table 3-2. Summary of Lowest Observed Effect Levels for Key Lead-Induced Health Effects in Adults (reproduced from CD, Table 8-6).

Lowest Observed Effect Blood Lead Level	Neurological Effects	Hematological Effects	Cardiovascular Effects	Renal Effects
30 µg/dL	Peripheral sensory nerve impairment	Erythrocyte protoporphyrin (EP) elevation in males		Impaired Renal Tubular Function
20 µg/dL	Cognitive impairment			
15 µg/dL	Postural sway	Erythrocyte protoporphyrin (EP) elevation in females Increased urinary δ-aminolevulinic acid		
10 µg/dL		Inhibition of δ-aminolevulinic acid dehydratase (ALAD)	Elevated blood pressure	
5 µg/dL			↓ (???)	Elevated serum creatine (↓ creatine clearance)
0 µg/dL				

Note: Arrows depict cases where weight of overall evidence strongly substantiates likely occurrence of type of effect in association with blood-Pb concentrations in range of 5-10 µg/dL, or possibly lower, as implied by (???). Although no evident threshold has yet been clearly established for those effects, the existence of such effects at still lower blood-Pb levels cannot be ruled out based on available data.

Source: Adapted/updated from Table 1-16 of U.S. Environmental Protection Agency (1986a).

3.3.1 Developing Nervous System

The nervous system has long been recognized as a target of Pb toxicity, with the developing nervous system affected at lower exposures than the mature system (CD, Sections 5.3, 6.2.1, 6.2.2, and 8.4). While blood Pb levels in U.S. children ages one to five years have decreased notable since the late 1970s, newer studies have investigated and reported associations of effects on the neurodevelopment of children with these more recent blood Pb levels (CD, Chapter 6). Functional manifestations of Pb neurotoxicity during childhood include sensory, motor, cognitive and behavioral impacts. Numerous epidemiological studies have reported neurocognitive, neurobehavioral, sensory, and motor function effects in children at blood Pb levels below 10 μg/dL (CD, Section 6.2). Further, "extensive experimental laboratory animal evidence has been generated that (a) substantiates well the plausibility of the epidemiologic findings observed in human children and adults and (b) expands our understanding of likely mechanisms underlying the neurotoxic effects" (CD, p. 8-25; Section 5.3).

Cognitive effects associated with Pb exposures that have been observed in epidemiological studies have included decrements in intelligence test results, such as the widely used IQ score, and in academic achievement as assessed by various standardized tests as well as by class ranking and graduation rates (CD, Section 6.2.16 and pp 8-29 to 8-30). As noted in the CD with regard to the latter, "Associations between Pb exposure and academic achievement observed in the above-noted studies were significant even after adjusting for IQ, suggesting that Pb-sensitive neuropsychological processing and learning factors not reflected by global intelligence indices might contribute to reduced performance on academic tasks" (CD, pp 8-29 to 8-30).

Other cognitive effects observed in studies of children have included effects on attention, executive functions, language, memory, learning and visuospatial processing (CD, Sections 5.3.5, 6.2.5 and 8.4.2.1), with attention and executive function effects associated with Pb exposures indexed by blood Pb levels below 10 μg/dL (CD, Section 6.2.5 and pp. 8-30 to 8-31). The evidence for the role of Pb in this suite of effects includes experimental animal findings (discussed in CD, Section 8.4.2.1; p. 8-31), which provide strong biological plausibility of Pb effects on learning ability, memory and attention (CD, Section 5.3.5), as well as associated mechanistic findings. Further, Pb-induced deficits observed in animal and epidemiological studies, for the most part, have been found to be persistent in the absence of markedly reduced environmental exposures (CD, Sections 5.3.5, 6.2.11, and 8.5.2). It is additionally important to note that there may be long-term consequences of such deficits over a lifetime. Poor academic skills and achievement can have "enduring and important effects on objective parameters of

success" later in life, as well as increased risk of antisocial and delinquent behavior (CD, Section 6.2.16).

Other neurological effects associated with Pb exposures indexed by blood Pb levels near or below 10 μg/dL include behavioral effects, such as delinquent behavior (CD, Sections 6.2.6 and 8.4.2.2), sensory effects, such as those related to hearing and vision (CD, Sections 6.2.7, 7.4.2.3 and 8.4.2.3), and deficits in neuromotor function (CD, p. 8-36).

Neurocognitive impact, specifically decrement in IQ in young children, is a focus of the quantitative risk assessment due to the strength of evidence for association with blood Pb levels below 10 μg/dL, and the strength of the dose-response information at these exposure levels.

As discussed in the CD (Section 8.4.2) and by Rice (1996), while there is no direct animal test parallel to human IQ tests, "in animals a wide variety of tests that assess attention, learning, and memory suggest that Pb exposure {of animals} results in a global deficit in functioning, just as it is indicated by decrements in IQ scores in children" (CD, p. 8-27). The animal and epidemiological evidence for this endpoint are consistent and complementary (CD, p. 8-44). As stated in the CD (p. 8-44):

> *Findings from numerous experimental studies of rats and of nonhuman primates, as discussed in Chapter 5, parallel the observed human neurocognitive deficits and the processes responsible for them. Learning and other higher order cognitive processes show the greatest similarities in Pb-induced deficits between humans and experimental animals. Deficits in cognition are due to the combined and overlapping effects of Pb-induced perseveration, inability to inhibit responding, inability to adapt to changing behavioral requirements, aversion to delays, and distractibility. Higher level neurocognitive functions are affected in both animals and humans at very low exposure levels (≤10 μg/dL), more so than simple cognitive functions.*

Further, "epidemiologic studies of Pb and child development have demonstrated inverse associations between blood Pb concentrations and children's IQ and other outcomes at successively lower Pb exposure levels" over the past 30 years (CD, p. 6-64). This is supported by multiple studies performed over the past 15 years (see CD, Section 6.2.13), with particularly compelling evidence for decrements in IQ at blood Pb levels below 10 μg/dL provided by a recent international pooled analysis of seven prospective studies (Lanphear et al., 2005; CD, Section 6.2.13). For example, this pooled analysis estimated a decline of 6.2 points (with a 95% confidence interval bounded by 3.8 and 8.6) in full scale IQ occurring between approximately 1 and 10 μg/dL blood Pb level, measured concurrent with the IQ test (CD, p. 6-76). This analysis (Lanphear et al., 2005) is relied upon in the quantitative risk assessment for this endpoint.

3.3.2 Adult Nervous System

The nervous system has long been recognized as a target of Pb toxicity (CD Sections 5.3.1, 8.4.2). For example, those chronically exposed in the workplace are at risk for various neurological effects including peripheral sensory nerve impairment, visuomotor and memory impairment, and postural sway abnormalities, with a blood Pb concentration >14 μg/dL being a possible threshold (CD, p. 6-87). Past occupational exposure also increases the risk of developing amyotrophic lateral sclerosis and motor neuron disease (CD, Section 6.3.5 and p. 6-87). Essential tremor is also associated with Pb exposures, particularly for those with genetic susceptibility (CD, Sections 6.3.5 and 6.3.6 and p. 6-86).

In elderly populations, significant associations have been reported between bone Pb levels and impaired cognitive performance or dysfunction (CD, Section 6.3.3 and 6.3.3.1), but not with blood Pb levels, perhaps indicating a role of cumulative and/or past Pb exposures (CD, p. 6-83). During demineralization of bone in the elderly, Pb may be released into the blood, thus augmenting blood Pb associated with current ambient exposures (CD, Section 4.3.2.4). An increased susceptibility among the elderly to Pb effects on cognitive function is supported by animal evidence (Section 5.3.7). With lifetime exposure, senescent animals have exhibited an increased susceptibility to Pb, due to the increased exposure from bone resorption, and an apparently greater sensitivity to the biochemical effects of Pb (CD, Section 5.3.7). Laboratory animal research in rats and monkeys also indicates a potential for cognitive function effects in the elderly to be related to physiological effects (regulation of protein thought to play a role in Alzheimer's disease) of Pb exposures in early childhood (CD, p. 5-67; Basha et al., 2005). Thus, early life exposure to Pb may contribute to neurocognitive effects later in life due to the redistribution of Pb body burden from bone to brain and by enhanced susceptibility caused by age-related degenerative changes in various organs, including brain (CD, p. 8-40).

3.3.3 Cardiovascular System

Epidemiologic and experimental toxicology studies provide strong support for the relationship between Pb exposure and increased adverse cardiovascular outcomes, including increased blood pressure, increased incidence of hypertension, and cardiovascular morbidity and mortality (CD, Sections 5.5, 6.5 and 8.4.3). The cardiovascular effect most frequently examined in epidemiological studies is increased systolic blood pressure in adults, which has been repeatedly associated with Pb exposure (CD, Sections 8.4.3, 8.6.3, 6.5.2.3, and 6.5.7). The association has been observed with Pb levels in bone and also, in some cohorts, with Pb in blood (including blood Pb levels below 10 μg/dL). A recent meta-analysis by Nawrot and others (2005), that included a range of blood Pb levels from 2.3 to 63.8 μg/dL, reported an association of increased systolic blood pressure and decreased diastolic pressure with increased blood Pb

level, including levels below 10 μg/dL. The magnitude of change observed has considerable significance at the population level (CD, p. 8-45, Section 8.6.3). The epidemiological evidence is supported by evidence in numerous animal studies of arterial hypertension with low Pb exposures, an effect that persists in animals long after cessation of exposure (CD, Sections 5.5 and 8.4.3).

Multiple studies reporting positive associations of blood pressure and hypertension with bone Pb levels highlight the important role of cumulative past Pb exposure in development of cardiovascular health effects (Sections 6.5.2.3 and 6.5.7). A study of young adults who lived as children in an area of high Pb exposures also indicates the potential role of childhood exposure. In this study, higher bone Pb levels were associated with higher systolic and diastolic blood pressure (CD, p. 6-138), while current blood Pb levels (mean of 2.2 μg/dL) were not (CD, p. 6-124).

Systolic blood pressure exerts a strong influence on more serious cardiovascular events by its role in hypertension and its adverse cardiovascular sequelae (CD, p. 8-83). Several analyses of National Health and Nutrition Examination Survey (NHANES) cohorts, including some recently released, have collectively suggested a "significant effect of Pb on cardiovascular mortality in the general U.S. population" (CD, p. 8-88, Sections 6.5.3.2 and 8.6.3). For example recent analyses of NHANES blood Pb data from 1976 to 1980 and 1988 to 1994 provide supportive evidence for an increased risk of cardiovascular mortality, consistent with projected likely increases in serious cardiovascular events (stroke, heart attack) resulting from Pb-induced increases in blood pressure (CD, Section 8.6.3).

3.3.4 Renal System

Lead nephrotoxicity is mediated by alterations in the glomerular filtration rate (CD, Sections 5.7.3 and 8.4.5). The interaction of Pb with the kidney, including occurrences and mechanisms of Pb uptake by and accumulation in the kidney, and associated cellular alterations, is well described in animal research (CD, Section 5.7). A set of screening tests involving markers of nephrotoxic effects have been established for screening individuals exposed to Pb occupationally or environmentally (CD, Section 5.7.1). In the epidemiological literature, associations between blood Pb and indicators of renal function impairment (e.g., measures of glomerular integrity, such as creatinine levels in urine) have been found at blood Pb levels extending below 10 μg/dL, to as low as ~2 to 4 μg/dL (CD, Sections 6.4.4.1.5 and 8.4.5). Associations are also observed with cumulative Pb dose, assessed via bone Pb, and longitudinal renal function decline (CD, p. 6-94), indicating the potential role of earlier exposures.

The findings for non-occupational populations since the last review provide "strong evidence that renal effects occur at much lower blood Pb levels than previously recognized"

(CD, p. 6-113). These findings of lower Pb renal effects thresholds in environmental compared to occupational research may be a result of potentially larger proportions of susceptible individuals in the general population as compared to occupational cohorts (CD, p. 6-107). The data available are not sufficient to determine whether these effects are related more to current blood-Pb levels, higher levels from past exposure, or both (CD, p. 8-49).

The findings regarding Pb exposures and renal effects are of particular concern with regard to certain susceptible subpopulations. At levels of exposure in the general U.S. population overall, Pb combined with other risk factors, such as diabetes, hypertension, or chronic renal insufficiency from causes unrelated to Pb, can result in clinically relevant effects. Notably, the size of such susceptible populations is increasing in the United States due to obesity (CD, p. 6-113). That is, Pb is recognized as acting cumulatively with other renal risk factors to cause early onset of renal insufficiency and/or a steeper rate of renal function decline in individuals already at risk for renal disease (CD, p. 6-107).

3.3.5 Heme Synthesis

It has long been recognized that Pb exposure is associated with disruption of heme synthesis in both children and adults. The evidence regarding effects on heme synthesis and other hematological parameters in animal and humans is strong, and includes documented quantitative relationships between exposure and effects in children and adults. Interference with heme synthesis was identified as one of the targets of low-level Pb toxicity in children during the time of the last NAAQS review (USEPA, 1990), and was the primary focus for the initial setting of the Pb NAAQS in 1978 (USEPA, 1978).

Mechanisms associated with Pb interference with heme synthesis include inhibition of the enzymes ALAD and ferrochelatase (Table 3-1; CD Sections 5.2.1, 6.9.1, 6.9.2; USEPA 1986). Inhibition of ALAD has been associated with increased blood Pb concentrations at and somewhat below 10 μg/dL, in children and adults (Tables 3-1 and 3-2; CD, Table 6-7). Blood Pb concentrations at and above approximately 15 μg/dL, in children, and 15-30 μg/dL, in adults, are associated with elevation of erythrocyte protoporphyrin (EP), and notable reductions in hemoglobin synthesis (Tables 3-1 and 3-1; CD, p. 8-47; USEPA, 1986). In the setting of the Pb NAAQS in 1978, the Agency concluded that "the state of elevated EP must be regarded as potentially adverse to the health of young children" (USEPA, 1978). Blood Pb concentrations at and above 40 ug/dL are associated with frank anemia, a clinical sign of severe Pb poisoning (CD, p. 8-47). The evidence regarding Pb disruption of heme synthesis and associated mechanisms is presented in detail in past CDs (USEPA 1986, 1977), with more recent findings, including the role of genetic polymorphisms, discussed in the current CD (Sections 8.4.4, 5.2.1, 6.9.1 and 6.9.2).

3.3.6 Immune System

Since the time of the last review, there has been substantial research on the immunotoxicity of Pb. As summarized in the CD, "studies across humans and a variety of animal models are in general agreement concerning both the nature of the immunotoxicity induced by Pb as well as the exposure conditions that are required to produce immunomodulation" (CD, p. 5-244, Section 5.9). Lead is distinguished from other immunotoxicants, however, by the fact that the most sensitive biomarkers of its immunotoxicity are associated with specific functional capacities that influence risk of disease, as opposed to being associated with changes in immune cell numbers or pathological changes of lymphatic system organs (CD, Section 5.9.1). The main immune system targets of Pb are macrophages and T lymphocytes, leading to a potential for increased tissue inflammation, reduced cell-mediated immunity, and increased risk of autoimmunity (See CD, Figure 5-18, Section 5.9.11). Additionally, Pb exposures in both animal and human studies are associated with increased production of IgE, an immunoglobulin involved in allergic responses and asthma (CD, Section 5.9.3.2). These effects are supported by evidence in neonatal and juvenile animals and have been reported in epidemiologic studies of children at blood Pb levels extending below 10 μg/dL (CD, p. 6-197 and Sections 5.9.10 and 8.4.6).

3.4 LEAD-RELATED IMPACTS ON PUBLIC HEALTH

In addition to the advances in our knowledge and understanding of Pb health effects at lower exposures (e.g., using blood Pb as the index), there has been some change with regard to the U.S. population Pb burden since the time of the last Pb NAAQS review. For example, the geometric mean blood Pb level for U.S. children aged 1-5, as estimated by the U.S. Centers for Disease Control, declined from 2.7 μg/dL (95% CI: 2.5-3.0) in the 1991-1994 survey period to 1.7 μg/dL (95% CI: 1.55-1.87) in the 2001-2002 survey period (CD, Section 4.3.1.3).[3] Blood Pb levels have also declined in the U.S. adult population over this time period (CD, Section 4.3.1.3). These observation however, should not be interpreted to mean that blood Pb levels declined in all communities, or uniformly by this amount. As noted in the CD, "blood-Pb levels have been declining at differential rates for various general subpopulations, as a function of income, race, and certain other demographic indicators such as age of housing" (CD, p. 8-21).

The following discussion draws from the CD to characterize subpopulations potentially at risk for Pb-related effects and potential public health impacts associated with exposure to ambient Pb.

[3] These levels are in contrast to the geometric mean blood Pb level of 14.9 μg/dL reported for U.S. children (aged 6 months to 5 years) in 1976-1980 (CD, Section 4.3.1.3).

3.4.1 At-risk Subpopulations

In considering at-risk subpopulations, we considered evidence regarding those with increased susceptibility (i.e., physiological factors contributing to a greater response for the same exposure), and those with increased exposure (including that resulting from behavior leading to increased contact with contaminated media). A behavioral factor of great impact on Pb exposure is the incidence of hand-to-mouth activity that is prevalent in very young children (CD, Section 4.4.3). Physiological factors include both conditions contributing to a subgroup's increased risk of effects at a given blood Pb level, and those that contribute to blood Pb levels higher than those otherwise associated with a given Pb exposure (CD, Section 8.5.3). We also considered evidence pertaining to vulnerability to pollution-related effects which additionally encompasses situations of elevated exposure, such as residing in old housing with Pb-containing paint or near sources of ambient Pb, as well as socioeconomic factors, such as reduced access to health care or low socioeconomic status (SES) (USEPA, 2003, 2005) that can contribute to increased risk of adverse health effects from Pb.

Three particular physiological factors contributing to increased risk of Pb effects at a given blood Pb level are recognized in the CD (e.g., CD, Section 8.5.3): age, health status, and genetic composition (or genotype). With regard to age, the susceptibility of young children to the neurodevelopmental effects of Pb is well recognized (e.g., CD, Sections 5.3, 6.2, 8.4, 8.5, 8.6.2), although the specific ages of vulnerability have not been established (CD, pp 6-60 to 6-64). Early childhood may also be a time of increased susceptibility for Pb immunotoxicity (CD, Sections 5.9.10, 6.8.3 and 8.4.6), and childhood exposures have been associated with increased risk of cardiovascular and neurodegenerative effects in adulthood (CD, p. 8-74). Health status is another physiological factor in that subpopulations with pre-existing health conditions may be more susceptible (as compared to the general population) for particular Pb-associated effects, with this being most clear for renal and cardiovascular outcomes. For example, African Americans as a group, have a higher frequency of hypertension than the general population or other ethnic groups (NCHS, 2005), and as a result may face a greater risk of adverse health impact from Pb-associated cardiovascular effects. A third physiological factor relates to genetic polymorphisms. That is, subpopulations defined by particular genetic polymorphisms (e.g., presence of the δ-aminolevulinic acid dehydratase-2 [ALAD-2] allele) have also been recognized as sensitive to Pb toxicity, which may be due to increased susceptibility to the same internal dose and/or to increased internal dose associated with same exposure (CD, p. 8-71, Sections 6.3.5, 6.4.7.3 and 6.3.6).

While early childhood is recognized as a time of increased susceptibility, a difficulty in identifying a discrete period of susceptibility from epidemiological studies has been that the period of peak exposure, reflected in peak blood Pb levels, is around 18-27 months when hand-

to-mouth activity is at its maximum (CD, p. 6-60). The earlier Pb literature described the first 3 years of life as a critical window of vulnerability to the neurodevelopmental impacts of Pb (CD, p. 6-60). Recent epidemiologic studies, however, have indicated a potential for susceptibility of children to concurrent Pb exposure extending to school age (CD, pp. 6-60 to 6-64). It may be that the influence of concurrent blood Pb (and exposures contributing to it) remains important until school age with regard to the potential to affect cognitive development (CD, pp. 6-63 to 6-64; Chen et al., 2005). The evidence indicates both the sensitivity of the first 3 years of life, and a sustained sensitivity throughout the lifespan as the human central nervous system continues to mature and be vulnerable to neurotoxicants (CD, Section 8.4.2.7). The animal evidence supports our understanding of specific periods of development with increased vulnerability to specific types of effect (CD, Section 5.3), and indicates a potential importance of exposures of duration on the order of months. Evidence of a differing sensitivity of the immune system to Pb across and within different periods of life stages indicates that Pb exposures of duration as short as weeks to months may contribute to some effects. For example, the animal evidence suggests that the gestation period is the most sensitive life stage followed by early neonatal stage, and within these life stages, critical windows of vulnerability are likely to exist (CD, Section 5.9 and p. 5-245).

Several physiological factors pertain to susceptibility by contributing to increased blood Pb levels (i.e., increased internal dose levels) over those otherwise associated with a given Pb exposure (CD, Section 8.5.3). These include nutritional status, which plays a role in Pb absorption from the GI tract (CD, Section 5.10.2.5); polymorphism for the vitamin D receptor, which studies suggest may contribute to increased Pb absorption from the GI tract (CD, Section 8.4.2.7); presence of the ALAD-2 allele, which studies suggest contribute to increased blood Pb levels (Section 8.5.3); and bone demineralization, such as occurs during pregnancy, lactation, and aging, which appears to influence Pb release from bone into the blood (CD, Section 4.3.2).

In summary, there are a variety of ways in which Pb exposed populations might be characterized and stratified for consideration of public health impacts. Age or lifestage was used to distinguish potential groups on which to focus the quantitative risk assessment (Chapter 4) in recognition of its influence on exposure and susceptibility, and young children were selected as the priority population for the risk assessment in consideration of the health effects evidence regarding endpoints of greatest public health concern and in recognition of effects on the developing nervous system as a sentinel endpoint for public health impacts of Pb (see Section 3.3). We recognize, however, other population subgroups as described above may also be at risk of Pb-related health effects of public health concern.

3.4.2 Potential Public Health Impact

There are several potential public health impacts associated with the current range of population blood Pb levels, including potential impacts on population IQ, heart disease, and chronic kidney disease (CD, Section 8.6). The quantitative implications of potential Pb-related population impacts related to these health impacts are discussed in the CD (Sections 8.6.2, 8.6.3 and 8.6.4). With regard to IQ, it is noted that, given a somewhat uniform manifestation of Pb-related decrements across the range of IQ scores in a population, "a downward shift in the mean IQ value is not associated only with a substantial increase in the percentage of individuals achieving very low scores, but also with substantial decreases in percentages achieving very high scores" (CD, p. 8-81). For example, for a population mean IQ of 100 (and standard deviation of 15), 2.3% of the population would score above 130, but a shift of the population to a mean of 95 results in only 0.99% of the population scoring above 130 (CD, pp. 8-81 to 8-82).

In emphasizing the need to recognize distinctions between population and individual risk, the CD notes that a "point estimate indicating a modest mean change on a health index at the individual level can have substantial implications at the population level" (CD, p. 8-77). For example, "the import of a decline for an individual's well-being is likely to vary depending on the portion of the IQ distribution" such that "for an individual functioning in the low range due to the influence of developmental risk factors other than Pb", a Pb-associated IQ decline of several points might be sufficient to drop that individual into the range associated with increased risk of educational, vocational, and social handicap (CD, p. 8-77). Similarly, "although an increase of a few mmHg in blood pressure might not be of concern for an individual's well-being, the same increase in the population mean might be associated with substantial increases in the percentages of individuals with values that are sufficiently extreme that they exceed the criteria used to diagnose hypertension" (CD, p. 8-77).

The magnitude of a public health impact is dependent upon the size of population affected and type or severity of the effect. As summarized in Section 3.4.1, there are several population groups that may be susceptible or vulnerable to effects associated with exposure to Pb. They include young children, particularly those in families of low SES, as well as individuals with hypertension, diabetes, and chronic renal insufficiency. Although comprehensive estimates of the size of these groups residing in proximity to policy-relevant sources of ambient Pb have not been developed, total estimates of these population subpopulations within the U.S. are substantial (Table 3-3).

Table 3-3. Population subgroups with characteristics that may contribute to increased susceptibility or vulnerability to Pb health effects.

	Children[a] Living in poverty	Adults[b] w. hypertension[c]	Adults[b] w. Diabetes	Adults[b] w. chronic kidney disease
Estimated number in U.S. population[d]	4.8 million (20%)[e]	~50 million (25.6%)[e]	18 million (8.7%)[e]	19.2 million (11%)[e]
Year for estimate	2005	1999-2002	2002	1988-1994
Reference	DeNavas-Walt et al., 2006	NCHS, 2005	CDC, 2003	Coresh et al., 2005

[a]Children less than 6 years of age.
[b]Individuals greater than 20 year of age.
[c]Hypertension, defined as blood pressure of 140/90 millimeters of mercury (mm Hg) or higher, using blood pressure lowering medications, or having been told at least twice by a physician or other health professional that they had high blood pressure (medical history).
[d]Note that there may be overlap among some groups (i.e., individuals may be counted in more than one subgroup).
[e]Percent of age group.

The limited information available on air and surface soil concentrations of Pb indicates elevated concentrations near stationary sources as compared with areas remote from such sources (CD, Sections 3.2.2 and 3.8). The air quality analyses presented in Chapter 2 indicate dramatically higher Pb concentrations at monitors near sources as compared with those more remote (Section 2.3.2.4). We are handicapped, however, in our ability to characterize the size of at-risk populations in areas influenced by policy-relevant sources of ambient Pb by the significant limitations of our monitoring and emissions information. For example, size and spatial coverage limitations of the current Pb monitoring network limits our ability to characterize the levels of airborne Pb in the U.S. today (see Section 2.3.2.1). Further, the available information on emissions and locations of sources indicates that the network is inconsistent in its coverage of the largest sources identified in the 2002 NEI, with monitors within a mile of only 2 of 26 facilities in the 2002 NEI with emissions greater than 5 tpy (Sect 2.3.2.1). Additionally, there are various uncertainties and limitations associated with source information in the NEI (Section 2.2.5).

In recognition of the significant limitations associated with the currently available information on Pb emissions and airborne concentrations in the U.S. and associated exposure of potentially at-risk populations, we have summarized the information in several different ways. None of these summaries is precisely what might be desired for this analysis, however, all are

informative with regard to considering the prevalence of airborne Pb emissions (and associated airborne Pb concentrations) and exposure of human populations.

Air quality analyses of the limited monitoring network indicates the numbers of monitoring sites exceeding alternate NAAQS levels, with consideration of different statistical forms (Section 2.3.2.5), and these analyses are summarized with regard to population size in counties home to those monitoring sites (Appendix 5.A). Information for the monitors and from the NEI indicates a range of source sizes in proximity to monitors at which various levels of Pb are reported. Together this information suggests that there is variety in the size of sources (in terms of Pb emissions, tpy) that may influence air Pb concentrations that may be of interest in this review. Identifying specific emissions levels of sources expected to result in air Pb concentrations of interest, however, would be informed by a comprehensive analysis using detailed source characterization information that has not been feasible within the time and data constraints of this review. Consequently, we have instead developed a summary of the emissions and demographic information for Pb sources that includes estimates of the numbers of people residing in counties in which the aggregate Pb emissions from NEI sources is greater than or equal to 0.1 tpy (Table 3-4) or in counties in which the aggregate Pb emissions is greater than or equal to 0.1 tpy per 1000 square miles (Table 3-5).

Table 3-4. Population size in counties with Pb emissions, by total emissions (tpy).

Total Pb Emissions in County (tpy)[*]	Number of Counties	Population (1,000's)	Under age 5 population (1,000's)
≥ 10.0	20	25,756	1,949
5.0 - 10.0	37	20,180	1,430
1.0 - 5.0	346	116,496	7,979
0.5 - 1.0	320	42,995	2,871
0.1 - 0.5	1,165	56,287	3,687
≥ 0.1	1,888	261,715	17,915

[a] 2002 NEI.

Table 3-5. Population size in counties with Pb emissions, by emissions density.

Total Pb Emissions Density in County (tpy/1000 square miles)	Number of Counties	Population (1,000's)	Under age 5 population (1,000's)
≥ 10.0	77	32,911	2,185
5.0 - 10.0	80	39,278	2,888
1.0 - 5.0	463	108,700	7,377
0.5 - 1.0	301	32,589	2,200
0.1 - 0.5	1,105	52,352	3,521
total ≥ 0.1	2,026	265,829	18,172

[a] 2002 NEI.

Additionally, the potential for historically deposited Pb near roadways to contribute to increased risks of Pb exposure and associated risk to populations residing nearby is suggested in the CD and also investigated in Chapter 4 of this document. Although estimates of the number of individuals, including children, living within close proximity to roadways specifically recognized for this potential have not been developed, these numbers may be substantial.[4]

3.5 SUMMARY AND CONCLUSIONS

Based on the available health effects evidence and the evaluation and interpretation of that evidence in the CD, summarized briefly above, the following conclusions have been drawn:

- Lead exposures occur both by inhalation and by ingestion. Ingestion of Pb-contaminated dust has a strong influence on blood Pb levels in children.
- Children, in general and especially low SES children, are at increased risk for Pb exposure and Pb-induced adverse health effects. This is due to several factors, including enhanced exposure to Pb via ingestion of soil Pb and/or dust Pb due to childhood hand-to-mouth activity and poor nutritional status.
- Once inhaled or ingested, Pb is distributed by the blood, with long-term storage accumulation in the bone. Bone Pb levels provide a strong measure of cumulative exposure which has been associated with many of the effects summarized below,

[4] For example, the 2005 American Housing Survey, conducted by the U.S. Census Bureau indicates that some 14 million (or approximately 13% of) housing units are "within 300 feet of a 4-or-more-lane roadway, railroad or airport" (U.S. Census Bureau, 2006). Additionally, estimates developed for Colorado, Georgia and New York indicate that approximately 15-30% of the populations in those states reside within 75 meters of a major roadway (i.e., a “Limited Access Highway”, “Highway”, “Major Road” or “Ramp”, as defined by the U.S. Census Feature Class Codes) (ICF, 2005).

although difficulty of sample collection has precluded widespread use in epidemiological studies to date.

- Blood levels of Pb are well accepted as an index of exposure (or exposure metric) for which associations with the key effects (see below) have been observed. In general, associations with blood Pb are most robust for those effects for which past exposure history poses less of a complicating factor, i.e., for effects during childhood.
- Epidemiological studies have observed significant associations between Pb exposures and a broad range of health effects. Many of these associations have been found at levels of blood Pb that are currently relevant for the U.S. population, with children having blood Pb levels of 5-10 μg/dL or, perhaps somewhat lower, being at notable risk.
- Pb exposure is associated with a variety of neurological effects in children, notably intellectual attainment, attention, and school performance. Both qualitative and quantitative evidence, with further support from animal research, indicates a robust effect of Pb exposure on neurocognitive ability at blood Pb levels levels in the range of 5 to 10 μg/dL, and some analyses appear to show Pb effects on intellectual attainment in young children with blood Pb levels ranging from 2 to 8 μg/dL
- Deficits in cognitive skills may have long-term consequences over a lifetime. Poor academic skills and achievement can have enduring and important effects on objective parameters of success in real life as well as increased risk of antisocial and delinquent behavior.
- For the quantitative risk assessment for neurocognitive ability in young children (described in Chapter 4), the staff concludes that that it is appropriate to use nonlinear concentration-response models that reflect the epidemiological evidence of a higher slope of the blood Pb concentration-response relationship at lower blood Pb levels.
- For children, the evidence is also robust for Pb-induced disruption of heme synthesis at blood Pb levels of approximately 15 μg/dL and higher. At blood Pb levels on the order of 10 μg/dL, and slightly lower, associations have been found with effects to the immune system, resulting in altered macrophage function, increased IgE levels and associated increased risk for autoimmunity and asthma.
- In adults, epidemiological studies have consistently demonstrated associations between Pb exposure and increased risk of adverse cardiovascular outcomes, including increased blood pressure and incidence of hypertension, as well as cardiovascular mortality. These associations have been observed with bone Pb and, for some studies with blood Pb levels below 10 μg/dL. Animal evidence provides confirmation of Pb effects on cardiovascular functions. For these Pb effects, particularly susceptible subpopulations include those with a higher baseline blood pressure. For example, African Americans, as a group, have greater incidence of elevated blood pressure than other ethnic groups.
- Renal effects in adults, evidenced by reduced renal function, have also been associated with Pb exposures indexed by bone Pb levels and also with blood Pb below 10 μg/dL, with the potential adverse impact of such effects being enhanced for susceptible

subpopulations including those with diabetes, hypertension, and chronic renal insufficiency.

- Other Pb associated effects in adults occurring at or just above 10 μg/dL include hematological (e.g., impact on heme synthesis pathway) and neurological effects, with animal evidence providing support of Pb effects on these systems and evidence regarding mechanism of action.

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4 CHARACTERIZATION OF HEALTH RISKS

4.1 INTRODUCTION

This chapter summarizes the human exposure and health risk assessments conducted in support of the current review (throughout the remainder of this chapter, the term "risk assessment" will be used to refer to both the human exposure and health risk assessments collectively, unless specific reference to either the human exposure or health risk assessments is required). There are two phases to the risk assessment for the current review: pilot and full-scale. The pilot phase was presented in the first draft Staff Paper and accompanying technical report (USEPA, 2006a, 2006b; ICF, 2006), and was the subject of a review by the Clean Air Scientific Advisory Committee (CASAC) on February 6 and 7, 2007 (Henderson, 2007a). The initial full-scale analyses were presented in the July 2007 draft report (USEPA, 2007a) and were the subject of a CASAC review at a public meeting on August 28 and 29, 2007 (Henderson, 2007b). In response to CASAC recommendations, additional analyses using a core modeling approach were conducted to complete the full-scale assessment. The complete full-scale assessment and background for these analyses, including CASAC advice on the pilot and draft full-scale assessment and the risk assessment performed for the previous review, are presented in the final Risk Assessment Report (USEPA, 2007b).

The focus for this Pb NAAQS risk assessment is on Pb derived from those sources emitting Pb to ambient air. In designing and implementing this assessment, we have been faced with significant limitations and complexity that go far beyond the situation for similar assessments typically performed for other criteria pollutants. Not only are we constrained by the timeframe allowed for this review (Section 1.2.3) in the context of the breadth of information to address, we are also constrained by significant limitations with regard to data and tools needed for the assessment. Further, the multimedia and persistent nature of Pb and the role of multiple exposure pathways contributes significant additional complexity to the assessment as compared to other assessments that focus only on the inhalation pathway.

Due to the limited data, models, and time available, we are not able to fully and completely characterize in this risk assessment all of the various complexities associated with Pb. Consequently, in our efforts to focus on and characterize risk associated with the ambient air-related sources and exposures, we have made a number of simplifying assumptions in a number of areas. Ambient air related sources are those emitting Pb into the ambient air (including resuspension of previously emitted Pb), and ambient air related exposures include inhalation of ambient air Pb as well as ingestion of Pb deposited out of the air (e.g., onto outdoor soil/dust or indoor dust). As illustrated in Figure 2-1 people are also exposed to Pb that originates from nonair sources, including leaded paint or drinking water distribution systems. For purposes of

this assessment, however, the Pb from these nonair sources is collectively referred to as "policy-relevant background" [1]. Although Pb in diet and drinking water sources may derive from Pb emitted into the ambient air, the contribution from air pathways to these exposure pathways could not be explicitly modeled, such that these exposures are treated as policy-relevant background. [2]

In the Risk Assessment Report (USEPA, 2007b), we have made every effort to completely describe the assessment design, data and methodology, to identify and describe limitations and simplifying assumptions, and to characterize our understanding of the associated uncertainty in the exposure and risk estimates. Due to the time constraints for preparation of this Staff Paper, the risk assessment is only briefly summarized in this Chapter. Key limitations and uncertainties associated with the design and associated results are briefly summarized in Section 4.2.7. We direct the reader to the Risk Assessment Report for a more complete presentation.

The remainder of this chapter is organized as follows. Section 4.1.1 provides an overview of the human health risk assessment completed in the last review of the Pb NAAQS in 1990 (USEPA, 1990a). Section 4.1.2 describes advice received from CASAC during this review. Section 4.2 provides a summary of the exposure and risk assessment, following which are separate sections dedicated to summaries of the key findings of the exposure assessment (Section 4.3) and risk assessment (Section 4.4).

4.1.1 Overview of Risk Assessment from Last Review

The risk assessment conducted in support of the last review used a case study approach to compare air quality scenarios in terms of their impact on the percentage of modeled populations that exceeded specific blood Pb levels chosen with consideration of the health effects evidence at that time (USEPA, 1990; USEPA, 1989). The case studies in that analysis, however, focused exclusively on Pb smelters including two secondary and one primary smelter and did not consider exposures in a more general urban context. Additionally, the analysis focused on children (birth through 7 years of age) and middle-aged men. The staff evaluated impacts of alternate NAAQS on numbers of children and men with blood Pb levels above levels of concern based on health effects evidence at that time. The primary difference between the risk assessment approach used in the current analysis and the assessment completed in 1990 involves the risk metric employed. Rather than estimating the percentage of study populations with exposures above blood Pb levels of interest as was done in the last review (i.e., 10, 12 and 15

[1] This categorization of policy-relevant sources and background exposures is not intended to convey any particular policy decision at this stage regarding the Pb standard.

[2] Further, although paint is a policy-relevant background source, for this analysis, it may be reflected somewhat in estimates developed for policy-relevant sources, due to modeling constraints (see USEPA, 2007b).

μg/dL), the current analysis estimates changes in health risk, specifically IQ loss, associated with Pb exposure for child populations at each of the case study locations with that IQ loss further differentiated between background Pb exposure and policy-relevant exposures.

4.1.2 CASAC Advice on Pilot and Initial Risk Analyses in this Review

The staff consulted with the CASAC on the draft analysis plan for the risk assessment (USEPA, 2006c) in June, 2006 (Henderson, 2006). Some key comments provided by CASAC members on the *Analysis Plan* included: (a) placing a higher priority on modeling the child IQ metric than the adult endpoints (e.g., cardiovascular effects), (b) recognizing the importance of indoor dust loading by Pb contained in outdoor air as a factor in Pb-related exposure and risk for sources considered in this analysis, and (c) concurring with use of the IEUBK biokinetic blood Pb model. Staff subsequently developed the pilot phase assessment, intended to test the risk assessment methodology being developed for the full-scale assessment. The pilot is described in the first draft Staff Paper and accompanying technical report (USEPA, 2006b; ICF 2006), which was discussed by the CASAC Pb panel on February 6-7 (Henderson, 2007a).

Results from the pilot assessment, together with comments received from CASAC and the public, informed decisions on the design of the full-scale analysis. The full-scale analysis included a substitution of a more generalized urban case study for the location-specific near roadway case study evaluated in the pilot. In addition, a number of changes were made in the exposure and risk assessment approaches, including the development of a new indoor dust Pb model focused specifically on urban residential locations and specification of additional IQ loss concentration-response (C-R) functions to provide greater coverage for potential impacts at lower exposure levels.

The draft full-scale assessment was presented in the July 2007 draft risk assessment report (USEPA, 2007a) that was released for public comment and provided to CASAC for review. In their review of the July draft risk assessment report, the CASAC Pb Panel made several recommendations for additional exposure and health risk analyses (Henderson, 2007b). Results from the initial full-scale analyses, along with comments from CASAC and the public resulted in additional modifications and enhancements to the full-scale assessment, resulting in a final version of the full-scale assessments which is described in this chapter and presented in greater detail in the accompanying Risk Assessment Report and associated appendices (USEPA, 2007b). These include specification of additional IQ loss concentration-response functions and the addition of a set of location-specific urban case studies.

4.2 DESIGN OF EXPOSURE AND RISK ASSESSMENTS

This section provides an overview of key elements in the design of the risk assessment. In the discussion below, we highlight key aspects of the assessment design, inputs, and methods, and we also highlight key uncertainties and limitations for each. In so doing, we have identified throughout this section, subsections of the Risk Assessment Report that provide the corresponding detail.

In this section, we first focus on how this assessment builds on the health effects evidence discussed in Chapter 3 with discussion of the endpoint, metric and model used here for risk quantitation (Section 4.2.1). We also discuss key aspects of the assessment design (Sections 4.2.2 and 4.2.3) and the modeling approaches (Sections 4.2.4 through 4.2.6). In this discussion, we only briefly summarize the modeling elements and refer the reader to the Risk Assessment Report for full descriptions of methodologies employed. Lastly, in Section 4.2.7, we highlight key limitations of the design and associated uncertainties in the resultant estimates. A more complete presentation of limitations, sensitivity of particular inputs or models, and uncertainties occurs in the Risk Assessment Report (USEPA, 2007b, Sections 3.5, 4.3 and 5.3.3).

4.2.1 Health Endpoint, Risk Metric and Concentration-response Functions

Of the health endpoints described in Section 3.3, the health endpoint on which we focused in the quantitative health risk assessment for this review is developmental neurotoxicity in children, with IQ decrement as the risk metric. Among the wide variety of health endpoints associated with Pb exposures, there is general consensus that the developing nervous system in young children is the most sensitive and that neurobehavioral effects (specifically neurocognitive deficits), including IQ decrements, appear to occur at lower blood levels than previously believed (i.e., at levels <10 μg/dL). For example, the overall weight of the available evidence, described in the CD, provides clear substantiation of neurocognitive decrements being associated in young children with blood Pb levels in the range of 5 to 10 μg/dL, and some analyses indicate Pb effects on intellectual attainment of young children ranging from 2 to 8 μg/dL (CD, Sections 6.2, 8.4.2 and 8.4.2.6). That is, while blood Pb levels in U.S. children ages one to five years have decreased notably since the late 1970s, newer studies have investigated and reported associations of effects on the neurodevelopment of children with these more recent blood Pb levels (CD, Chapter 6).

The evidence for neurotoxic effects in children is a robust combination of epidemiological and toxicological evidence (CD, Sections 5.3, 6.2 and 8.5). The epidemiological evidence is strongly supported by animal studies that substantiate the biological plausibility of the associations, and provides an understanding of mechanisms of action for the effects (CD, Section 8.4.2). The selection of children's IQ for the quantitative risk assessment

reflects consideration of the evidence presented in the CD as well as advice received from CASAC (Henderson, 2006, 2007a).

The epidemiological studies that have investigated blood Pb effects on IQ (see CD, Section 6.2.3) have considered a variety of specific blood Pb metrics, including: 1) blood concentration "concurrent" with the response assessment (e.g., at the time of IQ testing), 2) average blood concentration over the "lifetime" of the child at the time of response assessment (e.g., average of measurements taken over child's first 6 or 7 years), 3) peak blood concentration during a particular age range, and 4) early childhood blood concentration (e.g., the mean of measurements between 6 and 24 months age). All four specific blood Pb metrics have been correlated with IQ (see CD, p. 6-62; Lanphear et al., 2005). In the international pooled analysis by Lanphear and others (2005), however, the concurrent and lifetime averaged measurements were considered "stronger predictors of lead-associated intellectual deficits than was maximal measured (peak) or early childhood blood lead concentrations," with the concurrent blood Pb level exhibiting the strongest relationship (CD, p. 6-29). It is not clear in this case, or for similar findings in other studies, whether the cognitive deficits observed were due to Pb exposure that occurred during early childhood or were a function of concurrent exposure. Nevertheless, concurrent blood Pb levels likely reflected both ongoing exposure and preexisting body burden (CD, p. 6-32).

Using concurrent blood Pb level as the exposure metric and IQ as the response from the pooled dataset of seven international studies, Lanphear and others (2005) employed mathematical models of various forms, including linear, cubic spline, log-linear, and piece-wise linear, in their investigation of the blood Pb concentration-response relationship (CD, p. 6-29; Lanphear et al., 2005). They observed that the shape of the concentration-response relationship is nonlinear and the log-linear model provides a better fit over the full range of blood Pb measurements than a linear one (CD, p. 6-29 and pp. 6-67 to 6-70; Lanphear et al., 2005). In addition, they found that no individual study among the seven was responsible for the estimated nonlinear relationship between Pb and deficits in IQ (CD p. 6-30). Others have also analyzed the same dataset and similarly concluded that, across the range of the dataset's blood Pb levels, a log-linear relationship was a significantly better fit than the linear relationship ($p=0.009$) with little evidence of residual confounding from included model variables (CD, Section 6.2.13; Rothenberg and Rothenberg, 2005).

A nonlinear blood Pb concentration-response relationship is also suggested by several other studies that have observed that each μg/dL increase in blood Pb may have a greater effect on IQ at blood Pb levels below 10 μg/dL than at higher levels (CD, pp. 8-63 to 8-64). While this may at first seem at odds with certain fundamental toxicological concepts, a number of examples of non- or supralinear dose-response relationships exist in toxicology (CD, pp. 6-76 and 8-83 to

8-89).[3] With regard to the effects of Pb on neurodevelopmental outcome such as IQ, the CD suggests that initial neurodevelopmental effects at lower Pb levels may be disrupting very different biological mechanisms (e.g., early developmental processes in the central nervous system) than more severe effects of high exposures that result in symptomatic Pb poisoning and frank mental retardation (CD, p. 6-76). In comparing across the individual studies and the pooled analysis, it is observed that at higher blood Pb levels, the slopes derived for log-linear and linear models are almost identical, and for studies with lower blood Pb levels, the slopes appear to be steeper than those observed in studies involving higher blood Pb levels (CD, p. 8-78, Figure 8-7).

Given the evidence summarized here and described in detail in the CD (Chapters 6 and 8), and in consideration of CASAC recommendations (Henderson, 2006, 2007a, 2007b), the risk assessment for this review relies on the functions presented by Lanphear and others (2005) that relate absolute IQ as a function of concurrent blood Pb or of the log of concurrent blood Pb, and lifetime average blood Pb, respectively. As discussed above, the slope of the concentration-response relationship described by these functions is greater at the lower blood Pb levels (e.g., less than 10 μg/dL). The impact of the nonlinear slope is illustrated by the estimates of IQ decrements associated with increases in blood IQ for different ranges of blood Pb level reported for the log-linear model (Lanphear et al., 2005). These estimates were IQ decrements of 3.9 (with 95% confidence interval, CI, of 2.4-5.3), 1.9 (95% CI, 1.2-2.6) and 1.1 (95% CI, 0.7-1.5), for increases in concurrent blood Pb from 2.4 to 10 μg/dL, 10 to 20 μg/dL, and 20 to 30 μg/dL, respectively (Lanphear et al., 2005). For an increase in concurrent blood Pb levels from <1 to 10 ug/dL, the log-linear model estimates a decline of 6.2 points in full scale IQ which is comparable to the 7.4 point decrement in IQ for an increase in lifetime mean blood Pb levels up to 10 ug/dL observed in the Rochester study (CD, pp 6-30 to 6-31).

Several studies have examined the relationship of IQ decrement with blood Pb, quantified by a variety of metrics, at lower blood Pb levels. On a change in IQ per μg/dL basis, estimates of IQ decrement associated with blood Pb levels (concurrent, 24-month, peak, lifetime average or lifetime cumulative) below 10 μg/dL range from -0.4 to -1.8 (CD, Table 8.7). At the upper end of this range are the slopes derived for the subsets of children in the Rochester and Boston cohorts for which peak blood Pb levels were <10 μg/dL; these slopes are -1.8 (for concurrent blood Pb influence on IQ) and -1.6 (for 24-month blood Pb influence on IQ), respectively. The numbers of children in these low blood Pb subsets of the Rochester and Boston cohorts are 101 and 48, respectively. A similar stratification of the pooled dataset by Lanphear and others (2005)

[3] Similarly, a nonlinear concentration-response relationship was observed for the relationship between blood Pb levels and blood pressure in adults (CD, pp. 8-83 to 8-89).

yielded a slope for the linear function of IQ change associated with concurrent blood Pb of -0.8 for the subset of the children in the pooled data set for which maximal or peak blood Pb levels were below 10 μg/dL. Of the 1333 children in the full pooled dataset, there were 244 in this subset. When the full dataset was restricted to a still smaller subset of 103 children for which peak blood Pb levels were below 7.5 μg/dL the slope of concurrent blood Pb and IQ was -2.94 (Lanphear et al., 2005). The analysis of this latter subset supported the authors' conclusions that "for a given increase in blood lead, the lead-associated intellectual decrement for children with a maximal blood lead level <7.5 μg/dL was significantly greater than that observed for those with a maximal blood lead level $\geq$ 7.5 μg/dL (p-0.015)" and that "environmental lead exposure in children who have maximal blood lead levels < 7.5 μg/dL is associated with intellectual deficits". This subset was composed primarily of children from the Rochester cohort (69 children), with smaller numbers of children from five of the other 7 cohorts (Lanphear et al., 2005). The Rochester data included IQ test and concurrent blood Pb measurements taken at age 6 (Lanphear et al., 2005). The linear slope observed for this subset of the pooled dataset, however, was notably greater than that previously reported for the low blood Pb subset of the Rochester cohort at age 5 described above, and greater than those slopes from other studies for blood Pb < 10 μg/dL summarized in the CD (e.g., CD, Table 8-7), providing some uncertainty with regard to the precise magnitude of slope for the full range of blood Pb below 7.5 μg/dL.

As discussed in the CD, threshold blood Pb levels for these effects cannot be discerned from the currently available epidemiological studies, and the evidence in the animal Pb neurotoxicity literature does not define a threshold for any of the toxic mechanisms of Pb (CD, Sections 5.3.7 and 6.2).

In applying relationships observed with the pooled analysis (Lanphear et al., 2005) to the risk assessment, which includes blood Pb levels below the range represented by the pooled analysis, several alternative blood Pb concentration-response models were considered in recognition of a reduced confidence in our ability to characterize the quantitative blood Pb concentration-response relationship at the lowest blood Pb levels represented in the recent epidemiological studies. The functions considered and employed in the initial risk analyses for this review include the following.

- Log-linear function with low-exposure linearization, for both concurrent and lifetime average blood metrics, applies the nonlinear relationship down to the blood Pb concentration representing the lower bound of blood Pb levels for that blood metric in the pooled analysis and applies the slope of the tangent at that point to blood Pb concentrations estimated in the risk assessment to fall below that level.
- Log-linear function with cutpoint, for both concurrent and lifetime average blood metrics, also applies the nonlinear relationship at blood Pb concentrations above the lower bound of blood Pb concentrations in the pooled analysis dataset for that blood

metric, but then applies zero risk to all lower blood Pb concentrations estimated in the risk assessment.

- Two-piece linear function, for both concurrent and lifetime average blood metrics, applies a two-piece linear model derived from the log-linear function to all blood Pb concentrations estimated in the risk assessment.

In the additional risk analyses performed subsequent to the August 2007 CASAC public meeting (Section 4.1.2) using the core modeling approach, the first two functions listed above and the following two functions were employed (see Section 5.3.1 of the Risk Assessment Report for details on the forms of these functions as applied in this risk assessment).

- Population stratified dual linear function for concurrent blood Pb, derived from the pooled dataset stratified at peak blood Pb of 10 μg/dL and
- Population stratified dual linear function for concurrent blood Pb, derived from the pooled dataset stratified at 7.5 μg/dL peak blood Pb.

In interpreting risk estimates derived using the various functions, consideration should be given to the uncertainties with regard to the precision of the coefficients used for each analysis. The coefficients for the log-linear model from Lanphear et al. (2005) had undergone a careful development process, including sensitivity analyses, using all available data from 1,333 children. The shape of the exposure-response relationship was first assessed through tests of linearity, then by evaluating the restricted cubic spline model. After determining that the log-linear model provided a good fit to the data, covariates to adjust for potential confounding were included in the log-linear model with careful consideration of the stability of the parameter estimates. After the multiple regression models were developed, regression diagnostics were employed to ascertain whether the lead coefficients were affected by collinearity or influential observations. To further investigate the stability of the model, a random-effects model (with sites random) was applied to evaluate the results and also the effect of omitting one of the seven cohorts on the lead coefficient. In the various sensitivity analyses performed, the coefficient from the log-linear model was found to be robust and stable. The log-linear model, however, is not biologically plausible at very low blood Pb concentrations as they approach zero; therefore, in the first two functions the log-linear model is applied down to a cutpoint, selected based on the low end of the blood Pb levels in the pooled dataset, followed by a linearization or an assumption of zero risk at levels below that point.

In contrast, the coefficients from the two analyses using the population stratified dual linear function with cutpoints at 7.5 μg/dL and 10 μg/dL, peak blood Pb, have not undergone such careful development. These analyses were primarily done to compare the lead-associated decrement at lower blood lead concentrations and higher blood lead concentrations. For these analyses, the study population was stratified at the specified cutpoint and separate linear models

were fitted to the data above and below the cutpoint. The fit of the model or sensitivity analyses were not conducted (or reported) on these coefficients. While these analyses are quite suitable for the purpose of investigating whether the slope at lower concentration levels are greater compared to higher concentration levels, use of such coefficients in a risk analysis to assess public health impact may be inappropriate. Further, only 103 children had maximal blood lead levels less than 7.5 μg/dL and 244 children had maximal blood lead levels less than 10 μg/dL. While these children may better represent current blood lead levels, not fitting a single model using all available data may lead to bias. Slob et al. (2005) noted that the usual argument for not considering data from the high dose range is that different biological mechanisms may play a role at higher doses compared to lower doses. However, this does not mean a single curve across the entire exposure range cannot describe the relationship. The fitted curve merely assumes that the underlying dose-response follows a smooth curve over the whole dose range. If biological mechanisms change when going from lower to higher doses, this change will result in a gradually changing slope of the dose-response. The major strength of the Lanphear et al. (2005) study was the large sample size and the pooled analysis of data from seven different cohorts. In the case of the 7.5 μg/dL cutpoint, less than 10% of the available data is used in the analysis, with more than half of the data coming from one cohort (Rochester) and the six other cohorts contributing zero to 13 children to the analysis. Such an analysis dissipates the strength of the Lanphear et al. study.

In consideration of the preceding discussion, we place greater confidence in the log-linear model form compared to the dual-linear stratified models for our purposes in this risk assessment. Further, in considering risk estimates derived from the four core functions (log-linear function with low-exposure linearization, log-linear function with cutpoint, dual linear function, stratified at 7.5 μg/dL peak blood Pb, and dual linear function, stratified at 10 μg/dL peak blood Pb), we have assigned greatest confidence to risk estimates derived using the log-linear function with low-exposure linearization since this function (a) is a nonlinear function that describes greater response per unit blood Pb at lower blood Pb levels consistent with multiple studies identified in the discussion above, (b) is based on fitting a function to the entire pooled dataset (and hence uses all of the data in describing response across the range of exposures), (c) is supported by sensitivity analyses showing the model coefficients to be robust, and (d) provides an approach for predicting IQ loss at the lowest exposures simulated in the assessment (consistent with the lack of evidence for a threshold). Note, however, that risk estimates generated using the other three concentration-response functions are also presented to provide perspective on the impact of uncertainty in this key modeling step.

4.2.2 Case Studies

For this risk assessment, we have employed a case study approach to inform our understanding of risks associated with exposure of children in the U.S. to Pb associated with outdoor ambient air under current conditions, under conditions that would just meet the current NAAQS, and under conditions associated with just meeting a range of alternative NAAQS. The case study approach is described in Sections 2.2 (and subsections) and 5.1.3 of the Risk Assessment Report (USEPA, 2007b); the assessment scenarios are described in Sections 2.3 (and subsections) and 5.1.1 of that report.

The four types of case studies included in the assessment are the following:

- Location-specific urban case studies: Three urban case studies focused on specific urban areas (Cleveland, Chicago and Los Angeles) have been modeled to provide perspectives on the magnitude of ambient air Pb-related risk in specific urban locations. Ambient air Pb concentrations are characterized using source-oriented and other Pb-TSP monitors in these cities.
- General urban case study: Represents a nonlocation-specific analysis which uses several simplifying assumptions regarding ambient air Pb levels and demographics to produce a simplified representation of urban areas intended to inform our assessment of the impact of changes in ambient Pb concentrations on risk.
- Primary Pb smelter case study: This case study is modeled to estimate risk for children living in an area currently not in attainment with the current NAAQS, that is impacted by Pb emissions from a primary Pb smelter. As such, this case study characterizes risk for a specific highly exposed population and also provides insights on risk to child populations living in areas near large sources of Pb emissions.
- Secondary Pb smelter case study: This case study was included in the initial analyses for the full-scale assessment as an example of areas influenced by smaller point sources of Pb emissions. As discussed in Section 4.2.7 below, however, we have recognized a variety of significant limitations in the approaches employed for this case and associated large uncertainties in these results which preclude the use of this case study as illustrative of the larger set of areas influenced by similarly sized Pb sources. We note that risk estimates for this case study (presented in detail in the Risk Assessment Report) are lower than those for the other case studies.

4.2.3 Air Quality Scenarios

Air quality scenarios modeled for this analysis include (a) a current conditions scenario for the location-specific urban case studies, the general urban case study, and the secondary Pb smelter case study; (b) a current NAAQS scenario for the location-specific urban case studies, the general urban case study, and the primary Pb smelter case study; and (c) a range of alternative NAAQS scenarios for all case studies. The alternative NAAQS scenarios include 0.5, 0.2, 0.05, and 0.02 $\mu g/m^3$ (as maximum monthly averages) and 0.2 $\mu g/m^3$ (as a maximum quarterly average). The current NAAQS scenario for the urban case studies involved a "rolling

up" of ambient air Pb levels from current conditions. While EPA staff recognizes that it is extremely unlikely that Pb concentrations in urban areas (see Section 2.3.2.4) would rise to meet the current NAAQS and there are limitations and uncertainties associated with the approach used (as described in Section 4.2.7 below), this scenario was included to provide some perspective on risks associated with the current NAAQS relative to current conditions.

Details of the assessment scenarios, including a description of the derivation of Pb concentrations for air and other media are presented in Sections 2.3 (and subsections) and Section 5.1.1 of the Risk Assessment Report (USEPA, 2007b).

4.2.4 Categorization of Policy-relevant Exposure Pathways

To inform policy aspects of the Pb NAAQS review, we have attempted to parse the assessment estimates for blood Pb and IQ loss into the fraction associated with background sources (e.g., diet and drinking water) versus that associated with policy-relevant pathways, which include inhalation, outdoor soil/dust ingestion and indoor dust ingestion. We have further categorized the policy-relevant pathways into one of two categories, “recent air” or “past air”. Conceptually, the recent air category includes those pathways involving Pb that is or has recently been in the outdoor ambient air, including inhalation and ingestion of indoor dust Pb derived from recent ambient air (i.e., air Pb that has penetrated into the residence recently and loaded indoor dust). Past air includes exposure contributions from ingestion of outdoor soil/dust that is contacted on surfaces outdoors, and ingestion of indoor dust Pb that is derived from past air sources (i.e., impacts from Pb that was in the ambient air in the past and has not been recently resuspended into ambient air). In this assessment, as discussed further below, that portion of indoor dust Pb not associated with recent air is classified as "other" and, due to technical limitations, includes not only past air impacts, but also contributions from Pb paint. The reader is referred to Sections 2.4.3 and 3.2.2 of the Risk Assessment Report for additional detail on partitioning of exposure and risk between policy-relevant and background exposure pathways.

In simulating reductions in exposure associated with reducing ambient air Pb levels through alternative NAAQS (and increases in exposure if the current NAAQS was reached in certain case studies), our modeling has only affected the exposure pathways we categorize as recent air (inhalation and ingestion of that portion of indoor dust associated with outdoor ambient air). We have not simulated decreases in past air-related exposure pathways (e.g., reductions in outdoor soil Pb levels following reduction in ambient air Pb levels and a subsequent decrease in exposure through incidental soil ingestion and the contribution of outdoor soil to indoor dust). This approach is likely to underestimate reductions in ambient air related exposure and risk. Consequently, incremental reductions in exposure and risk estimated for alternative NAAQS considered in the full-scale analysis, which reflect simulated reductions in the recent air

category, are likely to be underpredictions of the impact of changes to the NAAQS on total Pb exposure and health risk.

Additionally, there is uncertainty related to parsing out exposure and risk between background sources and policy-relevant exposure pathways (and subsequent parsing of recent air and past air) resulting from a number of technical limitations. Key among these is that, while conceptually, Pb paint contributions to indoor dust Pb would be considered background and included in modeling background exposures, due to technical limitations related to indoor dust Pb modeling, ultimately, Pb paint was included as part of "other" indoor dust Pb (i.e., as part of past air exposure). This means that total exposure and risk associated with past air would be overestimated due to inclusion of Pb paint as part of "other" indoor dust Pb ingestion. Uncertainty related to parsing of exposure and risk between background sources and policy-relevant exposure pathways are discussed in Sections 2.4.3, 3.2.2 and 3.4 of the Risk Assessment Report.

In summary, because of limitations in the assessment design, data and modeling tools, the risk attributable to policy-relevant exposure pathways is bounded on the low end by the risk estimated for the “recent air” category and on the upper end by the risk estimated for the “recent air” plus “past air” categories.

4.2.5 Overview of Analytical Steps

The risk assessment includes four analytical steps, briefly discussed below. The reader is referred to Sections 2.4.4, 3.1, 3.2, 4.1, and 5.1 of the Risk Assessment Report for additional detail.

- *Characterization of Pb in ambient air*: The characterization of outdoor ambient air Pb levels uses one of three basic approaches in each case study: (a) establishment of exposure zones using source-oriented and non-source oriented monitors (location-specific urban case studies), (b) establishment of a single exposure zone with uniform levels of Pb in exposure media (general urban case study), or (c) air dispersion modeling of Pb released from operations associated with a particular facility (point source case studies).
- *Characterization of outdoor soil/dust and indoor dust Pb concentrations*: Outdoor soil Pb levels were estimated using empirical data (including site-specific and/or national datasets) and/or fate and transport modeling. Indoor dust Pb levels were predicted using a combination of (a) regression-based models that relate indoor dust to ambient air Pb and/or outdoor soil Pb, and (b) mechanistic models that predict indoor dust Pb based on key mechanisms (e.g., exchange of outdoor air with indoor air, deposition rates for Pb to indoor surfaces, house cleaning rates). For the point source case studies, regression-based models obtained from the literature or developed based on site-specific data were used, and a hybrid empirical-mechanistic model was developed and used for the urban case studies. The model for urban case studies was developed as the

available regression-based models had been developed largely based on residential exposures near large point sources and were not considered representative of more general urban exposures.

- *Characterization of blood Pb levels*: Blood Pb levels for each exposure zone are derived from central-tendency blood Pb concentrations estimated using the Integrated Exposure and Uptake Biokinetic (IEUBK) model. Concurrent or lifetime average blood Pb is estimated from the IEUBK outputs as described in Section 3.2.1.1 of the Risk Assessment Report. For the point source and location-specific urban case studies, a probabilistic exposure model is used to generate population distributions of blood Pb concentrations based on:

  (a) the central tendency blood Pb levels for each exposure zone,

  (b) demographic data for the distribution of children (less than 7 years of age) across exposure zones in a given study area, and

  (c) a GSD intended to characterize interindividual variability in blood Pb (e.g., reflecting differences in behavior and biokinetics related to Pb).

  For the general urban case study, as demographic data for a specific location are not considered, the GSD is applied directly to the central tendency blood Pb level to estimate a population distribution of blood Pb levels.

- *Risk characterization (estimating IQ loss)*: Concurrent or lifetime average blood Pb levels generated for each simulated child in each case study population are converted into total Pb-related IQ loss estimates using the concentration-response functions described in Section 4.2.1 (See Section 4.2.6 and Section 5.3.1 of the Risk Assessment Report). The estimates of IQ loss are presented in two ways: (a) population-weighted distributions of IQ loss from which specific population percentiles are identified, and (b) for the location-specific urban case studies, population risk incidence distributions providing estimates of the number of children with specific amounts of IQ loss in a particular case study. There are a range of uncertainties associated with the development and application of these concentration-response functions that are summarized in Sections 4.2.1 and 4.2.7.

The urban case studies differ from the point source case studies in terms of how ambient air Pb levels were characterized and in the specific mix of modeling and empirical data used to characterize Pb levels in exposure media (e.g., outdoor soil and indoor dust). Key elements of the approaches used in each of the case study categories are summarized below and described in more detail in the Risk Assessment Report.

- Location-specific urban case studies: Study areas were defined based on the monitoring data for that city in the 2003-2005 dataset analyzed in Chapter 2, with the monitor locations used to define the outer extent of each study area. This resulted in study areas with varying dimensions for the three cities including: Cleveland (5 miles by 5 miles), Chicago (20 miles by 5 miles) and Los Angeles (40 miles by 20 miles). For the Cleveland and Chicago case studies, two types of exposure zones were modeled: (a) zones associated with source oriented monitors and (b) zones associated with non

source oriented monitors. Source oriented exposure zones extend one mile out from each source-oriented monitor, and ambient air Pb for all children in those zones is assumed to be at the level of that monitor. Children in the study area not associated with these source-oriented zones are assigned ambient air Pb levels associated with the nearest non source oriented monitor. This approach essentially assumes that source oriented monitors have an impact close by, and that other monitors represent air Pb levels more common across the study area. Because the Los Angeles case study does not have any source oriented monitors, children in the study area were simply assigned to the nearest monitor. All three case studies were modeled using the same soil Pb levels as that used in the general urban case study (i.e., these were not location-specific). The remainder of the modeling used the same core modeling approach as employed for the general urban case study (see below). Demographic data (i.e., child counts) were based on US Census 2000 block group data. See Sections 5.1 and 5.2 of the Risk Assessment Report for details on these case studies.

- General urban case study: This case study was intended to characterize exposure and risk for a child urban population under several simplifying assumptions including (a) ambient air Pb levels for a given air quality scenario are assumed to be uniform across the study area and (b) demographics are assumed uniform across the study area. This essentially translates into a single study area with uniform ambient air Pb levels and population total. Two current conditions scenarios were assessed for this case study: (a) one based on the mean Pb-TSP level in U.S. urban areas of more than a million population, and (b) a second high-end scenario based on the 95^{th} percentile Pb-TSP level for U.S. urban areas of more than a million people. No demographic data were used in this case study, since it is not location specific.
- Primary and secondary Pb smelter case studies: Both of these case studies were modeled using air dispersion model-derived ambient air Pb levels. Each was also modeled using both a 10 km radius study area surrounding the facility and a smaller 1.5 km subarea, that omitted a large number of lower air Pb exposed children that influenced the full study area results toward "lower risk" exposure and risk distributions. These case studies used either U.S. Census 2000 block-level data (secondary Pb smelter case study) or a combination of block and block group-level data (primary Pb smelter case study) as the basis for generating population risk projections. There are differences in the specific mix of empirical data and modeling used for the two case studies to characterize Pb levels in indoor dust and outdoor soil/dust.

4.2.6 Generating Multiple Sets of Risk Results

In the initial analyses for the full-scale assessment, staff implemented multiple modeling approaches for each case study scenario in an effort to characterize the potential impact on exposure and risk estimates of uncertainty associated with the limitations in the tools, data and methods available for this risk assessment and with key analytical steps in the modeling approach (e.g., prediction of indoor dust Pb levels given changes in outdoor ambient air Pb, prediction of IQ loss given specific Pb blood levels). These multiple modeling approaches are described in Section 2.4.6.2 of the Risk Assessment Report. In consideration of comments

provided by CASAC (Henderson, 2007b) on these analyses regarding which modeling approach they felt had greater scientific support, we identified a smaller set of modeling combinations as the core approach for the subsequent new analyses (presented in Chapter 5 of the Risk Assessment Report). The core modeling approach is described in detail in Sections 5.1.2,5.2 and 5.3 of the Risk Assessment Report (USEPA, 2007b). This approach includes the following key elements:

- the hybrid indoor dust model specifically developed for urban residential applications,
- the IEUBK blood Pb model,
- the concurrent blood Pb metric,
- a geometric standard deviation (GSD) for concurrent blood Pb of 2.1 to characterize interindividual variability in blood Pb levels, and
- four different functions relating concurrent blood Pb to IQ loss, including two log-linear models (one with low-exposure linearization and one with a cutpoint) and two dual-linear models with stratification, one stratified at 7.5ug/dL peak blood Pb and the other at 10 ug/dL peak blood Pb (see Section 4.2.1).

The core modeling approach utilizes one overall modeling approach for estimating exposure for each case study and then combines this with the four concentration-response functions referenced above to derive four sets of risk results for each case study. Although we have included risk results based on applying all four concentration-response models to provide coverage for uncertainty related to this key modeling step, for reasons described in Section 4.2.1 above, we have greater confidence in the log-linear with low-exposure linearization concentration-response function (LLL) and the results generated using that function are emphasized below in summarizing risk estimates.

Exposure and risk estimates generated for the initial full set of modeling approaches applied to the general urban case study and the two point source case studies are presented in detail in Chapters 3 and 4 of the Risk Assessment Report (USEPA, 2007b). While the estimates for the core modeling approach described above are emphasized in presenting the exposure and risk results in this chapter, the fuller set of results provide additional perspective on uncertainty, especially in relation to exposure modeling.

In addition to analyzing multiple modeling approaches to address potential uncertainty in the overall analysis and illustrate the potential impact of that uncertainty on estimated exposure and risk levels, we have also evaluated performance of models applied in the assessment (see Sections 3.5.1 and 3.5.2 of the Risk Assessment Report) and performed sensitivity analyses to characterize the potential impact of uncertainty in key analysis steps on exposure and risk estimates (see Sections 4.3.2, 5.3.3.4 of the Risk Assessment Report).

4.2.7 Key Limitations and Uncertainties

As recognized in Section 4.1 above, we have made a number of simplifying assumptions in a number of areas of this assessment due to the limited data, models, and time available. We have attempted to completely describe these assumptions, and related limitations and uncertainties of the assessment design and results in the Risk Assessment Report (USEPA, 2007b). Key assumptions, limitations and uncertainties are only briefly identified below, and the reader is referred to the Risk Assessment Report for detailed discussion. The aspects of the assessment discussed below are considered by staff to be particularly important to the interpretation of the exposure and risk estimates. .

Limitations in Assessment and Case Study Designs and Associated Uncertainty

- *Temporal aspects*: As described in Section 2.4.1 of the Risk Assessment Report, exposure for the simulated child population begins at birth and continues for 7 years, with Pb concentrations in all exposure media remaining constant throughout the period, and children residing in the same exposure zone throughout the period. In characterizing exposure media concentrations, annual averages are derived and held constant through the seven year period. Exposure factors and physiological parameters vary with age of the cohort through the seven year exposure period, several exposure factors and physiological parameters are varied on an annual basis within the blood Pb modeling step (see Section 3.2 of the Risk Assessment Report). These aspects are a simplification of population exposures that contributes uncertainty to our exposure and risk estimates.
- *General urban case study*: This case study differs from the others in several ways (described in more detail in Risk Assessment Report, Section 2.2.1). It is by definition a general case study and not based on a specific location. There is a single exposure zone for the case study within which all media concentrations of Pb are assumed to be spatially uniform; that is, no spatial variation within the area is simulated (Risk Assessment Report, Sections 2.4.2, 3.1.1 and 4.3.1). Additionally, the case study does not rely on any specific demographic values. Within the single exposure zone a theoretical population of unspecified size is assumed to be uniformly distributed. Thus this case study is a simplified representation of urban areas intended to inform our assessment of the impact of changes in ambient Pb concentrations on risk, but which carries with it attendant uncertainties in our interpretation of the associated exposure and risk estimates. For example, the risk estimates for this case study, while generally representative of an urban residential population exposed to the specified ambient air Pb levels, cannot be readily related to a specific urban population. Specific urban populations are spatially distributed in a nonuniform pattern and experience ambient air Pb levels that vary through time and space. Consequently, interpretations of the associated blood Pb and risk estimates with regard to their relevance to specific urban residential exposures carry substantial uncertainty and presumably an upward bias in risk, particularly for large areas, across which air concentrations may vary substantially.

- *Point source case studies*: Dispersion modeling was used to characterize ambient air Pb levels in the point source case studies. This approach simulates spatial gradients related to dispersion and deposition of Pb from emitting sources. In the case of the point sources modeled, sources were limited to those associated with the smelter operations, and did not include other sources such as resuspension of roadside Pb not immediately related to facility operations, and other stationary sources of Pb within or near the study area. This means that, with distance from the facility, there is likely underestimation of ambient air-related Pb exposure because with increased distance from the facility there would be increasing influence of other sources relative to that of the facility. We believe this limitation to have more significant impact on risk estimates associated with the full study than on those for the subareas, and to perhaps have a more significant impact on risk estimates associated with the smaller secondary Pb smelter (see below).
- *Secondary Pb smelter case study*: Air Pb concentration estimates derived from the air dispersion modeling completed for the secondary Pb smelter case study are subject to appreciably greater uncertainty than that for those for the primary Pb smelter case study due to a number of factors, including: (a) a more limited and less detailed accounting of emissions and emissions sources associated with the facility (particularly fugitives), (b) a lack of prior air quality modeling analyses and performance analyses, and (c) a substantially smaller number of Pb-TSP monitors in the area that could be used to evaluate and provide confidence in model performance[4]. Further, as mentioned in the previous bullet, no air sources of Pb other than those associated with the facility were accounted for in the modeling. Given the relatively smaller magnitude of emissions from the secondary Pb smelter, the underestimating potential of this limitation with regard to air concentrations with distance from the facility has a greater relative impact on risk estimates for this case study than for the primary Pb smelter case study. The aggregate uncertainty of all of these factors has left us with low confidence in estimates for this case study. We note that exposure and risk estimates (presented in Chapters 3 and 4 of the Risk Assessment Report) are lower than those for the other case studies. Although we had initially intended to use this case study as an example of areas near stationary sources of intermediate size (smaller than the primary Pb smelter), our experience with this analysis indicates that substantially more data and multiple case studies differing in several aspects would be needed to broadly characterize risks for such a category of Pb exposure scenarios.

[4] The information supporting the air dispersion modeling for the primary Pb smelter case study (see Section 3.5.1.1 of the Risk Assessment Report) provides substantially greater confidence in estimates for that case study.

Limitations in Estimation of Ambient Air Pb concentrations and Associated Uncertainty

- *Location-specific urban case studies*: As recognized in Section 2.3.2.1, the Pb-TSP monitoring network is currently quite limited. The number of monitors available to represent air concentrations in these case studies ranged from six for Cleveland to 11 for Chicago. Accordingly, our estimates of the magnitude of and spatial variation of air Pb concentrations are subject to uncertainty associated with the limited data. In applying the available data to each of these case studies, exposure zones, one corresponding to each monitor, were created and each U.S. Census block group (and the children within that demographic unit) were distributed among the exposure zones. The details of the approach used are described in Section 5.1.3 of the Risk Assessment Report. Although this approach provides a spatial gradient across the study area due to differences in monitor values for each exposure zone, this approach assumes a constant concentration within each exposure zone (i.e., no spatial gradient within a zone). Additionally, the nearest neighbor approach to assign block groups to exposure zones assumes that a monitor pertains to all locations that are closer to that monitor than to any of the others in the study area. In reality, there may be different and more variable spatial gradients in a study area than those reflected in the approach used here. This introduces significant uncertainty into the characterization of risk for the urban case studies.
- *Current NAAQS air quality scenarios*: For the location-specific urban case studies, proportional roll-up procedures were used to adjust ambient air Pb concentrations up to just meet the current NAAQS (see Sections 2.3.1 and 5.2.2.1 of the Risk Assessment Report for detailed discussion). Staff recognizes that it is extremely unlikely that Pb concentrations in urban areas would rise to meet the current NAAQS and that there is substantial uncertainty with our simulation of such conditions. In these case studies we have simulated a proportional roll-up, such that it is assumed that the current spatial distribution of air concentrations (as characterized by the current data) is maintained and increased Pb emissions contribute to increased Pb concentrations, the highest of which just meets the current standard. There are many other types of changes within a study area that could result in a similar outcome such as increases in emissions from just one specific industrial operation that could lead to air concentrations in a part of the study area that just meet the current NAAQS, while the remainder of the study area remained largely unchanged (at current conditions). For the primary Pb smelter case study, where current conditions exceed the current NAAQS, attainment of the current NAAQS was simulated using air quality modeling, emissions and source parameters used in developing the 2007 proposed revision to the State Implementation Plan for the area (see Section 3.1.1.2 of the Risk Assessment Report for details).
- *Alternative NAAQS air quality scenarios*: In all case studies, proportional roll-down procedures were used to adjust ambient air Pb concentrations downward to attain alternative NAAQS (see Sections 2.3.1 and 5.2.2.1 of the Risk Assessment Report). We recognize that there is significant uncertainty in simulating conditions associated with the implementation of emissions reduction actions to meet a lower standard. There are a variety of changes other than that represented by a proportional roll-down that could result in air concentrations that just meet lower alternative standards. For example, control measures might be targeted only at the specific area exceeding

standard, resulting in a reduction of air Pb concentrations to the alternate standard while concentrations in the rest of the study area remain unchanged (at current conditions). Consequently, there is significant uncertainty associated with estimates for the alternate NAAQS scenarios.

Limitations in Estimation of Indoor Dust and Outdoor Soil/Dust Pb Concentrations and Associated Uncertainty

- *Estimates of outdoor soil/dust Pb concentrations*: Outdoor soil/dust Pb concentrations in all air quality scenarios have been set equal to the values for the current conditions scenarios. That is, we are not simulating an impact of changes in air Pb concentrations on soil concentrations, or the associated impact on dust concentrations, blood Pb and risk estimates. In areas where air concentrations have been greater in the past, however, implementation of a reduced NAAQS might be expected to yield reduced soil Pb levels over the long term. As described in Section 2.3.3 of the Risk Assessment Report, however, there is potentially significant uncertainty associated with this specification, particularly with regard to implications for areas in which a Pb source may locate where one of comparable size had not been previously. Additionally, we note that control measures implemented to meet alternative NAAQS may result in changes to soil Pb concentrations; these are not reflected in the assessment.
- *Estimates of indoor dust Pb concentrations for the urban case studies (application of the hybrid model)*: The hybrid mechanistic-empirical model for estimating indoor dust Pb for the urban case studies (see Section 3.1.4.1 of the Risk Assessment Report) has several sources of uncertainty that could significantly impact its estimates. These include: (a) failure to consider house-to-house variability in factors related to infiltration of outdoor ambient air Pb indoors and subsequent buildup on indoor surfaces, (b) limitations in data available on the rates and efficiency of indoor dust cleaning and removal, (c) limitations in the method for converting model estimates of dust Pb loading to dust Pb concentration needed for blood Pb modeling, and (d) the approach employed to partition estimates of dust Pb concentration into “recent air” and “other” components (see Section 5.3.3.4 of the Risk Assessment Report). These last two sources of uncertainty reduce our confidence in estimates of apportionment of dust Pb between "recent air" and "other". In recognition of this limitation, we have, in evaluating exposure and risk reduction trends related to reducing ambient air Pb levels, focused on changes in total blood Pb rather than on estimates of "recent air" blood Pb. See Section 4.3.1 of the Risk Assessment report for additional discussion of uncertainty associated with indoor dust modeling for the urban case studies, and Section 5.3.3.4 of the Risk Assessment Report for discussion of a sensitivity analysis of the approach used in estimating the “other” category of indoor dust Pb.
- *Estimates of indoor dust Pb concentrations for the primary Pb smelter case study (application of the site-specific regression model):* There is uncertainty associated with the site-specific regression model applied in the remediation zone (see Section 3.1.4.2 of the Risk Assessment Report), and relatively greater uncertainty associated with its application to air quality scenarios that simulate notably lower air Pb levels. Limitations in the dataset from which the model was derived limited its form to that of

a simple regression that predicts dust Pb concentration as a function of air Pb concentration plus a constant (intercept). We recognize, however, that there may be variables in addition to air that influence dust Pb concentrations and their absence in the regression contributes uncertainty to the resulting estimates. To the extent that these unaccounted for variables are spatially related to the smelter facility Pb sources, our estimates could be biased, not with regard to the absolute dust Pb concentration, but with regard to differences in dust Pb concentration estimate between different air quality scenarios. Those differences may be overestimated because of potential overestimation of the air coefficient and underestimation of the intercept in the regression model. Examples of such not-accounted-for variables are roadside dust Pb and historically contributions to current levels of indoor dust Pb (e.g., dust Pb contributed to a house in the past that continues to contribute to current dust Pb levels). See Sections 3.1.4.2, 3.5.1.3 and 4.3.1 of the Risk Assessment Report for additional discussion.

Limitations in Estimation of Blood Pb Concentrations and Associated Uncertainty

- *Characterizing interindividual variability using a GSD*: There is uncertainty associated with the GSD specified for each case study (see Sections 3.2.3 and 5.2.2.3 of the Risk Assessment Report for additional detail on GSDs). Two factors are described here as contributors to that uncertainty. Interindividual variability in blood Pb levels for any study population (as described by the GSD) will reflect, to a certain extent, spatial variation in media concentrations, including outdoor ambient air Pb levels and indoor dust Pb levels. For each case study, there is significant uncertainty in the specification of spatial variability in ambient air Pb levels and associated indoor dust Pb levels, as noted above. In addition, there are a limited number of datasets for different types of residential child populations from which a GSD can be derived (e.g., NHANES datasets for more heterogeneous populations and individual study datasets for likely more homogeneous populations near specific industrial Pb sources). This uncertainty associated with the GSDs introduces significant uncertainty in exposure and risk estimates for the 95^{th} population percentile.
- *Exposure pathway apportionment for higher percentile blood Pb level and IQ loss estimates:* Apportionment of blood Pb levels for higher population percentiles is specified to be the same as that estimated using the central tendency estimate of blood Pb in an exposure zone. This introduces significant uncertainty into projections of pathway apportionment for higher population percentiles of blood Pb and IQ loss. In reality, pathway apportionment may shift as you consider higher exposure percentiles. For example, paint and/or drinking water exposures may increase in importance, with air-related contributions decreasing as an overall percentage of blood Pb levels and associated risk. Because of this uncertainty related to pathway apportionment, as mentioned earlier, we have placed greater emphasis on estimates of total Pb exposure and risk in evaluating the impact of the current NAAQS and alternative NAAQS relative to current conditions.

Limitations in Estimation of IQ Loss and Associated Uncertainty

- *Relating blood Pb levels to IQ loss*: Specification of the quantitative relationship between blood Pb level and IQ loss is subject to significant uncertainty at lower blood Pb levels (e.g., below 5 µg/dL concurrent blood Pb). As discussed in Section 4.2.1, there are limitations in the datasets and concentration-response analyses available for characterizing the concentration-response relationship at these lower blood Pb levels. For example, the pooled international dataset analyzed by Lanphear and others (2005) includes relatively few children with blood Pb levels below 5 µg/dL and no children with levels below 1 µg/dL (see Section 4.2.1). Blood Pb levels in this region are a particular focus in this review. For example, as is the case for mean blood Pb levels nationally in the U.S. (CD, Section 4.3.1.3), concurrent blood Pb estimates for the median of the populations simulated in this assessment fall below 5 µg/dL (see Section 4.3). In recognition of the uncertainty in specifying a quantitative concentration-response relationship at such levels, our core modeling approach involves the application of four different functions to generate a range of risk estimates (see Section 4.2.6 and Section 5.3.1 of the Risk Assessment Report). The range of absolute IQ loss seen for a given case study/air quality scenario combination when modeled using the four concentration-response functions is typically close to a factor of 3. However, we note that the relative difference (in terms of percent change) in IQ loss between individual air quality scenarios (i.e., the pattern of risk reduction across air quality scenarios) is fairly consistent across all four models. However, the function producing higher overall risk estimates (the dual linear function, stratified at 7.5 µg/dL, peak blood Pb) will also produce larger absolute reductions in IQ loss compared with the other three functions.

4.3 EXPOSURE ASSESSMENT

Exposure results generated for the full-scale analysis are summarized in Tables 4-1 and 4-2 at the end of this section. These tables include estimates of blood Pb levels for the median and 95th population percentile, respectively. Each table presents estimated blood Pb levels resulting from total Pb exposure across all pathways (policy-relevant and background), as well as estimates of percent contribution from "recent air" and "recent plus past air" exposure categories. As noted in Section 4.2.4 (and Section 3.4 of the Risk Assessment Report), given the various limitations of our modeling tools, the contribution to blood Pb levels from air-related exposure pathways and current levels of Pb emitted to the air (including via resuspension) are likely to fall between contributions attributed to “recent air” and those attributed to “recent plus past air”. Key uncertainties regarding partitioning dust Pb into “recent air” and “other” categories are summarized in Section 4.2.7.

Time limitations in preparing this Staff Paper have resulted in our providing here only a brief summary of the exposure assessment results. However, these results need to be understood in the context of the broader and more comprehensive and detailed presentation provided in the Risk Assessment Report (USEPA, 2007b). Listed below are key observations related to the

exposure assessment based on estimates presented in Tables 4-1 and 4-2. This section is organized by ambient air quality scenario category beginning with observations regarding estimated exposures under current conditions, followed by observations related to the current NAAQS, and concluding with observations regarding the alternative NAAQS scenarios.

In presenting these observations, we reference both median and 95th population percentile estimates of concurrent blood Pb levels. It is important to note that 5 percent of the child study population at each case study would have exposures above the high-end exposures presented here, although due to technical limitations we believe that it is not possible at this point to reasonably predict the distribution of exposures for that top 5 percent.

Current Conditions

This section presents observations regarding the blood Pb estimates for the current conditions scenarios.

- Current Pb concentrations for the three location-specific urban case studies in terms of maximum quarterly average are 0.09, 0.14 and 0.36 μg/m^3 for the study areas in Los Angeles, Chicago and Cleveland, respectively. In terms of maximum monthly average the values are 0.17 μg/m^{3,} 0.31 μg/m^3 and 0.56 μg/m^3 for the study areas in Los Angeles, Chicago and Cleveland, respectively.
- Median estimates of total blood Pb level range from 1.7 to 1.8 μg /dL for the three location-specific urban case studies, with the percent associated with ambient air Pb estimated to fall between the estimate for recent air (17%) and that for recent plus past air (71%) (see Table 4-1). Estimates for the 95th percentile range from 5.9 to 6.1 μg/dL with the percent associated with ambient air Pb estimated to fall between 15% and 68% (see Table 4-2).
- Two current conditions scenarios were considered for the general urban case study: 0.14 μg/m^3 as a maximum quarterly average, the mean for large urban areas, and 0.87 μg/m^3 as a maximum quarterly average, the high-end estimate for large urban areas (see Table 4-1). Median estimates of total blood Pb for these two scenarios were very similar at 1.9 and 2.1 μg/dL (see Table 4-1). In both cases, the percent associated with ambient air Pb is estimated to fall between 32% and 76% of total Pb exposure. Estimated total blood Pb levels for the 95th percentile of the distribution are 6.5 μg/dL for the mean scenario and 7.2 μg/dL for the high-end scenario (see Table 4-2).

Current NAAQS

This section presents observations regarding blood Pb estimates for the current NAAQS scenario in which ambient air Pb levels are simulated to just meet the current NAAQS level of 1.5 μg/m^3, as a maximum quarterly average.

- Estimates of median total blood Pb for the current NAAQS scenario in the three location-specific urban case studies range from 2.1 to 3.0 μg/dL with the percent

associated with ambient air estimated to fall between the estimates for recent air (50-63%) and those for recent plus past air (81-86%) (see Table 4-1). Estimates of total blood Pb exposures for the 95th percentile range from 7.4 to 10.2 ug/dL with the percent associated with ambient air estimated to fall between the estimates for recent air (31-65%) and those for recent plus past air (73-87%) (see Table 4-2). The similarity of the proportion of the contribution from ambient air Pb to total blood Pb for the median and 95th percentile child scenarios suggests the key role played by the interindividual GSD in determining 95th percentile exposures for the location-specific urban case studies (i.e., spatial gradients, which are less significant in these urban case studies, are less important in producing elevated levels of exposure). Both the median and 95th percentile blood Pb estimates for current NAAQS suggest a significant increase in blood Pb levels compared with blood Pb levels estimated for the current conditions (see last section). Specifically, median blood Pb levels are approximately 1 µg/dL higher under the current NAAQS scenario compared with the current conditions scenario, while 95th percentile levels range from 1.5 to 4 µg/dL higher under the current NAAQS scenario.

- Blood Pb estimates for the general urban case study are slightly higher than estimates for the location-specific urban case studies, with a median estimate for total blood Pb of 3.1 µg/dL and a 95th percentile estimate of 10.6 µg/dL (see Tables 4-1 and 4-2, respectively). Contributions of ambient air Pb to total blood Pb are somewhat higher in this case study than they are for the location-specific urban case studies. This finding is expected since the location-specific urban case study assumes that the entire study area is at the current NAAQS level, while the location-specific urban case studies will have portions of the study areas at that level and the remainder at levels notably lower than the current NAAQS. Estimates for the median child for both the mean and high-end current conditions scenarios indicate a decrease of about 1 µg/dL in total blood Pb for the current conditions scenario compared with the current NAAQS scenario. The difference is even more pronounced for the 95th percentile child, with the mean current conditions scenario differing by 4 µg/dL from the current NAAQS scenario and the high-end current conditions scenario differing by 3.4 µg/dL.

- For the primary Pb smelter case study (full study area) the estimate of median total blood Pb is 1.5 µg/dL with 53% of this resulting from recent plus past air (see Table 4-1). The 95th percentile estimate for total blood Pb is 4.6 µg/dL, with 61% of this coming from recent plus past air (see Table 4-2). Both the median and 95th percentile total blood Pb estimates are significantly lower than those generated for the urban scenarios, reflecting in part the fact that characterization of ambient air Pb levels for the primary Pb smelter did not consider contributions from other sources besides the smelter (e.g., resuspension of road dust, other industrial sources) that might contribute to exposures further from the facility. This underprediction bias is more important for areas further from the facility which include a large segment of the modeled study population. By contrast, the general urban case study and location-specific urban case studies are based on monitoring data, which will reflect the contribution from all Pb sources in the vicinity of the monitors.

- Blood Pb estimates for the 1.5 km subarea of the primary Pb smelter case study were markedly elevated for the current NAAQS scenario compared with the corresponding

estimates for the full study area. This reflects the fact that the analysis of the subarea focused on a subpopulation experiencing significantly greater ambient air Pb levels (due to their proximity to the facility). The median estimate of total blood Pb level is 4.6 µg/dL with 87% of that exposure resulting from recent plus past air Pb (see Table 4-1). The 95th percentile estimate of total blood Pb level is 12.3 µg/dL, with 83% of that exposure resulting from recent plus past air Pb (see Table 4-2). The larger fraction of total Pb exposure associated with ambient air Pb for the subarea compared with the full study area (83 to 87% compared with 53 to 61%) indicates that elevated blood Pb levels for the subarea result from significantly greater ambient air Pb contributions.

Alternative NAAQS

This section presents observations regarding the blood Pb estimates resulting from modeling of exposure assuming that each of the case studies just meets each of the alternative NAAQS levels.

- The current air Pb concentrations in the study areas of the three location-specific urban case studies fall near and within the upper end of the range of alternate NAAQS considered (i.e., 0.2 µg/m^3 maximum monthly average to 0.5 µg/m^3 maximum quarterly average). In modeling alternate NAAQS for these case studies, we only considered those alternative NAAQS that were either equal to or lower than current conditions at each location. This meant that the three lowest alternative NAAQS were considered for Chicago, all of the alternative NAAQS were considered for Cleveland and only the two lowest were considered for Los Angeles. The remaining case studies were evaluated for the full set of alternative NAAQS.
- For the two location-specific urban case studies at which the higher alternative NAAQS (0.5 and 0.2 µg/m^3, maximum monthly average and 0.2 µg/m^3 maximum quarterly average) were simulated, median estimates of total blood Pb levels range from 1.7 to 1.8 µg/dL (see Table 4-1). This range is similar to the estimates for current conditions for the three case studies, which is expected given the similarity of current conditions to these alternative NAAQS. Estimates of median total blood Pb levels for the lowest alternative NAAQS (0.02 and 0.05 µg/m^3 maximum monthly average) show a slight reduction compared with current conditions (i.e., equal to or less than a 0.2 µg/dL reduction - see Table 4-1). Estimates of 95th percentile total blood Pb levels for the higher alternative NAAQS (0.5 and 0.2 µg/m^3 maximum monthly average and 0.2 µg/m^3 maximum quarterly average) range from 5.7 to 6.0 µg/dL (see Table 4-2). These estimates differ from those for current conditions by about 0.4 µg/dL or less. Estimates of 95th percentile total blood Pb levels under the lowest alternative NAAQS (0.02 and 0.05 µg/m^3 maximum monthly average) shows a significantly larger reduction in blood Pb levels compared to current conditions, with this drop ranging from roughly 0.5 to 0.8 µg/dL (see Table 4-2).
- The primary Pb smelter (full study area) shows no discernable reduction in median total blood Pb levels across any of the alternative NAAQS (see Table 4-1), again reflecting the fact that characterization of ambient air Pb levels for this study area did not consider sources other than the facility and consequently, portions of the study area

further from the facility have relatively low ambient air-related exposures. However, a moderate reduction in 95th percentile total blood Pb levels is seen across the alternative NAAQS, with the highest alternative NAAQS (0.5 µg/m^3 maximum monthly average) estimated at 4.2 µg/dL and the lowest alternate NAAQS (0.02 µg/m^3 maximum monthly average) estimated at 3.8 µg/dL (see Table 4-2).

- The primary Pb smelter (1.5km subarea) presents a much more substantial trend in reduction of estimated median total blood Pb levels across alternative NAAQS. Median total blood Pb levels are estimated at 3.2 µg/dL for the highest alternative NAAQS (0.5 µg/m^3 maximum monthly average), decreasing to 2.3 µg/dL for the intermediate alternative NAAQS (0.2 µg/m^3 maximum monthly average) and finally dropping to 1.6 µg/dL for the lowest alternative NAAQS (0.02 µg/m^3 maximum monthly average) (see Table 4-1). This trend is also seen for 95th percentile estimates of total blood Pb levels, but it is even stronger, with the highest alternative NAAQS estimated at 8.5 µg/dL, dropping to 6.1 µg/dL (for the 0.2 µg/m^3 maximum monthly average NAAQS) and reaching 4.2 µg/dL with the lowest alternative NAAQS (see Table 4-2).

Table 4-1. Summary of blood Pb estimates for median total blood Pb.

Air Quality Scenario (and case study)	Policy-relevant source contribution (percent) of total blood Pb		Concurrent blood Pb concentration (total Pb exposure) [a]
	Recent Air [b]	Recent Air plus Past Air [b]	
Location-specific (Chicago)			
Current NAAQS (1.5 µg/m^3, max quarterly)	63%	83%	3.0
Current conditions (0.14 µg/m^3 max quarterly; 0.31 µg/m^3 max monthly)	22%	67%	1.8
Alternative NAAQS (0.2 µg/m^3, max monthly)	17%	67%	1.8
Alternative NAAQS (0.05 µg/m^3, max monthly)	6%	69%	1.6
Alternative NAAQS (0.02 µg/m^3, max monthly)	1%	63%	1.6
Location-specific (Cleveland)			
Current NAAQS (1.5 µg/m^3, max quarterly)	57%	86%	2.1
Current conditions (0.36 µg/m^3 max quarterly; 0.56 µg/m^3 max monthly)	17%	67%	1.8
Alternative NAAQS (0.5 µg/m^3, max monthly)	39%	72%	1.8
Alternative NAAQS (0.2 µg/m^3, max quarterly)	12%	65%	1.7
Alternative NAAQS (0.2 µg/m^3, max monthly)	6%	65%	1.7
Alternative NAAQS (0.05 µg/m^3, max monthly)	1%	63%	1.6
Alternative NAAQS (0.02 µg/m^3, max monthly)	1%	63%	1.6
Location-specific (Los Angeles)			
Current NAAQS (1.5 µg/m^3, max quarterly)	50%	81%	2.6
Current conditions (0.09 µg/m^3 max quarterly; 0.17 µg/m^3 max monthly)	18%	71%	1.7
Alternative NAAQS (0.05 µg/m^3, max monthly)	13%	69%	1.6
Alternative NAAQS (0.02 µg/m^3, max monthly)	6%	63%	1.6
General urban			
Current NAAQS (1.5 µg/m^3, max quarterly)	61%	84%	3.1
Alternative NAAQS (0.5 µg/m^3, max monthly)	41%	73%	2.2
Current conditions –high-end (0.87 µg/m^3, max quarterly)	38%	76%	2.1
Alternative NAAQS (0.2 µg/m^3, max quarterly)	35%	75%	2.0
Current conditions - mean (0.14 µg/m^3, max quarterly)	32%	74%	1.9
Alternative NAAQS (0.2 µg/m^3, max monthly)	26%	74%	1.9
Alternative NAAQS (0.05 µg/m^3, max monthly)	12%	65%	1.7
Alternative NAAQS (0.02 µg/m^3, max monthly)	6%	69%	1.6
Primary Pb smelter - *full study area*			
Current NAAQS (1.5 µg/m^3, max quarterly)	NA	53%	1.5
Alternative NAAQS (0.5 µg/m^3, max monthly)		79%	1.4
Alternative NAAQS (0.2 µg/m^3, max quarterly)		50%	1.4
Alternative NAAQS (0.2 µg/m^3, max monthly)		36%	1.4
Alternative NAAQS (0.05 µg/m^3, max monthly)		50%	1.4
Alternative NAAQS (0.02 µg/m^3, max monthly)		64%	1.4
Primary Pb smelter - ***1.5km study area***			
Current NAAQS (1.5 µg/m^3, max quarterly)	NA	87%	4.6
Alternative NAAQS (0.5 µg/m^3, max monthly)		81%	3.2
Alternative NAAQS (0.2 µg/m^3, max quarterly)		72%	2.5
Alternative NAAQS (0.2 µg/m^3, max monthly)		78%	2.3
Alternative NAAQS (0.05 µg/m^3, max monthly)		65%	1.7
Alternative NAAQS (0.02 µg/m^3, max monthly)		69%	1.6

a - All values are rounded to one decimal place.
b -The term "past air" includes contributions from the outdoor soil/dust contribution to indoor dust, historical air contribution to indoor dust, and outdoor soil/dust pathways; "recent air" refers to contributions from inhalation of ambient air Pb or ingestion of indoor dust Pb predicted to be associated with outdoor ambient air Pb levels, with outdoor ambient air also potentially including resuspended, previously deposited Pb (see Section 2.4.3 of the Risk Assessment Report).

Table 4-2. Summary of blood Pb level estimates for 95th percentile total blood Pb.

Air Quality Scenario (and case study)	Policy-relevant contribution (percent) of total blood Pb		Concurrent blood Pb concentration (total Pb exposure) [a]
	Recent Air [b]	Recent plus Past Air [b]	
Location-specific (Chicago)			
Current NAAQS (1.5 µg/m^3, max quarterly)	65%	87%	10.2
Current conditions (0.14 µg/m^3 max quarterly; 0.31 µg/m^3 max monthly)	18%	68%	6.0
Alternative NAAQS (0.2 µg/m^3, max monthly)	25%	70%	6.0
Alternative NAAQS (0.05 µg/m^3, max monthly)	5%	65%	5.5
Alternative NAAQS (0.02 µg/m^3, max monthly)	4%	67%	5.4
Location-specific (Cleveland)			
Current NAAQS (1.5 µg/m^3, max quarterly)	31%	73%	7.4
Current conditions (0.36 µg/m^3 max quarterly; 0.56 µg/m^3 max monthly)	15%	67%	6.1
Alternative NAAQS (0.5 µg/m^3, max monthly)	25%	70%	6.0
Alternative NAAQS (0.2 µg/m^3, max quarterly)	12%	67%	5.8
Alternative NAAQS (0.2 µg/m^3, max monthly)	7%	67%	5.7
Alternative NAAQS (0.05 µg/m^3, max monthly)	2%	65%	5.4
Alternative NAAQS (0.02 µg/m^3, max monthly)	2%	66%	5.3
Location-specific (Los Angeles)			
Current NAAQS (1.5 µg/m^3, max quarterly)	52%	80%	8.9
Current conditions (0.09 µg/m^3 max quarterly; 0.17 µg/m^3 max monthly)	19%	68%	5.9
Alternative NAAQS (0.05 µg/m^3, max monthly)	9%	67%	5.5
Alternative NAAQS (0.02 µg/m^3, max monthly)	4%	65%	5.4
General urban			
Current NAAQS (1.5 µg/m^3, max quarterly)	60%	83%	10.6
Alternative NAAQS (0.5 µg/m^3, max monthly)	39%	76%	7.4
Current conditions –high-end (0.87 µg/m^3, max quarterly)	38%	75%	7.2
Alternative NAAQS (0.2 µg/m^3, max quarterly)	34%	74%	6.8
Current conditions - mean (0.14 µg/m^3, max quarterly)	29%	72%	6.5
Alternative NAAQS (0.2 µg/m^3, max monthly)	27%	72%	6.4
Alternative NAAQS (0.05 µg/m^3, max monthly)	12%	68%	5.7
Alternative NAAQS (0.02 µg/m^3, max monthly)	7%	67%	5.5
Primary Pb smelter - *full study area*			
Current NAAQS (1.5 µg/m^3, max quarterly)	NA	61%	4.6
Alternative NAAQS (0.5 µg/m^3, max monthly)		74%	4.2
Alternative NAAQS (0.2 µg/m^3, max quarterly)		60%	4.0
Alternative NAAQS (0.2 µg/m^3, max monthly)		63%	4.0
Alternative NAAQS (0.05 µg/m^3, max monthly)		50%	3.8
Alternative NAAQS (0.02 µg/m^3, max monthly)		84%	3.8
Primary Pb smelter - ***1.5km study area***			
Current NAAQS (1.5 µg/m^3, max quarterly)	NA	83%	12.3
Alternative NAAQS (0.5 µg/m^3, max monthly)		89%	8.5
Alternative NAAQS (0.2 µg/m^3, max quarterly)		89%	6.6
Alternative NAAQS (0.2 µg/m^3, max monthly)		80%	6.1
Alternative NAAQS (0.05 µg/m^3, max monthly)		78%	4.5
Alternative NAAQS (0.02 µg/m^3, max monthly)		71%	4.2

a - All values are rounded to one decimal place.
b -The term "past air" includes contributions from the outdoor soil/dust contribution to indoor dust, historical air contribution to indoor dust, and outdoor soil/dust pathways, while "recent air" refers to contributions from inhalation of ambient air Pb or ingestion of indoor dust Pb predicted to be associated with outdoor ambient air Pb levels, with outdoor ambient air also potentially including resuspended, previously deposited Pb (see Section 2.4.3 of the Risk Assessment Report).

4.4 RISK ASSESSMENT

Risk results generated for the full-scale analysis are summarized in Tables 4-3 through 4-8 at the end of this section. These tables present three types of risk metrics:

- **Estimates of IQ loss for all air quality scenarios**: Tables 4-3 and 4-4 present IQ loss estimates for total Pb exposure for each of the air quality scenarios simulated for each case study. Table 4-3 presents estimates for the population median and Table 4-4 presents results for the 95th percentile. To reflect the variation in estimates derived from the four different concentration-response functions, the lowest and highest estimates are presented for each scenario along with estimates for the LLL model. These are labeled in these tables as (a) low C-R function (this is either the dual linear with stratification at 10 µg/dL, peak blood Pb, or the log-linear with cutpoint, depending on the blood Pb level); (b) the LLL C-R function (log-linear with low-exposure linearization); and (c) the high C-R function (the dual linear with stratification at 7.5 µg/dL, peak blood Pb).
- **Estimates of IQ loss under the current NAAQS air quality scenario**: Tables 4-5 and 4-8 present estimated IQ loss for total Pb exposure based on simulation of just meeting the current NAAQS for the case studies to which the core modeling approach was applied. Specifically, Table 4-5 presents estimates of total Pb-related IQ loss for the population median, and Table 4-6 presents estimates for the 95th percentile. Both of these tables present estimated total IQ loss (reflecting both policy-relevant pathways and background sources) as well as the estimated range of IQ loss associated with policy-relevant exposures alone (bounded by estimates for "recent air" and for "recent plus past air").
- **IQ loss incidence estimates for the three location-specific urban case studies**: Estimates of the number of children projected to have total Pb-related IQ loss greater than one point are summarized in Table 4-7, and similar estimates for IQ loss greater than 7 points are summarized in 4-8. Also presented are the changes in incidence of the current NAAQS and alternative NAAQS scenarios compared to current conditions. Estimates are presented for each of the four concentration-response functions used in the core analysis. The complete set of incidence results is presented in Risk Assessment Report Appendix O, Section O.3.4.

Time limitations in preparing this Staff Paper have resulted in our providing here only a brief summary of the risk assessment results. These results, however, need to be understood in the context of the broader and more comprehensive and detailed presentation provided in the Risk Assessment Report (USEPA, 2007b). Listed below are key observations related to the risk assessment, and as such, they draw on estimates presented in Tables 4-3 through 4-8. These observations are organized by type of ambient air quality scenario beginning with observations regarding the current conditions scenarios, followed by observations regarding the current NAAQS scenarios, and concluding with observations regarding the set of alternative NAAQS evaluated.

As described in Section 4.2.5 above, we included four blood Pb concentration-response functions relating blood Pb and IQ loss in the core modeling approach to provide coverage for uncertainty in this key modeling step. However, for the reasons described in Section 4.2.1, we place greater confidence in one of these functions (the log-linear with low-exposure linearization, or LLL function). We note that, risk estimates generated using the LLL model fall intermediate between estimates generated using the other three functions. Estimates derived using the log-linear function with cutpoint and the dual-linear function with stratification at 10 µg/dL (peak blood Pb) yield the lowest risk estimates and the dual-linear function with stratification at 7.5 µg/dL (peak blood Pb) yields the highest risk estimates. Because of the greater confidence placed in the LLL function and because it generates risk estimates generally bounded by the other three functions, we emphasize risk estimates generated using the LLL function in this discussion. All risk estimates discussed in the observations below, unless otherwise noted, were generated using the LLL function (i.e., results presented in Tables 4-3 through 4-8 under the LLL heading).

It is important to point out that the range of absolute IQ loss estimates generated using the four models for a given case study and air quality scenario is large, typically around a factor of 3. This can be seen by considering the difference in total IQ loss between the "low C-R function" and "high C-R function" estimates presented for any case study and air quality scenario combination in Tables 4-3 and 4-4. However, the relative (proportional) change in IQ loss across air quality scenarios (i.e., the pattern of risk reduction across air quality scenarios for the same case study) is fairly consistent across all four models. This suggests that there may be significant uncertainty in estimates of absolute IQ loss for a median or 95^{th} percentile child with exposures related to a given ambient air Pb level. Accordingly, we have greater confidence in predicting incremental changes in IQ loss across air quality scenarios.

In presenting these risk observations, as with presentation of the exposure observations, we reference both median and 95^{th} percentile estimates of total IQ loss. It is important to note that, similar to the exposure assessment, 5 percent of the child study population at each case study would have risk levels above the 95^{th} percentile IQ loss estimates presented here, although due to technical limitations of our modeling tools, we believe that it is not possible at this point to reasonably predict the distribution of risk levels for that top 5 percent.

Current Conditions

The following observations are with regard to estimation of Pb-related IQ loss for current conditions at each of the case studies. Unless otherwise stated, all risk estimates discussed below were generated using the LLL model (designated as "LLL" in the risk results tables).

- As mentioned earlier in discussing the exposure results, current conditions for the three location-specific urban case studies in terms of maximum quarterly average air Pb concentrations are 0.09, 0.14 and 0.36 $\mu g/m^3$ for the study areas in Los Angeles, Chicago and Cleveland, respectively. In terms of maximum monthly average the ambient air Pb levels range from 0.17 $\mu g/m^3$ (maximum monthly average for Los Angeles case study) to 0.56 $\mu g/m^3$ maximum monthly average for Cleveland case study. The estimate for the Chicago case study is between these at 0.31 $\mu g/m^3$ as a maximum monthly average.
- For the three location-specific urban case studies, median total IQ loss is estimated at 4.2 points using the LLL function and from 1.4 to as much as 5.2 points across functions (see Table 4-3). Estimates for 95th percentile total IQ loss range from 7.5 to 7.6 points for the LLL and as much as 4.1 to 11.4 points across all four functions (see Table 4-4).
- Two current conditions scenarios were considered for the general urban case study: one based on the mean value for ambient air Pb levels in large urban areas (0.14 $\mu g/m^3$ as a maximum quarterly average) and a high-end ambient air Pb level in large urban areas (0.87 $\mu g/m^3$ as a maximum quarterly average). Estimates of median total IQ loss for these two scenarios were very similar at 4.5 and 4.7 points for the mean and high-end current conditions, respectively. Estimated 95th percentile total IQ losses are also fairly similar for the two current conditions scenarios, with the mean scenario at 7.7 points and the high-end scenario at 8 points. The range of estimates across all concentration-response models is similar to that for the location-specific urban areas (see above).

Current NAAQS

This section presents observations regarding estimates of Pb-related IQ loss for the current NAAQS scenario for each of the case studies. Note, that discussion of the contribution of recent air and recent plus past air to total IQ loss has been reserved for the current NAAQS air quality scenario, since this scenario was modeled for all of the case studies. As noted above, all risk estimates discussed below were generated using the LLL model (i.e., designated as "LLL" in the risk results tables), unless otherwise noted.

- Estimates of median total IQ loss for the current NAAQS scenarios in the three location-specific urban case studies range from 4.7 to 5.6 points, with between 2.7 and 3.4 of that coming from recent air and 3.9 to 4.7 coming from recent plus past air (see Table 4-5). Estimates of 95th percentile total IQ loss range from 8.1 to 9 points, with between 2.6 and 5.8 points coming from recent air and 5.9 and 7.6 points coming from recent plus past air (see Table 4-6). Both the median and 95th percentile risk estimates for current NAAQS are significantly higher than those for current conditions. Specifically, median IQ loss is approximately 0.5 to 1 point higher under the current NAAQS, while 95th percentile levels are 0.5 to 1.5 points higher under the current NAAQS (see Tables 4-3 and 4-4, respectively). While the size of the increase in blood Pb levels is significantly higher for the 95th percentile child than for the median, the

size of risk increase is similar because the blood Pb increase for the 95th percentile is taking place in a flatter portion of the concentration-response function. Conversely, blood Pb increases for the median population percentile are occurring in a steeper portion of the curve, thereby resulting in a similar increase in IQ loss for the median and 95th percentile child. Risk estimates generated using the other three IQ loss concentration-response functions provides a considerably wider range of predicted IQ loss. For example, the estimated median total IQ loss ranges from 1.7 to 8.8 points when all four concentration-response models are considered (compared with 4.7 to 5.6 points presented above for the LLL model alone - see Table 4-3).

- Population risk modeling completed for the three location-specific urban case studies provides estimates of changes in the number of children with Pb-related IQ loss greater than one point for the current NAAQS scenario compared to the current conditions scenario. It is estimated that an additional 1% of the modeled child populations at the three urban locations would have total IQ loss greater than 1.0 point under the current NAAQS scenario. Specifically, the Chicago study area would have nearly 6,000 more children with this magnitude of Pb-related IQ loss (from a total study population of some 400,000), Cleveland would have approximately 100 (from a total study population of 14,000) and Los Angeles would have approximately 4,000 more children (from a total study population of approximately 370,000) with total Pb-related IQ loss greater than 1.0 point under the current NAAQS scenario (see Table 4-7).
- By contrast, it is estimated that an additional 5% to 17% of the modeled child populations at the three location-specific case study areas would move from having total IQ loss below 7 IQ points to above 7 IQ points if current conditions increased to levels near the current NAAQS. Specifically, the Chicago study area would have some 70,000 more children with Pb-related IQ loss greater than 7 points, the Cleveland study area would have some 600 more and Los Angeles would have some 35,000 more children with this magnitude of Pb-related IQ loss (see Table 4-8). The increases in total IQ loss estimates are distributed across the distribution for each population with some of the children moving between higher IQ loss categories (above the 7 point demarcation). The prediction that shifting ambient air levels up to the current NAAQS would have a greater impact on the number of children with Pb-related IQ loss of greater than 7 points compared with the number with greater than one point of IQ loss reflects the overall shape of the distribution of total Pb-related IQ loss estimates for these study areas under current conditions. The majority of children in the three urban study areas are projected to have IQ loss due to total Pb exposure that is significantly greater than 1 point (the median is around 4 points - see Table 4-3). Therefore, a hypothetical increase in ambient air Pb levels to just meet the current NAAQS is predicted to yield a larger change in the number of children with relatively higher IQ loss (in the range of 7 points) than on the number of children with IQ loss in the range of 1, since current conditions estimates for Pb-related IQ loss are already well above 1 point.
- Current NAAQS scenario risk estimates for the general urban case study are slightly higher than those for the location-specific urban case studies, with estimates for median total IQ loss of 5.8 points and 95th percentile estimates of 9.1 points of loss (see Tables 4-3 and 4-4, respectively). The fraction of IQ loss associated with ambient air Pb

exposure for the general urban case study is similar to that estimated for the location-specific urban case studies. Total Pb-related IQ loss estimates (at both the median and 95th percentile) are about 1 point higher than those for the two current conditions scenarios (see Tables 4-7 and 4-8). The similarity of the change in risk for the median and 95th population percentiles (despite greater differences in blood Pb level reductions), reflects the different slopes of the concentration-response functions at the different blood Pb levels.

- The primary Pb smelter case study (full study area) had an estimated median total IQ loss for the current NAAQS scenario of 3.8 points, with approximately 1.9 points resulting from recent plus past air (see Table 4-5). The estimate of 95th percentile total IQ loss for the current NAAQS scenario is 6.8 points, with 4.2 points of this coming from recent plus past air (see Table 4-6).
- The 1.5 km subarea of the primary Pb smelter case study had markedly elevated risk levels for the current NAAQS scenario, compared with the full study area. This reflects the fact that the analysis of the subarea focused on a subpopulation experiencing significantly greater ambient air Pb levels due to proximity to the facility. Median total IQ loss is estimated at 6.8 points with 6.0 of those points resulting from recent plus past air Pb (see Table 4-5). The estimate for 95th percentile total IQ loss is 9.5 points, with 8.0 of those points resulting from recent plus past air Pb (see Table 4-6). The larger fraction of total Pb risk associated with ambient air Pb for the subarea compared with the full study area indicates the greater impact of ambient air Pb in this area.

Alternative NAAQS

This section presents observations regarding modeling of Pb-related IQ loss for each of the case studies for the alternative NAAQS scenarios. As noted above, all risk estimates discussed below were generated using the LLL model (i.e., designated as "LLL" in the risk results tables), unless otherwise stated.

- In modeling the alternate NAAQS for the three location-specific urban case studies, we assessed those alternative NAAQS that were either equal to or lower than current conditions at each location. Thus, the three lowest alternative NAAQS were considered for Chicago, all of the alternative NAAQS were considered for Cleveland and only the two lowest were considered for Los Angeles. The remaining case studies were evaluated for the full range of alternative NAAQS.
- For the three location-specific urban case studies, median total IQ loss for the higher alternative NAAQS (0.5 and 0.2 $\mu g/m^3$ as monthly maximum average and 0.2 $\mu g/m^3$as a maximum quarterly average) is estimated to range from 4.1 to 4.2 points (see Table 4-3). This range is very close to the current conditions estimates for these three case studies, as would be expected since these alternative NAAQS fall near the range of current conditions at these locations. Estimates of median total IQ loss for the lower alternative NAAQS levels (0.02 and 0.05 $\mu g/m^3$ as a maximum monthly average) show a slight reduction in total IQ loss for the median child compared with current

conditions (i.e., equal to or less than a 0.2 IQ point reduction). Estimates of 95th percentile IQ loss for the higher alternative NAAQS (0.5 and 0.2 μg/m^3 as monthly maximum average and 0.2 μg/m^3 as a maximum quarterly average) range from 7.4 to 7.5 points (see Table 4-4). Under the lower alternative NAAQS levels (0.02 and 0.05 μg/m^3 maximum monthly average) 95th percentile IQ loss estimates also show a slight reduction in the degree of IQ loss, compared to current conditions, with this drop ranging from roughly 0.1 to 0.3 IQ points. The relatively lower reduction in IQ loss compared with blood Pb levels for the 95th percentile child (between the intermediate and the lower alternative NAAQS levels) reflects the IQ loss concentration-response functions which have a flatter curve at the higher blood Pb levels associated with the 95th percentile child, which translates into reduced magnitudes of change in IQ loss.

- Population risk modeling in the three location-specific urban case studies for a simulated reduction in ambient air Pb levels from current conditions to just meeting the lower alternative NAAQS levels (0.02 and 0.05 μg/m3 as a monthly maximum average) projects that approximately 0.5 to 1% of the modeled child populations involved would have total IQ loss shift from above one IQ point to below one IQ point. The size of the reduction in terms of number of children is approximately 3,000 children for the Chicago study area, 100 for the Cleveland study area and 2,000 for the Los Angeles area (see Table 4-7).

- By contrast, a comparison of the scenarios for the lower NAAQS to current conditions for the location-specific urban areas indicates that approximately 1 to 2 % of the total modeled child populations involved would have total IQ loss shift from above 7 point to below 7 points as a result of the decrease in ambient air Pb levels. The size of the reduction in terms of number of children is approximately 8,000 children for the Chicago study area, 300-400 for the Cleveland study area and 5,000 for the Los Angeles study area (see Table 4-8).

- The primary Pb smelter (full study area) is estimated to have a 0.1 point reduction in total IQ loss for the median population percentile across any of the alternative NAAQS (see Table 4-3). A somewhat larger reduction in total IQ loss (0.3 points) is estimated for the 95th population percentile with the highest alternative NAAQS (0.5 μg/m^3 maximum monthly average) estimated at 6.6 points and the lowest alternative NAAQS level (0.02 μg/m^3 maximum monthly average) estimated at 6.3 μg/dL (Table 4-4).

- The 1.5 km subarea of the primary Pb smelter risk estimates indicate a much more substantial trend in total IQ loss reduction across alternative NAAQS scenarios for the median population percentile. Estimates of total Pb-related IQ loss are 5.8 points for the highest alternative NAAQS (0.5 μg/m^3 maximum monthly average), decreasing to 5.0 points with the intermediate alternative NAAQS (0.2 μg/m^3 maximum monthly average) and dropping to 4.0 points with the lowest alternative NAAQS level (0.02 μg/m^3 maximum monthly average - see Table 4-3). This trend is also seen with the 95th population percentile, with the magnitude of IQ loss reduction matching that seen for the median population percentile. Specifically, for the highest alternative NAAQS the IQ loss is estimated at 8.5 points, dropping to 7.6 points for the 0.2 μg/m^3 maximum monthly average, and reaching 6.5 points for the lowest alternative NAAQS (see Table 4-4).

Table 4-3. Summary of risk estimates for medians of total-exposure risk distributions.

Case Study and Air Quality Scenario	Points IQ loss (total Pb exposure)[a]		
	Low C-R function estimate	LLL[b]	High C-R function estimate
Location-specific (Chicago)			
Current NAAQS (1.5 μg/m³, max quarterly)	2.4	5.6	8.8
Current conditions (0.14 μg/m³ max quarterly; 0.31 μg/m³ max monthly)	1.4	4.2	5.2
Alternative NAAQS (0.2 μg/m³, max monthly)	1.4	4.2	5.2
Alternative NAAQS (0.05 μg/m³, max monthly)	1.3	4.0	4.8
Alternative NAAQS (0.02 μg/m³, max monthly)	1.3	4.0	4.7
Location-specific (Cleveland)			
Current NAAQS (1.5 μg/m³, max quarterly)	1.7	4.7	6.3
Current conditions (0.36 μg/m³ max quarterly; 0.56 μg/m³ max monthly)	1.4	4.2	5.2
Alternative NAAQS (0.5 μg/m³, max monthly)	1.4	4.2	5.2
Alternative NAAQS (0.2 μg/m³, max quarterly)	1.4	4.1	5.0
Alternative NAAQS (0.2 μg/m³, max monthly)	1.3	4.1	4.9
Alternative NAAQS (0.05 μg/m³, max monthly)	1.3	4.0	4.7
Alternative NAAQS (0.02 μg/m³, max monthly)	1.2	3.9	4.6
Location-specific (Los Angeles)			
Current NAAQS (1.5 μg/m³, max quarterly)	2.1	5.3	7.7
Current conditions (0.09 μg/m³ max quarterly; 0.17 μg/m³ max monthly)	1.4	4.2	5.1
Alternative NAAQS (0.05 μg/m³, max monthly)	1.3	4.0	4.8
Alternative NAAQS (0.02 μg/m³, max monthly)	1.3	4.0	4.7
General Urban			
Current NAAQS (1.5 μg/m³, max quarterly)	2.5	5.8	9.2
Alternative NAAQS (0.5 μg/m³, max monthly)	1.7	4.8	6.4
Current conditions – high-end (0.87 μg/m³ max quarterly)	1.7	4.7	6.3
Alternative NAAQS (0.2 μg/m³, max quarterly)	1.6	4.6	5.9
Current conditions - mean (0.14 μg/m³ max quarterly)	1.5	4.5	5.6
Alternative NAAQS (0.2 μg/m³, max monthly)	1.5	4.4	5.6
Alternative NAAQS (0.05 μg/m³, max monthly)	1.3	4.1	5.0
Alternative NAAQS (0.02 μg/m³, max monthly)	1.3	4.0	4.8
Primary Pb smelter - full study area			
Current NAAQS (1.5 μg/m³, max quarterly)	1.2	3.8	4.4
Alternative NAAQS (0.5 μg/m³, max monthly)	1.0	3.7	4.2
Alternative NAAQS (0.2 μg/m³, max quarterly)	0.9	3.6	4.2
Alternative NAAQS (0.2 μg/m³, max monthly)	0.9	3.6	4.1
Alternative NAAQS (0.05 μg/m³, max monthly)	0.9	3.6	4.0
Alternative NAAQS (0.02 μg/m³, max monthly)	0.9	3.6	4.1
Primary Pb smelter - 1.5km subarea			
Current NAAQS (1.5 μg/m³, max quarterly)	3.7	6.8	11.2
Alternative NAAQS (0.5 μg/m³, max monthly)	2.6	5.8	9.4
Alternative NAAQS (0.2 μg/m³, max quarterly)	2.0	5.2	7.4
Alternative NAAQS (0.2 μg/m³, max monthly)	1.9	5.0	6.9
Alternative NAAQS (0.05 μg/m³, max monthly)	1.4	4.2	5.1
Alternative NAAQS (0.02 μg/m³, max monthly)	1.3	4.0	4.8

a - Estimates below 1.0 are rounded to one decimal place, all values below 0.05 are presented as <0.1 and values between 0.05 and 0.1 as 0.1. All values above 1.0 are rounded to the nearest whole number.
b-Log-linear with low-exposure linearization concentration-response function.

Table 4-4. Summary of risk estimates for 95th percentile of total-exposure risk distributions.

Case Study and Air Quality Scenario	Points IQ loss (total Pb exposure)[a]		
	Low C-R function estimate	LLL[b]	High C-R function estimate
Location-specific (Chicago)			
Current NAAQS (1.5 μg/m³, max quarterly)	4.7	9.0	12.1
Current conditions (0.14 μg/m³ max quarterly; 0.31 μg/m³ max monthly)	4.1	7.5	11.4
Alternative NAAQS (0.2 μg/m³, max monthly)	4.1	7.5	11.4
Alternative NAAQS (0.05 μg/m³, max monthly)	4.1	7.3	11.3
Alternative NAAQS (0.02 μg/m³, max monthly)	4.1	7.3	11.3
Location-specific (Cleveland)			
Current NAAQS (1.5 μg/m³, max quarterly)	4.3	8.1	11.6
Current conditions (0.36 μg/m³ max quarterly; 0.56 μg/m³ max monthly)	4.1	7.6	11.4
Alternative NAAQS (0.5 μg/m³, max monthly)	4.1	7.5	11.4
Alternative NAAQS (0.2 μg/m³, max quarterly)	4.1	7.4	11.3
Alternative NAAQS (0.2 μg/m³, max monthly)	4.1	7.4	11.3
Alternative NAAQS (0.05 μg/m³, max monthly)	4.0	7.2	11.3
Alternative NAAQS (0.02 μg/m³, max monthly)	4.0	7.2	11.3
Location-specific (Los Angeles)			
Current NAAQS (1.5 μg/m³, max quarterly)	4.5	8.6	11.8
Current conditions (0.09 μg/m³ max quarterly; 0.17 μg/m³ max monthly)	4.1	7.5	11.4
Alternative NAAQS (0.05 μg/m³, max monthly)	4.1	7.3	11.3
Alternative NAAQS (0.02 μg/m³, max monthly)	4.0	7.2	11.3
General Urban			
Current NAAQS (1.5 μg/m³, max quarterly)	4.7	9.1	12.1
Alternative NAAQS (0.5 μg/m³, max monthly)	4.3	8.1	11.6
Current conditions – high-end (0.87 μg/m³ max quarterly)	4.3	8.0	11.6
Alternative NAAQS (0.2 μg/m³, max quarterly)	4.2	7.9	11.5
Current conditions - mean (0.14 μg/m³ max quarterly)	4.2	7.7	11.5
Alternative NAAQS (0.2 μg/m³, max monthly)	4.2	7.7	11.4
Alternative NAAQS (0.05 μg/m³, max monthly)	4.1	7.4	11.3
Alternative NAAQS (0.02 μg/m³, max monthly)	4.1	7.3	11.3
Primary Pb smelter - full study area			
Current NAAQS (1.5 μg/m³, max quarterly)	3.7	6.8	11.2
Alternative NAAQS (0.5 μg/m³, max monthly)	3.4	6.6	11.1
Alternative NAAQS (0.2 μg/m³, max quarterly)	3.2	6.5	11.1
Alternative NAAQS (0.2 μg/m³, max monthly)	3.2	6.4	11.1
Alternative NAAQS (0.05 μg/m³, max monthly)	3.1	6.3	11.0
Alternative NAAQS (0.02 μg/m³, max monthly)	3.1	6.3	11.0
Primary Pb smelter - 1.5km subarea			
Current NAAQS (1.5 μg/m³, max quarterly)	5.0	9.5	12.4
Alternative NAAQS (0.5 μg/m³, max monthly)	4.5	8.5	11.8
Alternative NAAQS (0.2 μg/m³, max quarterly)	4.2	7.8	11.5
Alternative NAAQS (0.2 μg/m³, max monthly)	4.1	7.6	11.4
Alternative NAAQS (0.05 μg/m³, max monthly)	3.6	6.8	11.1
Alternative NAAQS (0.02 μg/m³, max monthly)	3.3	6.5	11.1

a - Estimates below 1.0 are rounded to one decimal place, all values below 0.05 are presented as <0.1 and values between 0.05 and 0.1 as 0.1. All values above 1.0 are rounded to the nearest whole number.
b-Log-linear with low-exposure linearization concentration-response function.

Table 4-5. Median IQ loss estimates for the current NAAQS scenario.

Case study	IQ Loss from policy relevant exposures (recent air plus past air) [a]						Total IQ loss (total Pb exposure) [b]		
	Low C-R function estimates		LLL C-R function estimates		High C-R function estimates				
	Recent air	Recent air + past air	Recent air	Recent air + past air	Recent air	Recent air + past air	Low C-R function estimates	LLL C-R function estimates	High C-R function estimates
Location-specific (Chicago)	1.4	2.0	3.4	4.7	5.6	7.4	2.4	5.6	8.8
Location-specific (Cleveland)	0.6	1.4	2.8	3.9	2.1	4.6	1.7	4.7	6.3
Location-specific (Los Angeles)	1.1	1.7	2.7	4.2	4.0	6.2	2.1	5.3	7.7
General urban	1.5	2.1	3.5	4.8	5.6	7.7	2.5	5.8	9.2
Primary Pb smelter-full area		0.6 [c]		1.9		2.3	1.2	3.8	4.4
Primary Pb smelter - subarea		3.2		6.0		9.4	3.7	6.8	11.2

a - These columns present the IQ loss estimated to result from policy-relevant Pb exposure, including recent air and recent plus past air. Estimates for the low C-R function, the LLL C-R function and the high C-R function are presented. The term "past air" includes contributions from the outdoor soil/dust contribution to indoor dust, historical air contribution to indoor dust, and outdoor soil/dust pathways, while "recent air" refers to contributions from inhalation of ambient air Pb or ingestion of indoor dust Pb predicted to be associated with outdoor ambient air Pb levels, with outdoor ambient air also potentially including resuspended, previously deposited Pb (see Section 2.4.3).
b- These columns present the estimates of total IQ loss resulting from total Pb exposure (policy-relevant plus background). Results are presented for the low C-R function, the LLL C-R function and the high C-R function.
c- Risk estimates are not presented for recent air for the primary Pb smelter case study (See Section 3.2.2 of the Risk Assessment Report).

Table 4-6. 95th percentile IQ loss estimates for the current NAAQS scenario

Case study	IQ Loss from policy relevant exposures (recent air plus past air) [a]						Total IQ loss (total Pb exposure) [b]		
	Low C-R function estimates		LLL C-R function estimates		High C-R function estimates				
	Recent air	Recent air + past air	Recent air	Recent air + past air	Recent air	Recent air + past air	Low C-R function estimates	LLL C-R function estimates	High C-R function estimates
Location-specific (Chicago)	3.0	4.0	5.8	7.6	7.7	10.3	4.7	9.0	12.1
Location-specific (Cleveland)	1.4	3.1	2.6	5.9	3.7	8.5	4.3	8.1	11.6
Location-specific (Los Angeles)	2.3	3.6	4.4	6.9	6.1	9.5	4.5	8.6	11.8
General urban	2.9	3.9	5.5	7.6	7.3	10.1	4.7	9.1	12.1
Primary Pb smelter - full area		2.3		4.2		6.8	3.7	6.8	11.2
Primary Pb smelter - subarea		4.2		8.0		10.4	5.0	9.5	12.4

a – These columns present the IQ loss estimated to result from policy-relevant Pb exposure, including recent air and recent plus past air. Estimates for the low C-R function, the LLL C-R function and the high C-R function are presented. The term "past air" includes contributions from the outdoor soil/dust contribution to indoor dust, historical air contribution to indoor dust, and outdoor soil/dust pathways, while "recent air" refers to contributions from inhalation of ambient air Pb or ingestion of indoor dust Pb predicted to be associated with outdoor ambient air Pb levels, with outdoor ambient air also potentially including resuspended, previously deposited Pb (see Section 2.4.3).
b- These columns present the estimates of total IQ loss resulting from total Pb exposure (policy-relevant plus background). Results are presented for the low C-R function, the LLL C-R function and the high C-R function.
c- Risk estimates are not presented for recent air for the primary Pb smelter case study (see Section 3.2.2 of the Risk Assessment Report).

Table 4-7. Incidence of children with >1 point Pb-related IQ loss.

Air Quality Scenario (for location-specific urban case studies)	dual linear - stratified at 7.5 µg/dL peak blood Pb		log-linear with linearization		dual linear - stratified at 10 µ/dL peak blood Pb		log-linear with cutpoint	
	Incidence of >1 point IQ loss	Delta (change in incidence compared to current conditions)	Incidence of >1 point, IQ loss	Delta (change in incidence compared to current conditions)	Incidence of >1 point, IQ loss	Delta (change in incidence compared to current conditions)	Incidence of >1 point, IQ loss	Delta (change in incidence compared to current conditions)
Chicago (total modeled child population: 396,511)								
Chicago Current Conditions (Mean)	391,602		389,754		271,031			
Current NAAQS (1.5 µg/m^3 Maximum Quarterly)	395,797	4,195	395,528	5,773	347,415	76,384	314,053	77,795
Alternative NAAQS (0.2 µg/m^3 Maximum Monthly)	391,158	-444	389,461	-293	271,444	412	235,559	-698
Alternative NAAQS (0.05 µg/m^3 Maximum Monthly)	389,572	-2,030	387,407	-2,347	253,775	-17,256	224,394	-11,864
Alternative NAAQS (0.02 µg/m^3 Maximum Monthly)	389,176	-2,427	386,630	-3,125	249,865	-21,166	219,294	-16,963
Cleveland (total modeled child population: 13,990)								
Cleveland Current Conditions (Mean)	13,809		13,745					
Current NAAQS (1.5 µg/m^3 Maximum Quarterly)	13,893	84	13,857	112	10,664	1,137	9,769	1,254
Alternative NAAQS (0.2 µg/m^3 Maximum Quarterly)	13,770	-38	13,703	-42	9,221	-305	8,160	-354
Alternative NAAQS (0.5 µg/m^3 Maximum Monthly)	13,789	-20	13,720	-25	9,497	-29	8,464	-51
Alternative NAAQS (0.2 µg/m^3 Maximum Monthly)	13,759	-50	13,694	-51	9,083	-443	8,010	-505
Alternative NAAQS (0.05 µg/m^3 Maximum Monthly)	13,729	-80	13,642	-103	8,785	-741	7,720	-795
Alternative NAAQS (0.02 µg/m^3 Maximum Monthly)	13,720	-88	13,628	-117	8,736	-790	7,668	-846
Los Angeles (total modeled child population: 372,252)								
Los Angeles Current Conditions (Mean)	282,216		280,711		191,675			
Current NAAQS (1.5 µg/m^3 Maximum, Quarterly)	285,272	3,056	284,945	4,234	240,988	49,313	226,608	56,134
Alternative NAAQS (0.05 µg/m^3 Maximum Monthly)	281,112	-1,104	279,658	-1,053	183,395	-8,280	161,914	-8,560
Alternative NAAQS (0.02 µg/m^3 Maximum Monthly)	280,740	-1,476	279,057	-1,654	180,745	-10,930	158,234	-12,240

Table 4-8. Incidence of children with >7 points Pb-related IQ loss.

Air Quality Scenario (location-specific urban case studies)	dual linear - stratified at 7.5 ug/dL peak blood Pb		log-linear with linearization		dual linear - stratified at 10 ug/dL peak blood Pb		log-linear with cutpoint	
	Incidence of > 7 points IQ loss	Delta (change in incidence compared to current conditions)	Incidence of > 7 points IQ loss	Delta (change in incidence compared to current conditions)	Incidence of > 7 points IQ loss	Delta (change in incidence compared to current conditions)	Incidence of > 7 points IQ loss	Delta (change in incidence compared to current conditions)
Chicago (total modeled child population: 396,511)								
Chicago Current Conditions (Mean)	136,709		33,664		63		1,015	
Current NAAQS (1.5 µg/m^3 Maximum Quarterly)	244,401	107,692	100,159	66,495	555	492	5,226	4,211
Alternative NAAQS (0.2 µg/m^3 Maximum Monthly)	136,067	-642	32,546	-1,118	48	-16	1,007	-8
Alternative NAAQS (0.05 µg/m^3 Maximum Monthly)	120,706	-16,003	27,367	-6,297	16	-48	864	-151
Alternative NAAQS (0.02 µg/m^3 Maximum Monthly)	117,819	-18,890	26,027	-7,637	8	-56	690	-325
Cleveland (total modeled child population: 13,990)								
Cleveland Current Conditions (Mean)	4,834		1,212		3		46	
Current NAAQS (1.5 µg/m^3 Maximum Quarterly)	6,139	1,305	1,858	647	4	2	105	59
Alternative NAAQS (0.2 µg/m^3 Maximum Quarterly)	4,525	-309	1,073	-139	1	-2	40	-6
Alternative NAAQS (0.5 µg/m^3 Maximum Monthly)	4,806	-28	1,180	-31	1	-2	43	-3
Alternative NAAQS (0.2 µg/m^3 Maximum Monthly)	4,424	-410	1,026	-186	1	-2	43	-3
Alternative NAAQS (0.05 µg/m^3 Maximum Monthly)	4,106	-728	886	-326	0	-3	24	-22
Alternative NAAQS (0.02 µg/m^3 Maximum Monthly)	4,051	-783	866	-345	0	-3	27	-18
Los Angeles (total modeled child population: 372,252)								
Los Angeles Current Conditions (Mean)	94,684		22,665		23		732	
Current NAAQS (1.5 µg/m^3 Maximum, Quarterly)	158,171	63,487	57,834	35,168	183	160	3,771	3,038
Alternative NAAQS (0.05 µg/m^3 Maximum, Monthly)	87,303	-7,382	19,781	-2884	11	-11	624	-109
Alternative NAAQS (0.02 µg/m^3 Maximum, Monthly)	83,909	-10,775	17,939	-4726	17	-6	498	-235

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5 THE PRIMARY LEAD NAAQS

5.1 INTRODUCTION

This chapter presents staff conclusions and recommendations for the Administrator to consider in deciding whether the existing primary Pb standard should be revised and, if so, what revision is appropriate. Our conclusions and recommendations are based on the assessment and integrative synthesis of information presented in the CD, staff analyses and evaluations presented in Chapters 2 through 4 herein, and the comments and advice of CASAC and interested parties who commented on an early draft of this document and the related Risk Assessment Report.

In recommending policy options for the Administrator's consideration, we note that the final decision on retaining or revising the current primary Pb standard is largely a public health policy judgment to be made by the Administrator. The Administrator's final decision should draw upon scientific information and analyses about health effects, population exposure and risks, as well as judgments about the appropriate response to the range of uncertainties that are inherent in the scientific evidence and analyses. Our approach to informing these judgments, discussed more fully below, is based on a recognition that the available health effects evidence generally reflects a continuum consisting of ambient levels at which scientists generally agree that health effects are likely to occur, through lower levels at which the likelihood and magnitude of the response become increasingly uncertain.

This approach is consistent with the requirements of the NAAQS provisions of the Act and with how EPA and the courts have historically interpreted the Act. These provisions require the Administrator to establish primary standards that, in the Administrator's judgment, are requisite to protect public health with an adequate margin of safety. In so doing, the Administrator seeks to establish standards that are neither more nor less stringent than necessary for this purpose. The Act does not require that primary standards be set at a zero-risk level but rather at a level that avoids unacceptable risks to public health, including the health of sensitive groups.

The following discussion starts with background information on the current standard in Section 5.2, including both the basis for derivation of the current standard and considerations and conclusions from the Staff Paper prepared in the last review. The general approach used in this current review to evaluate the adequacy of the current standard and identify policy alternatives is summarized in Section 5.3. Staff conclusions and recommendations with regard to the adequacy of the current standard are discussed in Section 5.4, and conclusions and recommendations with regard to elements of alternative standards for consideration are discussed in Section 5.5. Key uncertainties and research recommendations related to setting a primary lead standard are identified in Section 5.6.

5.2 BACKGROUND ON THE CURRENT STANDARD

5.2.1 Basis for Setting the Current Standard

The current primary standard is set at a level of 1.5 μg/m^3, measured as lead in TSP, not to be exceeded by the maximum arithmetic mean concentration averaged over a calendar quarter. The standard was set in 1978 to provide protection to the public, especially children as the particularly sensitive population subgroup, against Pb-induced adverse health effects (43 FR 46246). The basis for selecting each of the elements of the standard is described below.

5.2.1.1 Level

EPA's objective in selecting the level of the current standard was "to estimate the concentration of lead in the air to which all groups within the general population can be exposed for protracted periods without an unacceptable risk to health" (43 FR 46252). Consistent with section 109 of the Clean Air Act, the Agency selected a level for the current standard that was below the concentration that was at that time identified as a threshold for adverse health effects (i.e., 40 μg/dl blood Pb), so as to provide an adequate margin of safety. As stated in the notice of final rulemaking,

"This estimate was based on EPA's judgment in four key areas:

(1) Determining the 'sensitive population' as that group within the general population which has the lowest threshold for adverse effects or greatest potential for exposure. EPA concludes that young children, aged 1 to 5, are the sensitive population.

(2) Determining the safe level of total lead exposure for the sensitive population, indicated by the concentration of lead in the blood. EPA concludes that the maximum safe level of blood lead for an individual child is 30 μg Pb/dl and that population blood lead, measured as the geometric mean, must be 15 μg Pb/dl in order to place 99.5 percent of children in the United States below 30 μg Pb/dl.

(3) Attributing the contribution to blood lead from nonair pollution sources. EPA concludes that 12 μg Pb/dl of population blood lead for children should be attributed to nonair exposure.

(4) Determining the air lead level which is consistent with maintaining the mean population blood lead level at 15 μg Pb/dl [the maximum safe level]. Taking into account exposure from other sources (12 μg Pb/dl), EPA has designed the standard to limit air contribution after achieving the standard to 3 μg Pb/dl. On the basis of an estimated relationship of air lead to blood lead of 1 to 2, EPA concludes that the ambient air standard should be 1.5 μg Pb/m^3." (43 FR 46252)

EPA's judgments in these key areas, as well as margin of safety considerations, are discussed in the following subsections.

5.2.1.1.1 Sensitive Population

The assessment of the science that was presented in the 1977 CD (USEPA, 1977), indicated young children, aged 1 to 5, as the population group at particular risk from Pb exposure. Children were recognized to have a greater physiological sensitivity than adults to the effects of Pb and a greater exposure. In identifying young children as the sensitive population, EPA also recognized the occurrence of subgroups with enhanced risk due to genetic factors, dietary deficiencies or residence in urban areas. Yet information was not available to estimate a threshold for adverse effects for these subgroups separate from that of all young children. Additionally, EPA recognized both a concern regarding potential risk to pregnant women and fetuses, and a lack of information to establish that these subgroups are more at risk than young children. Accordingly, young children, aged 1 to 5, were identified as the group which has the lowest threshold for adverse effects of greatest potential for exposure (i.e., the sensitive population) (43 FR 46252).

5.2.1.1.2 Maximum Safe Blood Level

In identifying the maximum safe exposure, EPA relied upon the measurement of Pb in blood (43 FR 46252-46253). The physiological effect of Pb that had been identified as occurring at the lowest blood Pb level was inhibition of an enzyme integral to the pathway by which heme (the oxygen carrying protein of human blood) is synthesized, i.e., delta-aminolevulinic acid dehydratase (δ-ALAD). The 1977 CD reported a threshold for inhibition of this enzyme in children at 10 μg Pb/dL. The 1977 CD also reported a threshold of 15-20 μg/dL for elevation of protoporphyrin (EP), which is an indication of some disruption of the heme synthesis pathway. EPA concluded that this effect on the heme synthesis pathway (indicated by EP) was potentially adverse. EPA further described a range of blood levels associated with a progression in detrimental impact on the heme synthesis pathway. At the low end of the range (15-20 μg/dL), the initial detection of EP associated with blood Pb was not concluded to be associated with a significant risk to health. The upper end of the range (40 μg/dL), the threshold associated with clear evidence of heme synthesis impairment and other effects contributing to clinical symptoms of anemia, was regarded as clearly adverse to health. EPA also recognized the existence of thresholds for additional adverse effects (e.g., nervous system deficits) occurring for some children at just slightly higher blood Pb levels (e.g., 50 μg/dL). Additionally, EPA stated that the maximum safe blood level should not be higher than the blood Pb level recognized by the CDC as "elevated" (and indicative of the need for intervention). In 1978, that level was 30 μg/dL[1].

[1] The CDC subsequently revised their advisory level for children's blood Pb to 25 μg/dL in 1985, and to 10 μg/dL 1991. More details on this level are provided in Section 3.2.

Having identified the maximum safe blood level in individual children, EPA next made a public health policy judgment regarding the target mean blood level for the U.S. population of young children (43 FR 46252-46253). With this judgment, EPA identified a target of 99.5 percent of this population to be brought below the maximum safe blood Pb level. This judgment was based on consideration of the size of the sensitive subpopulation, and the recognition that there are special high-risk groups of children within the general population. The population statistics available at the time (the 1970 U.S. Census) indicated a total of 20 million children younger than 5 years of age, with 15 million residing in urban areas and 5 million in center cities where Pb exposure was thought likely to be “high”. Concern about these high-risk groups influenced EPA’s determination of 99.5 percent, deterring EPA from selecting a population percentage lower than 99.5 (43 FR 46253). EPA then used standard statistical techniques to calculate the population mean blood Pb level that would place 99.5 percent of the population below the maximum safe level. Based on the then available data, EPA concluded that blood Pb levels in the population of U.S. children were normally distributed with a geometric standard deviation of 1.3. Based on standard statistical techniques, EPA determined that a thus described population in which 99.5 percent of the population has blood Pb levels below 30 μg/dL would have a geometric mean blood level of 15 μg/dL. EPA described 15 μg/dL as "the maximum safe blood lead level (geometric mean) for a population of young children" (43 FR 46247).

5.2.1.1.3 Nonair Contribution

When setting the current NAAQS, EPA recognized that the air standard needed to take into account the contribution to blood Pb levels from Pb sources unrelated to air pollution. Consequently, the calculation of the current NAAQS included the subtraction of Pb contributed to blood Pb from nonair sources from the estimate of a safe mean population blood Pb level. Without this subtraction, EPA recognized that the combined exposure to Pb from air and nonair sources would result in a blood Pb concentration exceeding the safe level (43 FR 46253).

In developing an estimate of this nonair contribution, EPA recognized the lack of detailed or widespread information about the relative contribution of various sources to children’s blood Pb levels, such that an estimate could only be made by inference from other empirical or theoretical studies, often involving adults. Additionally, EPA recognized the expectation that the contribution to blood Pb levels from nonair sources would vary widely, was probably not in constant proportion to air Pb contribution, and in some cases may alone exceed the target mean population blood Pb level (43 FR 46253-46254).

The amount of blood Pb attributed to nonair sources was selected based primarily on findings in studies of blood Pb levels in areas where air Pb levels were low relative to other locations in U.S. The air Pb levels in these areas ranged from 0.1 to 0.7 $\mu g/m^3$. The average of

the reported blood Pb levels for children of various ages in these areas was on the order of 12 μg/dL. Thus, 12 μg/dL was identified as the nonair contribution, and subtracted from the population mean target level of 15 μg/dL to yield a value of 3 μg/dL as the limit on the air contribution to blood Pb.

5.2.1.1.4 Air Pb Level

In determining the air Pb level consistent with an air contribution of 3 μg Pb/dL, EPA reviewed studies assessed in the 1977 CD that reported changes in blood Pb with different air Pb levels. These studies included a study of children exposed to Pb from a primary Pb smelter, controlled exposures of adult men to Pb in fine particulate matter, and a personal exposure study involving several male cohorts exposed to Pb in a large urban area in the early 1970s (43 FR 46254). Using all three studies, EPA calculated an average slope or ratio over the entire range of data. That value was 1.95 (rounded to 2 μg/dL blood Pb concentration to 1 $\mu g/m^3$ air Pb concentration), and is recognized to fall within the range of values reported in the 1977 CD. On the basis of this 2-to-1 relationship, EPA concluded that the ambient air standard should be 1.5 $\mu g\ Pb/m^3$ (43 FR 46254).

5.2.1.1.5 Margin of Safety

In consideration of the appropriate margin of safety during the development of the current NAAQS, EPA identified the following factors: (1) the 1977 CD reported multiple biological effects of Pb in practically all cell types, tissues and organ systems, of which the significance for health had not yet been fully studied; (2) no beneficial effects of Pb at then current environmental levels were recognized; (3) data were incomplete as to the extent to which children are indirectly exposed to air Pb that has moved to other environmental media, such as water, soil and dirt, and food; (4) Pb is chemically persistent and with continued uncontrolled emissions would continue to accumulate in human tissue and the environment; and (5) the possibility that exposure associated with blood Pb levels previously considered safe might influence neurological development and learning abilities of the young child (43 FR 46255). Recognizing that estimating an appropriate margin of safety for the air Pb standard was complicated by the multiple sources and media involved in Pb exposure, EPA chose to use margin of safety considerations principally in establishing a maximum safe blood Pb level for individual children (30 μg Pb/dL) and in determining the percentage of children to be placed below this maximum level (about 99.5 percent). Additionally, in establishing other factors used in calculating the standard, EPA used margin of safety considerations in the sense of making careful judgment based on available data, but these judgments were not considered to be at the precautionary extreme of the range of data available at the time (43 FR 46251).

EPA further recognized that, because of the variability between individuals in a population experiencing a given level of Pb exposure, it was considered impossible to provide the same margin of safety for all members in the sensitive population or to define the margin of safety in the standard as a simple percentage. EPA believed that the factors it used in designing the standards provided an adequate margin of safety for a large proportion of the sensitive population. The Agency did not believe that the margin was excessively large or on the other hand that the air standard could protect everyone from elevated blood Pb levels (43 FR 46251).

5.2.1.2 Averaging Time, Form, and Indicator

The averaging time for the current standard is a calendar quarter. In the decision for this aspect of the standard, the Agency also considered a monthly averaging period, but concluded that "a requirement for the averaging of air quality data over calendar quarter will improve the validity of air quality data gathered without a significant reduction in the protectiveness of the standards." As described in the notice for this decision (43 FR 46250), this conclusion was based on several points, including the following:

- An analysis of ambient measurements available at the time indicated that the distribution of air Pb levels was such that there was little possibility that there could be sustained periods greatly above the average value in situations where the quarterly standard was achieved.
- A recognition that the monitoring network may not actually represent the exposure situation for young children, such that it seemed likely that elevated air Pb levels when occurring would be close to Pb air pollution sources where young children would typically not encounter them for the full 24-hour period reported by the monitor.
- Medical evidence available at the time indicated that blood Pb levels re-equilibrate slowly to changes in air exposure, a finding that would serve to dampen the impact of short-term period of exposure to elevated air Pb.
- Direct exposure to air is only one of several routes of total exposure, thus lessening the impact of a change in air Pb on blood Pb levels.

The statistical form of the current standard is as a not-to-be-exceeded or maximum value. EPA set the standard as a ceiling value with the conclusion that this air level would be safe for indefinite exposure for young children (43 FR 46250).

The indicator is total airborne Pb collected by a high volume sampler (43 FR 46258). EPA's selection of Pb-TSP as the indicator for the standard was based on explicit recognition both of the significance of ingestion as an exposure pathway for Pb that had deposited from the air and of the potential for Pb deposited from the air to become re-suspended in respirable size particles in the air and available for human inhalation exposure. As stated in the final rule, "a significant component of exposure can be ingestion of materials contaminated by deposition of

lead from the air," and that, "in addition to the indirect route of ingestion and absorption from the gastrointestinal tract, non-respirable Pb in the environment may, at some point become respirable through weathering or mechanical action" (43 FR 46251).

5.2.2 Policy Options Considered in the Last Review

During the 1980s, EPA initiated a review of the air quality criteria and NAAQS for Pb. CASAC and the public were fully involved in this review, which led to the publication of a criteria document with associated addendum and a supplement (USEPA, 1986a, 1986b, 1990a), an exposure analysis methods document (USEPA, 1989) and a staff paper (USEPA, 1990b).

Total emissions to air were estimated to have dropped by 94 percent between 1978 and 1987, with the vast majority of it attributed to the reduction of Pb in gasoline. Accordingly, the focus of the last review was on areas near stationary sources of Pb emissions. Although such sources were not considered to have made a significant contribution (as compared to Pb in gasoline) to the overall Pb pollution across large, urban or regional areas, Pb emissions from such sources were considered to have the potential for a significant impact on a local scale. Air Pb concentrations, and especially soil and dust Pb concentrations had been associated with elevated levels of Pb absorption in children and adults in numerous Pb point source community studies. Exceedances of the current NAAQS were found at that time only in the vicinity of nonferrous smelters or other point sources of Pb.

In summarizing and interpreting the health evidence presented in the 1986 CD and associated documents, the 1990 Staff Paper described the collective impact on children of the effects at blood Pb levels above 15 μg/dL as representing a clear pattern of adverse effects worthy of avoiding. This is in contrast to EPA's identification of 30 μg/dL as a safe blood Pb level for individual children when the NAAQS was set in 1978. The Staff Paper further stated that at blood Pb levels of 10-15 μg/dL there was a convergence of evidence of Pb-induced interference with a diverse set of physiological functions and processes, particularly evident in several independent studies showing impaired neurobehavioral function and development. Further, the available data did not indicate a clear threshold in this blood Pb range. Rather, it suggested a continuum of health risks down to the lowest levels measured.[2]

For the purposes of comparing the relative protectiveness of alternative Pb NAAQS, the staff conducted analyses to estimate the percentages of children with blood Pb levels above 10 μg/dL and above 15 μg/dL for several air quality scenarios developed for a small set of stationary source exposure case studies. These analyses omitted the subset of young children, whom it was considered could not be substantially affected by any changes in atmospheric Pb

[2] In 1991, the CDC reduced their advisory level for children's blood Pb from 25 μg/dL to 10 μg/dL.

emissions under different standards, such as those with excessive pica[3] and/or those living in overtly deteriorated Pb-paint homes. The results of the analyses of child populations living near two Pb smelters indicated that substantial reductions in Pb exposure could be achieved through just meeting the current Pb NAAQS. According to the best estimate analyses, over 99.5% of children living in areas significantly affected by the smelters would have blood Pb levels below 15 μg/dL if the current standard was achieved. Progressive changes in this number were estimated for the alternative monthly Pb NAAQS levels evaluated in those analyses, which ranged from 1.5 $\mu g/m^3$ to 0.5 $\mu g/m^3$.

In light of the health effects evidence available at the time, the Staff Paper presented air quality, exposure, and risk analyses, and other policy considerations, as well as the following staff conclusions with regard to the primary Pb NAAQS (USEPA, 1990b, pp. xii to xiv):

1) "The range of standards … should be from 0.5 to 1.5 $\mu g/m^3$."
2) "A monthly averaging period would better capture short-term increases in lead exposure and would more fully protect children's health than the current quarterly average."
3) "The most appropriate form of the standard appears to be the second highest monthly averages *{sic}* in a 3-year span. This form would be nearly as stringent as a form that does not permit any exceedances and allows for discounting of one "bad" month in 3 years which may be caused, for example, by unusual meteorology."
4) "With a revision to a monthly averaging time more frequent sampling is needed, except in areas, like roadways remote from lead point sources, where the standard is not expected to be violated. In those situations, the current 1-in-6 day sampling schedule would sufficiently reflect air quality and trends."
5) "Because exposure to atmospheric lead particles occurs not only via direct inhalation, but via ingestion of deposited particles as well, especially among young children, the hi-volume sampler provides a reasonable indicator for determining compliance with a monthly standard and should be retained as the instrument to monitor compliance with the lead NAAQS until more refined instruments can be developed."

Based on its review of a draft Staff Paper, which contained the above recommendations, the CASAC strongly recommended to the Administrator that EPA should actively pursue a public health goal of minimizing the lead content of blood to the extent possible, and that the Pb NAAQS is an important component of a multimedia strategy for achieving that goal (CASAC, 1990, p. 4). In noting the range of levels recommended by staff, CASAC recommended consideration of a revised standard that incorporates a "wide margin of safety, because of the risk

[3] Pica is an eating disorder typically defined by persistent cravings to eat non-food items.

posed by Pb exposures, particularly to the very young whose developing nervous system may be compromised by even low level exposures" (id., p. 3). More specifically, CASAC judged that a standard within the range of 1.0 to 1.5 $\mu g/m^3$ would have "relatively little, if any, margin of safety;" that greater consideration should be given to a standard set below 1.0 $\mu g/m^3$; and, to provide perspective in setting the standard, it would be appropriate to consider the distribution of blood Pb levels associated with meeting a monthly standard of 0.25 $\mu g/m^3$, a level below the range considered by staff (id.).

After consideration of the documents developed during the review, EPA chose not to propose revision of the NAAQS for Pb. During the same time period, the Agency published and embarked on the implementation of a broad, multi-program, multi-media, integrated national strategy to reduce Pb exposures (USEPA, 1991). As part of implementing this integrated Pb strategy, the Agency focused efforts primarily on regulatory and remedial clean-up actions aimed at reducing Pb exposures from a variety of non-air sources judged to pose more extensive public health risks to U.S. populations, as well as on actions to reduce Pb emissions to air, particularly near stationary sources. This focus reflected in part the dramatic reduction of Pb in gasoline that occurred since the standard was set in 1978, which resulted in orders-of-magnitude reductions in airborne emissions of Pb, and a significant shift in the types of sources with the greatest Pb emissions. EPA established standards for Pb-based paint hazards and Pb dust cleanup levels in most pre-1978 housing and child-occupied facilities. Additionally, EPA has developed standards for the management of Pb in solid and hazardous waste, oversees the cleanup of Pb contamination at Superfund sites, and has issued regulations to reduce Pb in drinking water (http://www.epa.gov/lead/regulation.htm). Beyond these specific regulatory actions, the Agency's Lead Awareness Program has continued to work to protect human health and the environment against the dangers of Pb by conducting research and designing educational outreach activities and materials (http://www.epa.gov/lead/). Actions to reduce Pb emissions to air during the 1990s included enforcement of the NAAQS, as well as the promulgation of regulations under Section 112 of the Clean Air Act, including national emissions standards for hazardous air pollutants (including Pb compounds) at primary and secondary Pb smelters, as well as other Pb sources.

5.3 APPROACH FOR CURRENT REVIEW

To evaluate whether it is appropriate to consider retaining the current primary Pb standard, or whether consideration of revisions is appropriate, we adopted an approach in this review that builds upon the general approach used in the initial setting of the standard, as well as that used in the last review, and reflects the broader body of evidence and information now available. As summarized above, the 1978 rulemaking decisions were based on an integration of information on health effects associated with exposure to ambient Pb**;** expert judgment on the adversity of such effects on individuals; and policy judgments as to when the standard is requisite to protect public health with an adequate margin of safety, which were informed by air quality and related analyses, quantitative exposure and risk assessments when possible, and qualitative assessment of impacts that could not be quantified. The decision in the last review not to propose any revision to the Pb NAAQS was made in conjunction with the Agency's implementation of a broad integrated Pb strategy to reduce Pb exposures through various regulatory and awareness programs that focused on important non-air sources judged to pose more extensive public health risks, reflecting in part the dramatic reduction of Pb in gasoline since the standard was set.

In conducting this assessment, staff is again aware of the dramatic alteration in the basic patterns of air lead emissions in the U.S. since the standard was set that was evident during the last review as well. In addition to the dramatic reduction of Pb in gasoline, an additional circumstance that has changed since the standard was set is the enactment of Clean Air Act Amendments of 1990, which amended the Clean Air Act Section 112 to list Pb compounds as hazardous air pollutants (HAP) and to require technology-based and risk-based standards, as appropriate, for major stationary sources of HAP. Staff is also aware that these significantly changed circumstances have raised the question in this review of whether it is still appropriate to maintain a NAAQS for Pb or to retain Pb on the list of criteria pollutants. As a result, this assessment considers the status of Pb as a criteria pollutant and assesses whether revocation of the standard is an appropriate option for the Administrator to consider.

In developing conclusions and identifying policy options for the Pb standard in this review, staff has taken into account both evidence-based and quantitative exposure- and risk-based considerations. A series of general questions frame our approach to reaching conclusions and identifying options for consideration by the Administrator in deciding whether to retain or revise the current primary Pb standard. Our review of the adequacy of the current standard (section 5.4) addresses questions such as the following:

- To what extent does newly available information reinforce or call into question evidence of associations with effects identified in the last review?

- To what extent has evidence of new effects and/or sensitive populations become available since the last review?
- To what extent have important uncertainties identified in the last review been reduced and have new uncertainties emerged?
- To what extent does newly available information reinforce or call into question any of the basic elements of the current standard?
- To what extent does available information and current circumstances regarding basic patterns of air lead emissions in the U.S. reinforce or call into question the need to maintain a standard for Pb or to retain Pb on the list of criteria pollutants?

To the extent that the available information suggests that revision of the current standard may be appropriate to consider, we also address whether the currently available information supports consideration of a standard that is either more or less protective by addressing questions such as the following:

- Is there evidence that associations, especially likely causal associations, extend to air quality levels that are as low as or lower than had previously been observed, and what are the important uncertainties associated with that evidence?
- Are exposures of concern and health risks estimated to occur in areas that meet the current standard; are they important from a public health perspective; and what are the important uncertainties associated with the estimated risks?

To the extent that there is support for consideration of a revised standard, we then consider the specific elements of the standard (section 5.5) to identify ranges of standards (in terms of an indicator, averaging time, level, and form) that we conclude would be appropriate for the Administrator to consider in making public health policy judgments, based on the currently available information, as to the degree of protection that is requisite to protect public health with an adequate margin of safety. In so doing, we address the following questions:

- Does the evidence provide support for considering a different Pb indicator?
- Does the evidence provide support for considering different averaging times?
- What ranges of levels and forms of alternative standards are supported by the evidence, and what are the uncertainties and limitations in that evidence?
- To what extent do specific levels and forms of alternative standards reduce the estimated exposures of concern and risks attributable to Pb, and what are the uncertainties associated with the estimated exposure and risk reductions?

5.4 ADEQUACY OF THE CURRENT STANDARD

In considering the adequacy of the current standard, staff first considered whether it is appropriate to maintain a NAAQS for Pb or to retain Pb on the list of criteria pollutants. As noted above, this question has arisen in this review as a result of the dramatic alteration in the basic patterns of air Pb emissions in the U.S. since the standard was set, that primarily reflects the dramatic reduction of Pb in gasoline, which resulted in orders-of-magnitude reductions in airborne emissions of Pb and a significant shift in the types of sources with the greatest Pb emissions. In addition, Section 112 of the Clean Air Act was amended in 1990 to include Pb compounds on the list of hazardous air pollutants and to require EPA to establish technology-based emission standards for those listed major source categories emitting Pb compounds, and to establish risk-based standards, as appropriate, for those categories of sources.

In considering this issue, staff notes that CASAC specifically examined several scientific issues and related public health (and public welfare) issues that the CASAC Lead Review Panel[4] judged to be essential in determining whether delisting Pb or revoking the Pb NAAQS would be appropriate options for the Administrator to consider. In its letter to the Administrator of March 27, 2007, based on its review of the first draft Staff Paper (Henderson, 2007a; Attachment A), CASAC's examination of these issues was framed by the following series of questions:

(1) *Does new scientific information accumulated since EPA's promulgation of the current primary Lead NAAQS of 1.5 µg/m^3 in 1978 suggest that science previously overstated the toxicity of lead?*

(2) *Have past regulatory and other controls on lead decreased PbB [blood lead] concentrations in human populations so far below levels of concern as to suggest there is now an adequate margin of safety inherent in those PbB levels?*

(3) *Have the activities that produced emissions and atmospheric redistribution of lead in the past changed to such an extent that society can have confidence that emissions will remain low even in the absence of NAAQS controls?*

(4) *Are airborne concentrations and amounts of lead sufficiently low throughout the United States that future regulation of lead exposures can be effectively accomplished by regulation of lead-based products and allowable amounts of lead in soil and/or water?*

(5) *If lead were de-listed as a criteria air pollutant, would it be appropriately regulated under the Agency's Hazardous Air Pollutants (HAP) program?*

For the reasons presented in its March 2007 letter (Attachment A), the CASAC Lead Review Panel judged that the answer to each of these questions was "*no*," leading the Panel to

[4] This Lead Panel includes the statutorily defined seven-member CASAC and additional subject-matter experts needed to provide an appropriate breadth of expertise for this review of the Pb NAAQS.

conclude that "the existing state of science is consistent with continuing to list ambient lead as a criteria pollutant for which fully-protective NAAQS are required" (id, p. 5). Further, in a subsequent letter to the Administrator of September 27, 2007, based on its review of the second draft Risk Assessment Report (Henderson, 2007b; Attachment B), CASAC strongly reiterated its opposition to any considered delisting of Pb, and expressed its unanimous support for maintaining fully-protective NAAQS (id., p. 2).

Staff also notes the receipt of comments from the public on this issue. In a comment submitted on the first draft Staff Paper, an industry group urged the Agency to delist Pb as a criteria pollutant. Many other public comments on this issue, received as comments on the first draft Staff Paper, were against revoking the Pb NAAQS or delisting Pb as a criteria pollutant.

Staff concurs with the reasoning that the CASAC Lead Review Panel used in reaching its judgments on these questions and agrees with the conclusion that currently available information does not support either delisting Pb as a criteria pollutant or revoking, and not replacing, the current Pb NAAQS. In particular, we note that notwithstanding the dramatic changes in the basic patterns of air Pb emissions in the U.S. since the standard was set, Pb continues to be emitted into the ambient air from numerous and diverse mobile and stationary sources. Further, currently available studies provide evidence of adverse health effects associated with blood lead levels and environmental exposures well below those previously identified, and we note that there is now no discernable threshold for such effects in contrast to the thresholds that had previously been inferred. While there is substantial evidence that segments of the population continue to have blood lead levels that are clearly of concern, there is only limited evidence on which to base an assessment of the extent to which airborne Pb contributes to these blood Pb levels. Nonetheless, we believe that the available information is sufficient to infer that ambient air Pb contributes to air pollution that may reasonably be anticipated to negatively impact public health and that further reductions in ambient air Pb would likely benefit public health. In the absence of evidence to the contrary, we believe it is appropriate to retain the NAAQS authority as part of a broad strategy to control Pb exposures in sensitive populations. Further, we note that there is the potential for lead emissions to increase above present levels in the absence of a Pb NAAQS, such as through increased capacity at Pb processing facilities or through the possible conversion of secondary Pb smelting facilities to primary smelting operations. In addition, while we recognize that airborne Pb emissions can be reduced for some source categories through regulatory actions under the hazardous air pollutant program, that program is focused on stationary sources and is not directed toward other types of sources, including mobile sources and related resuspension, that contribute to Pb in the ambient air. For the reasons identified here, we recommend that consideration not be given to delisting Pb as a criteria pollutant or to revoking, and not replacing, the Pb NAAQS.

Having reached the general conclusion that it is appropriate to maintain a NAAQS for Pb, we discuss below the available evidence (section 5.4.1) and quantitative exposure- and risk-based considerations (section 5.4.2) to more fully inform consideration of the adequacy of the current standard. We also take into account the views expressed by CASAC and public commenters (section 5.4.3) in reaching staff conclusions on the adequacy of the current standard (section 5.4.4).

5.4.1 Evidence-based Considerations

In considering the broad array of health effects evidence assessed in the CD with respect to the adequacy of the current standard, staff has focused on those health endpoints associated with the Pb exposure and blood levels most pertinent to ambient exposures (Chapter 3). Additionally, we give particular weight to evidence available today that differs from that available at the time the standard was set with regard to its support of the current standard.

First, with regard to the sensitive population, the susceptibility of young children to the effects of Pb is well recognized, in addition to more recent recognition of effects of chronic exposure to low level Pb with advancing age (CD, Sections 5.3.7 and pp. 8-73 to 8-75). As summarized in Chapter 3 and discussed in detail in the CD, the prenatal period and early childhood are periods of increased susceptibility to Pb exposures, with robust evidence of adverse effects on the developing nervous system that generally appear to persist into later childhood and adolescence (CD, Section 6.2). Thus, while we also recognize the sensitivity of the elderly and other particular subgroups (e.g., see Section 3.4.1), as at the time the standard was set, young children continue to be recognized as the key sensitive population for Pb exposures.

With regard to the exposure levels at which adverse health effects occur, the current evidence demonstrates the occurrence of adverse health effects at appreciably lower blood Pb levels than those demonstrated by the evidence at the time the standard was set. At the time the standard was set the physiological effects identified as occurring at the lowest blood Pb levels were those associated with production of anemia. EPA recognized as clearly adverse the impairment of heme synthesis and other Pb-related effects which were identified to result in clinical symptoms of anemia in children above a blood Pb level of 40 µg/dl, thus identifying this blood Pb level as an approximate threshold for adverse health effects of Pb (USEPA, 1977; 43 FR 46252).[5]

[5] At the time the standard was set, inhibition of delta-aminolevulinic acid dehydratase (δ-ALAD), an enzyme integral to hemoglobin synthesis, was demonstrated to occur at a blood Pb level as low as 10 µg/dL. Effects of lead on cellular synthesis of heme, as indicated by elevation of EP was considered potentially adverse to the health of young children and EP elevation could be correlated with blood Pb levels as low as 15 to 20 µg/dL.

This change in the evidence since the time the standard was set is reflected in changes made by the CDC in their advisory level for Pb in children's blood, and changes they have made in their characterization of that level (described more fully in Section 3.2). In 1978, when the current Pb NAAQS was established, the CDC recognized a level of 30 μg/dL blood Pb as warranting individual intervention (CDC, 1991). In 2005, with consideration of a review of the evidence by their advisory committee, CDC revised their statement on *Preventing Lead Poisoning in Young Children*, specifically recognizing the evidence of adverse health effects in children with blood Pb levels below 10 μg/dL and the data demonstrating that no "safe" threshold for blood Pb had been identified (CDC, 2005).

As discussed extensively in the CD and summarized in Chapter 3, the current evidence demonstrates the occurrence of a variety of adverse health effects, including those on the developing nervous system, associated with blood Pb levels extending well below 10 μg/dL to 5 μg/dL and possibly lower. For example, we note in particular the international pooled analysis by Lanphear and others (2005), studies of individual cohorts such as the Rochester, Boston, and Mexico City cohorts (Canfield et al., 2003a; Canfield et al., 2003b; Bellinger and Needleman, 2003; Tellez-Rojo et al., 2006), the study of African-American inner-city children from Detroit (Chiodo et al., 2004), and the cross-sectional analysis of a nationally representative sample from the NHANES, in which the mean blood Pb level was 1.9 μg/dL (Lanphear et al., 2000).

Further, current evidence does not indicate a threshold for the more sensitive health endpoints such as adverse effects on the developing nervous system (CD, pp. 5-71 to 5-74 and Section 6.2.13). This differs from the Agency's inference of a threshold of 40 μg/dL blood Pb for the most sensitive health endpoint identified in the 1978 rulemaking, i.e., impairment of heme synthesis and other effects which result in childhood anemia.

As when the standard was set in 1978, we recognize that there remain today contributions to blood Pb levels from nonair sources. Estimating contributions from nonair sources is complicated by the persistent nature of Pb. For example, Pb that is a soil or dust contaminant today may have been airborne yesterday or many years ago. The studies currently available and reviewed in the CD that evaluate the multiple pathways of Pb exposure do not usually distinguish between outdoor soil/dust Pb resulting from historical emissions and outdoor soil/dust Pb resulting from recent emissions. Further, while indoor dust Pb has been identified as being a predominant contributor to children's blood Pb, available studies do not distinguish the

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However, because of an absence of evidence of impairment on heme synthesis at levels below 40 μg/dL, EPA did not recognize the inhibition of δ-ALAD at lower blood Pb levels as adverse to health. At that time, EPA stated that it considered that above blood levels of 30 μg/dL, EP elevation has progressed to the extent that it should be considered an adverse health effect, and that the effects on heme synthesis seen at 40 μg/dL and above are clearly adverse (43 FR 46251-46253).

different pathways (air-related and other) contributing to indoor dust Pb. The exposure assessment performed for this review has employed available data and methods to develop estimates intended to inform a characterization of these pathways (see Chapter 4).

Consistent with reductions in air Pb concentrations[6] which contribute to blood Pb, nonair contributions have also been reduced. For example, Pb contributions to diet have been reported to have declined significantly since 1978, perhaps as much as 70% or more between then and 1990 (WHO, 1995) and the 2006 CD identifies a drop in dietary Pb intake by 2 to 5 year olds of 96% between the early 1980s and mid 1990s. The 1977 CD included a dietary Pb intake estimate for the general population of 100 to 350 µg Pb/day (USEPA 1977, p. 1-2) and the 2006 CD cites recent studies indicating a dietary intake ranging from 2 to 10 µg Pb/day (CD, Section 3.4 and p. 8-14). Reductions in elevated blood Pb levels in urban areas indicate that other nonair contributions to blood Pb (e.g., drinking water distribution systems, and Pb-based paint) have also been reduced since the late 1970s. In their March 2007 letter to the Administrator, the CASAC Pb Panel recommended that 1.0-1.4 µg/dL or lower be considered as an estimate of the nonair component of blood Pb.

As in 1978, the evidence demonstrates that Pb in ambient air contributes to Pb in blood, with the pertinent exposure routes including both inhalation and ingestion (CD, Sections 3.1.3.2, 4.2 and 4.4; Hilts et al., 2003). In 1978, the evidence indicated a quantitative relationship between ambient air Pb and blood Pb – i.e., the ratio describing the increase in blood Pb per unit of air Pb – that ranged from 1:1 to 1:2 (USEPA, 1977). In setting the standard, the Agency relied on a ratio of 1:2, i.e., 2 µg/dL blood Pb per 1 µg/m^3 air Pb (43 FR 46252). The evidence now and in the past on this relationship is limited by the circumstances in which the data are collected. We do not have specific measurements of Pb in blood that derived from Pb that had been in the air. Rather, we have estimates of the relationship between Pb concentrations in air and Pb levels in blood, developed from populations in differing Pb exposure circumstances, which inform us on this point. Many of the currently available reviews of estimates for air-to-blood ratios, which include air contributions from both inhalation and ingestion exposure pathways, indicate that such ratios generally fall between 1:3 to 1:5, with some as high as 1:10 or higher (USEPA 1986a, pp. 11-99 to 11-100 and 11-106; Brunekreef, 1984). Findings of a recent study of changes in children's blood Pb levels associated with reduced Pb emissions and associated air concentrations near a Pb smelter in Canada indicates a ratio on the order of 1:7 (CD, pp. 3-23 to 3-24; Hilts et al., 2003). In their advice to the Agency, CASAC identified values of 1:5 as used by the World Health Organization (2000) and 1:10 as supported by an

[6] As described in Section 2.3.2.2, air Pb concentrations nationally are estimated to have declined more than 90% since the early 1980s.

empirical analysis of changes in air Pb and changes in blood Pb between 1976 and the time when the phase-out of Pb from gasoline was completed (Henderson, 2007a). While there is uncertainty in the absolute value of the air-to-blood relationship, the current evidence indicates a notably greater ratio, with regard to increase in blood Pb, than the 1978 1:2 relationship e.g., on the order of 1:3 to 1:10.

Taken together, the dramatic reduction in the blood Pb level associated with adverse health effects, the evidence for a greater ratio between blood Pb and air Pb, and the lack of evidence for a threshold for the sensitive health endpoint, we conclude that the current evidence calls into question the adequacy of the current standard. In particular, there is now no recognized safe level of Pb in children's blood and studies appear to show adverse effects at mean concurrent blood Pb levels as low as 2 µg/dL (CD, pp. 6-31 to 6-32; Lanphear et al., 2000), as compared to EPA's view in 1978 that 30 µg/dL was a maximum safe blood Pb level for an individual child, and that 15 µg/dL was the maximum safe blood Pb level for a population of young children (43 FR 46246-46256). Further, while the nonair contribution to blood Pb has declined, perhaps to a range of 1.0-1.4 µg/dL, the air-to-blood ratio appears to be higher at today's lower blood Pb levels than the estimates at the time the standard was set, with current estimates on the order of 1:3 to 1:5 and perhaps up to 1:10. Therefore, considered in light of the framework employed in setting the standard in 1978, the more recently available evidence suggests a level for the standard that is lower by an order of magnitude or more.

5.4.2 Exposure- and Risk-based Considerations

In addition to the evidence-based considerations, staff has also considered exposures and health risks estimated to occur upon just meeting the current Pb standard to help inform judgments about the extent to which exposure and risk estimates may be judged to be important from a public health perspective, taking into account key uncertainties associated with the estimated exposures and risks.

As discussed in the previous section (Section 5.4.1), young children are the sensitive population of primary focus in this review. Accordingly, as described in Chapter 4, the exposure and risk assessment estimates Pb exposure for children (less than 7 years of age), and associated risk of neurocognitive effects in terms of IQ decrements. In addition to the risks (IQ decrement) that we quantitatively estimated, we recognize that there may be long-term adverse consequences of such deficits over a lifetime, that there is evidence of other health effects occurring at similar or higher exposures for young children, and that other health evidence demonstrates associations between Pb exposure and adverse health effects in adults (see Chapter 3). As in Chapter 4 (Section 4.4), we focus predominantly on risk estimates derived using the log-linear with low-

exposure linearization (LLL) concentration-response function, also noting the range associated with the other three functions.

In interpreting the quantitative risk estimates for IQ decrement, we are aware of the significant implications of potential shifts in the distribution of IQ for the exposed population (e.g., CD, Sections 8.6.1 and 8.6.2; Bellinger, 2004; Needleman et al., 1982; Weiss, 1988; Weiss, 1990). As noted in the CD, a modest change in the mean for a health index at the individual level can have substantial implications at the population level (CD, p. 8-77). For example for an individual functioning in the low range of IQ due to the influence of risk factors other than Pb, a Pb-associated IQ decline of a few points might be sufficient to drop that individual into the range associated with increased risk of educational, vocational, and social handicap (CD, p. 8-77). Further as noted in Section 3.4.2, given a somewhat uniform manifestation of Pb-related decrements across the range of IQ scores in a population, "a downward shift in the mean IQ value is not associated only with a substantial increase in the percentage of individuals achieving very low scores, but also with substantial decreases in percentages achieving very high scores" (CD, p. 8-81). As recognized in Section 5.4.3, the CASAC Pb Panel has advised on this point that "a population loss of 1-2 IQ points is highly significant from a public health perspective" (Henderson, 2007a, p. 6).

In considering the risk estimates, we will describe both those for the median and for an upper percentile, the 95th. In so doing, we emphasize that in setting the standard in 1978, the Agency accorded risk management significance to the 99.5th percentile by selecting a mean blood Pb level intended to bring 99.5 percent of the population to or below the then described maximum safe blood Pb level (see Section 5.2.1.1.2). Similarly, in their advice to the Agency in this review, CASAC stated that "the primary lead standard should be set so as to protect 99.5% of the population" (Henderson, 2007a, p. 6). In considering estimates from the quantitative assessment that will inform conclusions consistent with this objective, however, we and CASAC also recognize uncertainties in our risk estimates at the edges of the distribution and consequently report the 95th percentile as our estimate of the high end of the risk distribution (Henderson, 2007b, p. 3). In so doing, however, we note that there are individuals in the population expected to have higher risk, the consideration of which is important given the risk management objectives for the current standard with regard to the 99.5th percentile.

As summarized in Chapter 4 and discussed in more detail in the Risk Assessment Report (e.g., Sections 2.4.3, 3.2.2 and 3.4), in addition to estimating IQ loss associated with the combined exposure to Pb from all exposure pathways, we have estimated IQ loss for two policy-relevant categories of exposure pathways. These are "recent air", which conceptually is intended to include contributions to blood Pb associated with Pb that has recently been in the air, and "past air", intended to include contributions to blood Pb associated with Pb that was in the air in

the past but not in the air recently. In the exposure modeling conducted for the risk assessment, the exposure pathways assigned to the "recent air" category were inhalation of ambient air Pb and ingestion of the component of indoor dust Pb that is predicted to be associated with ambient air concentrations. The exposure pathways assigned to the "past air" category were ingestion of outdoor soil/dust Pb and ingestion of the component of indoor dust Pb not assigned to recent air. As recognized in Chapter 4 and described more fully in Sections 2.4.3 and 3.2.2 of the Risk Assessment Report, there are various limitations associated with our modeling tools that affected our estimates for these two categories. As a result, blood Pb levels and associated risks of greatest interest in this review – those associated with exposure pathways involving ambient air Pb and current levels of Pb emitted to the air (including via resuspension) - are likely to fall between estimates for recent air and those for the sum of recent plus past air. Accordingly, we have considered that those two sets of estimates provide a range of interest, with regard to policy-relevant Pb, in this review.

In considering the adequacy of the current standard, it is important to note that the standard is currently met throughout the country with very few exceptions. Further, the national composite average maximum quarterly mean based on 198 active monitoring sites during 2003-2005 is 0.17 $\mu g/m^3$, an order of magnitude below the current standard (Section 2.3.2.4). Review of the current monitoring network in light of current information on Pb sources and emissions, however, indicates that we do not have monitors near many of the larger sources and leads us to conclude that we are likely underestimating the extent of occurrences of relatively higher Pb concentrations (Section 2.3.2.1).

We have estimated exposure and risk associated with <u>current conditions</u> in a general urban case study and in three location-specific urban case studies in areas where air concentrations fall significantly below the current standard. For the general urban case study, which is a simplified representation of urban areas (see summary of limitations and uncertainties in Section 4.2.7), median estimates of total Pb-related IQ loss range from 1.5 to 6.3 points (across all four concentration-response functions), with estimates based on the LLL function of 4.5 and 4.7 points, for the mean and high-end current conditions scenarios, respectively (Table 4-3). Associated estimates for exposure pathway contributions to total IQ loss (LLL estimate) at the population median in these two scenarios indicate that IQ loss associated with policy-relevant Pb falls somewhere between 1.3 and 3.6 points (Risk Assessment Report, Table 5-9 entries for recent air and recent air plus past air). At the 95th percentile for total IQ loss (LLL estimate), IQ loss associated with policy-relevant Pb is estimated to fall somewhere between 2.2 and 6.0 points (Risk Assessment Report, Table 5-9).

For the three location-specific urban case studies, median estimates of total Pb-related IQ loss for current conditions range from 1.4 to 5.2 points (across all four concentration-response

functions), with estimates based on the LLL function all being 4.2 points (Table 4-3).[7] Median IQ loss associated with policy-relevant Pb (LLL function) is estimated to fall between 0.6 to 2.9 points IQ loss (Risk Assessment Report, Table 5-9). The 95th percentile estimates for total Pb-related IQ loss across the three location-specific urban case studies range from 4.1 to 11.4 points (across all four concentration-response functions), with estimates based on the LLL function ranging from 7.5 to 7.6 points (Table 4-4). At the 95th percentile for the three location-specific urban case studies, IQ loss associated with policy-relevant Pb (LLL function) is estimated to fall between 1.2 to 5.2 points IQ loss (Risk Assessment Report, Table 5-10).

In order to more completely consider exposure and risk associated with the current standard, we have developed estimates for a case study (including additional focus on a small subarea) based on air quality projected to just meet the standard in a location of the country where air concentrations do not meet the current NAAQS (the primary Pb smelter case study). In so doing, we consider it extremely unlikely that air concentrations in urban areas across the U.S. that are currently well below the current standard would increase to just meet the standard. However, we recognize the potential for air Pb concentrations in some areas currently well below the standard to increase to just meet the standard by way of, for example, expansion of existing sources (e.g., facilities operating as secondary smelters exercising previously used capabilities as primary smelters) or by the congregation of multiple Pb sources in adjacent locations. Accordingly, we have simulated scenario (increased Pb concentrations to just meet the current standard) in a general urban case study and in three location-specific urban case studies. In this scenario, we note substantial uncertainty in simulating how the profile of Pb concentrations might change in the hypothetical case where concentrations increase to just meet the current standard.

Turning first to the estimates of total blood Pb for the current NAAQS scenario simulated for the location-specific urban case studies (e.g., Tables 4-1 and 4-2), we consider the extent to which exposures associated with increased air Pb concentrations that simulate just meeting the current standard are estimated to increase blood Pb levels in young children. The magnitude of this for the median total blood Pb ranges from 0.3 µg/dL (an increase of 20 percent) in the case of the Cleveland study area, for which current conditions are estimated to be approximately one fourth of the current NAAQS, up to approximately 1 µg/dL (an increase of 50 to 70%) for the

[7] As described in Chapter 4 (and Section 5.2.3.2 of the Risk Assessment Report), although the maximum quarterly average concentration for the highest monitor in each study area differs among the three areas by a factor of 4 (0.09 to 0.36 ug/m3), the population weighted air Pb concentrations for these three study areas are more similar and differ by approximately a factor of 2, with the study area with highest maximum quarterly average concentration having a lower population weighted air concentration that is more similar to the other two areas. This similarity in population weighted concentrations explains the finding of similar total IQ loss across the three study areas.

Chicago and Los Angeles study areas, for which current conditions are estimated to be at or below one tenth of the current NAAQS.

Our estimates of IQ loss (for child with median total IQ loss estimate) associated with recent air plus past air Pb at exposures allowed by just meeting the current NAAQS in the primary Pb smelter case study (Table 4-5) differ when considering the full study area (10 km radius) or the 1.5 km radius subarea. Estimates for median IQ loss associated with the recent air plus past air category of exposure pathways for the full study area range from 0.6 point to 2.3 points (for the range of concentration-response functions), while these estimates for the subarea range from 3.2 points to 9.4 points IQ loss (Tables 4-5). The estimates (recent plus past) for the median based on the LLL concentration-response function are 1.9 points IQ loss for the full study area and 6.0 points for the subarea. The 95th percentile estimates of *total* IQ loss in the subarea range from 5.0 to 12.4 points, with an associated range for the recent air plus past air of 4.2 to 10.4 points (Table 4-6).

For the current NAAQS scenario in the three location-specific case studies, estimates of IQ loss associated with policy-relevant Pb for the median total IQ loss range from 0.6 points loss (recent air estimate using low-end concentration-response function) to 7.4 points loss (recent plus past air estimate using the high-end concentration-response function) (Table 4-5). The corresponding estimates based on the LLL concentration-response function range from 2.7 points (lowest location-specific recent air estimate) to 4.7 points IQ loss (highest location-specific recent plus past air estimate) (Table 4-5). The comparable estimates of IQ loss for children at the 95th percentile range from 2.6 to 7.6 points for the LLL concentration-response function (Table 4-6).

Further, in comparing current NAAQS scenario estimates to current conditions estimates for the three location-specific urban case studies, we estimate a difference in total Pb-related IQ loss for the median of about 0.5 to 1.4 points using the LLL C-R function and a similar magnitude of difference for the 95th percentile (Tables 4-3 and 4-4). The corresponding estimate for the general urban case study is 1.1 to 1.3 points higher total Pb-related IQ loss for the current NAAQS scenario compared to the two current conditions scenarios (Tables 4-3 and 4-4).

Our estimates of median and 95th percentile IQ loss associated with policy-relevant Pb exposure for air quality scenarios under current conditions (which meet the current NAAQS) and, particularly those reflecting increased air Pb concentrations simulated to just meet the current standard, indicate levels of IQ loss that may reasonably be judged to be highly significant from a public health perspective. Further, for the three location-specific urban case studies, the estimated differences in incidences of children with IQ loss greater than one point and with IQ loss greater than seven points in comparing current conditions to those associated with the current NAAQS indicate the potential for significant numbers of children to be negatively

affected if air Pb concentrations increased to levels just meeting the current standard. Estimates of the additional number of children with IQ loss greater than 1 point (based on the LLL concentration-response function) in these three study areas for the current NAAQS scenario compared to current conditions range from 100 to 6,000 across the 3 locations (Table 4-7). The corresponding estimates for the additional number of children with IQ loss greater than 7 points for the current NAAQS as compared to the current conditions scenario range from 600 to 35,000 (Table 4-8). These latter values for the change in incidence of children with greater than 7 points Pb-related IQ loss represent 5 to 17 percent of the children (aged less than 7 years of age) in these study areas. This increase corresponds to approximately a doubling in the number of children with this magnitude of Pb-related IQ loss in the study area most affected.

Beyond our finding of estimated decrements in IQ for policy-relevant exposures associated with the current NAAQS that are clearly of a magnitude that might be reasonably be judged to be highly significant from a public health perspective, there are other, unquantified adverse neurocognitive effects that may occur at similarly low exposures which might additionally contribute to reduced academic performance, which may have adverse consequences over a lifetime (Section 3.3.1; CD, pp. 8-29 to 8-30). Additional impacts at low levels of childhood exposure summarized in Chapter 3 and described in detail in the CD, that were not quantified in the risk assessment, include: other neurological effects (sensory, motor, cognitive and behavioral), immune system effects (including some related to allergic responses and asthma), and early effects related to anemia. Taken together, we judge that the quantified IQ effects associated with the current NAAQS and other, non-quantified effects are important from a public health perspective, indicating a need for consideration of revision of the standard to provide an appreciable increase in public health protection.

5.4.3 CASAC Advice and Recommendations

In our consideration of the adequacy of the current standard, in addition to the evidence- and risk/exposure-based information discussed above, we have also considered the advice and recommendations of CASAC, based on their review of the CD and the earlier draft of this document and the related technical support document, as well as comments from the public on earlier drafts of this document and the related technical support document[8]. With regard to the public comments, those that addressed adequacy of the current standard concluded that the current standard is inadequate and should be revised, suggesting appreciable reductions in the

[8] All written comments submitted to the Agency will be available in the docket for this rulemaking, as will be transcripts of the public meetings held in conjunction with CASAC's review of the earlier draft of this document and the first draft of the related technical support document, and of draft and final versions of the CD on which this document is based.

level. No comments were received expressing the view that the current standard is adequate. One comment was received arguing not that the standard was inadequate but rather that conditions justified that it should be revoked.

In both the 1990 review and this review of the standard set in 1978, CASAC has recommended consideration of more health protective NAAQS. In CASAC's review of the 1990 staff paper, as discussed in Section 5.2.2, they generally recommended consideration of levels below 1.0 $\mu g/m^3$, specifically recommended analyses of a standard set at 0.25 $\mu g/m^3$, and also recommended a monthly averaging time (CASAC, 1990). In two letters to the Administrator during the current review, CASAC has consistently recommended that the primary NAAQS should be "substantially lowered" from the current level of 1.5 $\mu g/m^3$ to a level of "0.2 $\mu g/m^3$ or less" (Henderson, 2007a, b). CASAC drew support for this recommendation from the current evidence, described in the CD, of health effects occurring at dramatically lower blood Pb levels than those indicated by the evidence available when the standard was set. Citing specific studies (referenced in their March 2007 letter, Attachment A), CASAC stated (Henderson, 2007a, p. 3):

> *Despite the dramatic decrease in environmental lead exposure, lead toxicity remains a major public health problem. Environmental lead exposure in children has been associated with increased risks for reading problems, school failure, Attention Deficit Hyperactivity Disorder (ADHD), delinquency, and criminal behavior (6–10). Among U.S. children, eight to fifteen years old, those in the highest quintile (> 2 μg/dl) of lead exposure were four times more likely to have doctor-diagnosed ADHD (11). Moreover, there is no evidence of a threshold for the adverse consequences of lead exposure; studies show that the decrements in intellectual (cognitive) functions in children are proportionately greater at PbB [blood Pb] concentrations < 10 μg/dl, the concentration considered acceptable by the Centers for Disease Control (11–14).*
>
> *Lead's effects extend beyond childhood. In adults, lead exposure is a risk factor for some of the most prevalent diseases or conditions of industrialized society, including cardiovascular disease and renal disease (16–20). There is also compelling evidence that the risks for mortality from stroke and myocardial infarction are increased at PbB [blood Pb] concentrations below 10 μg/dl, which is considerably lower than those considered acceptable for adults (19). Finally, although less definitive, there is also evidence that lead exposure during pregnancy is a risk factor for spontaneous abortion or miscarriage at PbB [blood Pb] concentrations < 10 μg/dl (21).*

CASAC concluded that the current Pb NAAQS "are totally inadequate for assuring the necessary decreases of lead exposures in sensitive U.S. populations below those current health hazard markers identified by a wealth of new epidemiological, experimental and mechanistic studies", and stated that "Consequently, it is the CASAC Lead Review Panel's considered judgment that

the NAAQS for Lead must be decreased to fully-protect both the health of children and adult populations" (Henderson, 2007a, p. 5).

5.4.4 Staff Conclusions and Recommendations

Staff has considered the appropriateness of maintaining a NAAQS for Pb and the retention of Pb on the list of criteria pollutants in light of the changed circumstances since the current standard was set in 1978. We conclude that the currently available information does not support either delisting Pb as a criteria pollutant or revoking, and not replacing, the current Pb NAAQS, a conclusion that has also been reached by CASAC. Accordingly, we recommend that Pb not be delisted as a criteria pollutant and that the Pb NAAQS not be revoked.

In considering the adequacy of the current standard, staff gives great weight to the body of available evidence that is much expanded from that available when the current standard was set, and which demonstrates the occurrence of adverse health effects at appreciably lower blood Pb levels than those demonstrated by the evidence at the time the standard was set. Further, the current health effects evidence and findings in our exposure assessment, like the information available at the time the standard was set, supports the conclusion that airborne Pb exposure pathways (by inhalation and ingestion) contribute to blood Pb levels in young children, and that the proportion of the contribution from these pathways to total blood Pb levels is likely larger than that estimated when the standard was set and may be several times higher.

In areas projected to just meet the current standard, the quantitative estimates of risk (for IQ decrement) associated with policy-relevant Pb indicate risk of a magnitude that may reasonably be judged to be highly significant from a public health perspective, and which CASAC has judged to be so. Further, although the current monitoring data indicate few areas with airborne Pb near or just exceeding the current standard, the staff recognizes significant limitations with our current monitoring network that indicate we are likely underestimating the extent of occurrences of relatively higher Pb concentrations (Section 2.3.2.1).

In summary, staff draws conclusions with regard to the adequacy of the current standard from both the evidence and from the exposure and risk assessments, in light of related limitations and uncertainties. We conclude that the overall body of evidence clearly calls into question the adequacy of the current standard and provides strong support for consideration of an alternative Pb standard that would provide an appreciable improvement in health protection for sensitive groups, including most notably young children, against an array of effects, most importantly including effects on the developing nervous system. We also conclude that risks projected to remain upon meeting the current standard, based on the exposure and risk assessment, are indicative of risks to sensitive groups that can reasonably be judged to be important from a public health perspective, which reinforces our conclusion that consideration should be given to

revising the level of the standard so as to provide increased public health protection. Accordingly, we recommend that the Administrator consider revision of the primary NAAQS for Pb. Staff conclusions and recommendations for the indicator, averaging time, form and level for an alternative, more protective primary standard for Pb are discussed in the following sections.

5.5 ELEMENTS OF THE STANDARD

The four elements of the standard – indicator, averaging time, form and level serve to define the standard and must be considered collectively in evaluating the health and welfare protection afforded by the standards. In the previous section, staff concluded that the current standard is not adequate and should be revised. In considering a revision to the current standard in the subsequent sections, we consider each of the four elements of the standard as to how they might be revised to provide a primary standard for Pb that is requisite to protect public health.

5.5.1 Indicator

The indicator for the current standard is Pb-TSP. When the standard was set, EPA considered identifying Pb-PM_{10} as the indicator in response to comments expressing concern that because only a fraction of airborne particulate matter is respirable, an air standard based on total air Pb is unnecessarily stringent. The Agency responded that while it agreed that some Pb particles are too small or too large to be deposited in the respiratory system, a significant component of exposures can be ingestion of materials contaminated by deposition of Pb from the air. In addition to the route of ingestion and absorption from the gastrointestinal tract, nonrespirable Pb in the environment may, at some point, become respirable through weathering or mechanical action. EPA concluded that total airborne Pb, both respirable and nonrespirable fractions should be addressed by the air standard.

In the 1990 Staff Paper, staff reconsidered this issue in light of information regarding limitations of the high-volume sampler used for the Pb-TSP measurements and concurred with the continued use of TSP as the indicator (USEPA, 1990):

> *Given that exposure to lead occurs not only via direct inhalation, but via ingestion of deposited particles as well, especially among young children, the hi-vol provides a more complete measure of the total impact of ambient air lead … Despite its shortcomings, the staff believes the high-volume sampler will provide a reasonable indicator for determination of compliance …*

In their advice to the Agency CASAC recommended consideration of a change in the indicator to utilize low-volume PM_{10} sampling (Henderson, 2007a, b). In so doing, CASAC recognized the "importance of coarse dust contributions to total Pb ingestion", that a scaling of the NAAQS level would be needed to accommodate the loss of very large coarse-mode Pb

particles, and that concurrent Pb-PM_{10} and Pb-TSP sampling would be needed to inform development of scaling factors (Henderson, 2007b). The September 2007 CASAC letter states that the CASAC Lead Panel "strongly encourages the Agency to consider revising the Pb reference method to allow sample collection by PM_{10}, rather than TSP samplers, accompanied by analysis with low-cost multi-elemental techniques like X-Ray Fluorescence (XRF) or Inductively Coupled Plasma–Mass Spectroscopy (ICP-MS)." While recognizing the importance of coarse dust contributions to total Pb exposure via the ingestion route and acknowledging that TSP sampling is likely to capture additional very coarse particles which are excluded by PM_{10} samplers, the Panel raised some concerns. The concerns regarded the precision and variability of TSP samplers and the inability to efficiently capture the non-homogeneity of very coarse particles in a national monitoring network. The Panel indicated that these concerns may need to be addressed in implementing additional monitoring sites and an increased frequency of sample collection that might be required with the substantial reduction in the level of the standard and the shorter averaging time that they recommend (Henderson, 2007b).

In considering the appropriate indicator, staff concurs with previous Agency conclusions that the health evidence indicates that Pb in all particle size fractions, not just respirable Pb, contributes to Pb in blood and to associated health effects. Additionally, the currently available information, from a very small set of collocated Pb-TSP and Pb-PM_{10} monitoring sites, does not support the derivation of a scaling factor which might be used to derive a level for the standard in terms of Pb-PM_{10} as the indicator.

The staff recognizes, however, that an indicator that exhibits low spatial variability is desirable such that it facilitates implementation of an effective monitoring network, i.e., one that assures identification of areas with the potential to exceed the NAAQS. For an indicator with low spatial variability, attainment/nonattainment outcomes would be less sensitive to exact placement of monitors. However, staff notes that there is an inherent tension between the perspective that Pb-TSP has high spatial variability and the expectation that a national scaling factor between the two indicators is possible.

There are several options that staff suggests be considered that might improve the available database and facilitate consideration of such a move in the future, while retaining Pb-TSP as the indicator for the NAAQS at this time. For example, the Administrator might consider describing a FEM in terms of PM_{10} that might be acceptably applied on a site-by-site basis where an appropriate relationship between Pb-TSP and Pb-PM_{10} can be developed based on site-specific data. Alternatively, use of such a FEM might be approved, in combination with more limited Pb-TSP monitoring, in areas where the Pb-TSP data indicate ambient Pb levels are well below the NAAQS level. These examples are intended purely for purposes of illustrating the

types of options the Administrator might consider. Specific details of any options will need to be supported by appropriate data analyses.

To the extent that Pb-PM_{10} exhibits less spatial variability and that a scaling factor can be developed from Pb-PM_{10} data to level for the standard in terms of Pb-TSP, staff recommends the Administrator consider moving to a Pb-PM_{10} indicator in the future. One of the issues to consider when moving to a Pb-PM_{10} indicator is whether regulating concentrations of Pb-PM_{10} will lead to appropriate controls on Pb emissions from sources with a large percentage of Pb in the greater than 10 micron size range (e.g., fugitive dust emissions from Pb smelters). It is reasonable to believe that Pb-PM_{10}/Pb-TSP ratios are sensitive to distance from emissions sources (due to faster deposition of larger particles). As such, the use of a Pb-PM_{10} indicator may have a significant influence on the degree of Pb controls needed from emission sources.

In conclusion, the staff recommends retaining Pb-TSP as the indicator for the primary standard, coincident with activities intended to encourage collection and development of datasets that will improve our understanding of national and site-specific relationships between PM_{10} and Pb-TSP to support a more informed consideration of indicator during the next review. Staff suggests that such activities be inclusive of uses of FEMs such as those described above where sufficient data are available to adequately demonstrate a relationship between Pb-TSP and Pb-PM_{10}.

5.5.2 Averaging Time and Form

In considering alternative Pb standards that would provide increased public health protection, staff has taken into account both evidence-based and exposure- and risk-based considerations. We have also considered analyses of monitoring data comparing metrics reflecting both averaging time and form.

The basis for the averaging time of the current standard (Section 5.2.1.2) reflects consideration of the evidence available when the Pb NAAQS were promulgated in 1978. At that time, the Agency had concluded that the level of the standards, 1.5 $\mu g/m^3$, would be a "safe ceiling for indefinite exposure of young children" (43 FR 46250), and that the slightly greater possibility of elevated air lead levels within the quarterly averaging period as contrasted to the monthly averaging period proposed in 1977 (43 FR 63076), was not significant for health. These conclusions were based in part on the Agency's interpretation of the health effects evidence as indicating that 30 µg/dL was the maximum safe level of blood Pb for an individual child.

As discussed in Sections 3.3 and 5.4.1, the currently available health effects evidence indicates a variety of neurological effects, as well as immune system and hematological effects, associated with levels below 10 µg/dL as a central tendency metric of study cohorts of young children. Further, there is currently no blood Pb level for an individual child recognized to be

without adverse effect. Accordingly, to the extent that air Pb contributes to variation in blood Pb, we currently cannot identify a safe ceiling for indefinite exposure of young children.

Additionally, several aspects of the current health effects evidence for Pb pertain to the consideration of averaging time:

- Children are exposed to ambient Pb via inhalation and ingestion, with Pb taken into the body absorbed through the lungs and through the gastrointestinal tract. Studies on Pb uptake, elimination and distribution show that Pb is absorbed into peripheral tissues in adults within a few days (USEPA 1986a; USEPA 1990b, p. IV-2) Absorption of Pb from the gastrointestinal tract appears to be greater and faster in children as compared to adults (CD, Section 4.2.1). Once absorbed, it is quickly distributed from plasma to red blood cells and throughout the body.
- Lead accumulates in the body and is only slowly removed, with bone Pb serving as a blood Pb source for years after exposure and as a source of fetal Pb exposure during pregnancy (CD, Sections 4.3.1.4 and 4.3.1.5).
- Blood Pb levels, including levels of the toxicologically active fraction, respond quickly to increased Pb exposure, such that an abrupt increase in Pb uptake rapidly changes blood Pb levels, with the time to reach a new quasi-steady state with the total body burden after such an occurrence projected to be approximately 75 to 100 days (CD, p. 4-27).
- The elimination half-life, which describes the time for blood Pb levels to stabilize after a reduction in exposure, for the dominant phase for blood Pb responses to changes in exposure is on the order of 20 to 30 days for adults (CD, p. 4-25). Blood elimination half-lives are influenced by contributions from bone. Given the tighter coupling in children of bone stores with blood levels, children's blood Pb is expected to respond more quickly than adults (CD, pp. 4-20 and 4-27).
- Data from NHANES II and an analysis of the temporal relationship between gasoline consumption data and blood lead data generally support the inference of a prompt response of children's blood Pb levels to changes in exposure in that children's blood lead levels and the number of children with elevated blood Pb levels appear to respond to monthly variations in Pb emissions from Pb in gasoline (EPA, 1986a, p. 11-39; Rabinowitz and Needleman, 1983; Schwartz and Pitcher, 1989).
- The evidence with regard to sensitive neurological effects is limited in what it indicates regarding the specific duration of exposure associated with effect, although it indicates both the sensitivity of the first 3 years of life and a sustained sensitivity throughout the lifespan as the human central nervous system continues to mature and be vulnerable to neurotoxicants (CD, Section 8.4.2.7). The animal evidence supports our understanding of periods of development with increased vulnerability to specific types of effect (CD, Section 5.3), and indicates a potential importance of exposures on the order of months.
- Evidence of a differing sensitivity of the immune system to Pb across and within different periods of life stages indicates a potential importance of exposures as short as weeks to months duration. For example, the animal evidence suggests that the gestation period is the most sensitive life stage followed by early neonatal stage, and

within these life stages, critical windows of vulnerability are likely to exist (CD, Section 5.9 and p. 5-245).

Further, evidence described in the CD and the risk analyses described in Chapter 4 indicate that ingestion of dust is the predominant exposure pathway for young children to policy-relevant Pb, and that there is a strong association between indoor dust Pb levels and children's blood Pb levels. Further, a recent study of dustfall near an open window in New York City indicates that airborne Pb can contribute Pb in dust on interior surfaces at a median loading of 4.8 ug/ft^2 (52 ug/m^2) per week (CD, p. 3-28; Caravanos et al., 2006). The response time in rooms or houses with closed windows would be slower. But this study indicates that during times when windows are open, there is a relatively rapid response of indoor dust Pb loading to airborne Pb.

While some of these aspects of the health effects evidence would be consistent with a quarterly averaging time, taken as a whole, and in combination with information on potential response time for indoor dust Pb levels, there is support for consideration of an averaging time shorter than a calendar quarter.

Another consideration cited in selection of the averaging time when the standards were first promulgated in 1978 was that an analysis of ambient measurements available at the time indicated that the distribution of air Pb levels was such that there was little possibility that there could be sustained periods greatly above the average value in situations where the quarterly standard was achieved. This may have been related to the pattern of lead emissions at the time the standard was set, which differed from the pattern today in that, due to emissions from cars and trucks at that time, emissions were more spatially distributed. The air quality analysis in Chapter 2 for 2003-2005 indicates the presence of areas in the U.S. currently where temporal variability does create differences between average quarterly levels and levels sustained for shorter than quarterly periods. For example, four percent of the monitoring sites in the 3-year analysis dataset that meet the current standard as an average over a calendar quarter exceed the level of the current standard when considering an average for any individual month (see Section 2.3.2.5). The same analysis indicates that this number is as high as ten percent for some alternate lower levels.

In further considering the appropriate form of the standard that might accompany a shorter averaging time, the staff has considered analyses using the air quality data for 2003-2005 (Section 2.3.2). Maximum quarterly average and various monthly statistics were derived for each year across the three year Pb-TSP dataset and also for the entire three year period. The latter time period is consistent with the three calendar year attainment period that has been adopted for the ozone and particulate matter NAAQS subsequent to the promulgation of the Pb NAAQS, and was a recommendation of the 1990 Staff paper. For the three year period, the

monthly statistics derived are maximum monthly mean, second maximum monthly mean, average of three overall highest monthly means and average of three annual maximum monthly means; these statistical forms were also considered in the 1990 Staff paper. Additionally, the maximum and 2nd maximum monthly means for each year of the three year data set were derived, as well as the averages of these individual year statistics.

With regard to comparison of monthly forms with the maximum quarterly mean, the average Pb-TSP maximum monthly mean among all 189 sites in the analysis is notably higher (nearly a factor of two) than the average of the average maximum quarterly mean among these sites. Further, this difference is slightly greater for source-oriented sites than non source-oriented sites or urban sites (e.g., a factor of approximately 1.8 as compared to one of approximately 1.6), indicating perhaps an influence of variability in emissions. The alternate forms of a monthly averaging time that were analyzed yield an across-site average that is similar although slightly higher than the quarterly average (e.g., Figure 2-8).

In the 1990 Staff Paper, analyses of computer simulated values and of Pb measurements collected at point source oriented sites included consideration of six different forms for a monthly average (USEPA, 1990b). These analyses focused on forms computed over a three calendar year attainment period to be consistent with multi-year formats adopted for the ozone and particulate matter NAAQS (subsequent to the promulgation of the Pb NAAQS). From these analyses, staff demonstrated that the maximum monthly average yielded the highest design value[9], and, among an intermediate design value set of three alternatives, the simplest form was the second maximum month.

The analyses described in Chapter 2 consider both a period of three calendar years and one of an individual calendar year (with the form of the current standard being the maximum quarterly mean in any one year). These analyses indicate that with regard to either single-year or 3-year statistics for the 2003-2005 dataset, a 2nd maximum monthly mean yields very similar, although just slightly greater, numbers of sites exceeding various alternate levels as a maximum quarterly mean, with both yielding fewer exceedances than a maximum monthly mean. Other forms included in a subset of the analyses involved the average of metrics across a three-year period, such as the average of the 3 maximum monthly means in a three-year period and the average of three annual maximum monthly means.

In considering whether it is appropriate to change the form to apply to a three-year period, as is common practice for NAAQS for other pollutants, from the current single-year period, staff took into account the following. In a three-year approach, a monitor would be

[9] The design value is the estimated air concentration at a specific location in terms of the standard (i.e., with regard to averaging time, form and indicator).

considered to be in violation of the NAAQS as of a certain date if in any of the three previous calendar years with sufficiently complete data, the value of the selected form of the indicator (e.g., maximum monthly average, second maximum monthly average) exceeded the level of the NAAQS. A monitor, once having violated the NAAQS, would not be considered to have attained the NAAQS until three years have passed without such an exceedance. This three-year approach would provide more stability in the air quality management process, and would help ensure that areas initially found to be violating the NAAQS have effectively controlled the contributing lead emissions before being redesignated to attainment/maintenance. Analysis of Pb concentration data has shown instances in which a monitor has exceeded a Pb concentration in the range recommended for consideration in one year, not exceeded it the second year, and exceeded it again in the third year, apparently not due to substantial changes in nearby emissions but rather to meteorological variability and the monitoring schedule. A three-year approach for Pb would be consistent with the period used for the current NAAQS for PM_{10}, $PM_{2.5}$, and ozone. EPA established three-year periods for those NAAQS to provide more stability to the air quality management process, consistent with CASAC recommendation made during the respective NAAQS review processes.

In their review of the 1990 Staff Paper during the last review, the CASAC Pb panel concurred with the staff recommendation to express the lead NAAQS as a monthly standard not to be exceeded more than once in three years. Similarly, the current CASAC, in their advice to the Agency during this review, has recommended that the Agency consider changing from a calendar quarter to a monthly averaging time (Henderson, 2007a). In making that recommendation, CASAC emphasized support from studies that suggest that blood Pb concentrations respond at shorter time scales than would be captured completely by quarterly values, as indicated by their description of their recommendation for adoption of a monthly averaging time as "more protective of human health in light of the response of blood lead concentrations that occur at sub-quarterly time scales" (Henderson, 2007a).

With regard to form of the standard, the current CASAC Pb Panel stated that one could "consider having the lead standards based on the second highest monthly average, a form that appears to correlate well with using the maximum quarterly value", while also indicating that "the most protective form would be the highest monthly average in a year" (Henderson, 2007a).

The following observations support consideration of an averaging time on the order of a calendar month or quarter: 1) the health evidence indicates that very short exposures can lead to increases in blood Pb lead levels, 2) the time period of response of indoor dust Pb to airborne Pb can be on the order of weeks and, 3) the health evidence indicates that adverse effects may occur with exposures during relatively short windows of susceptibility, such prenatally and as in developing infants. The staff also recognizes the limited evidence specific to the consideration

of the particular duration of sustained airborne Pb levels having the potential to contribute to the adverse health effects identified as most relevant to this review.

After considering the current evidence and analyses of air quality, we conclude that this information provides support for an averaging time at least as short as a calendar quarter and for considering a change of the averaging time to a calendar month. In considering a form for a monthly averaging time, staff concludes that a form of maximum or second maximum would be appropriate. Further, staff concludes that it is appropriate to also consider changing the duration of the time period evaluated in considering attainment, to a three-year period as is common practice for NAAQS for other pollutants (from the current single-year period).

5.5.3 Level

Staff's consideration of alternative levels for the primary Pb NAAQS that would provide greater protection against the array of Pb-related adverse health effects than that afforded by the current standard builds on our conclusion that the overall body of evidence indicates that the current standard is inadequate to protect public health and should be appreciably lower (Section 5.4).

5.5.3.1 Evidence-based Considerations

As an initial matter, staff recognizes obstacles that preclude using the epidemiological evidence directly as the basis for selecting appropriate levels for the Administrator to consider. As summarized in Chapter 3 and discussed in greater depth in the CD (Sections 4.3 and 6.1.3), the epidemiological evidence that associates Pb exposures with health effects uses blood Pb as the dose metric. Further, for the health effects receiving greatest emphasis in this review (neurological effects on the developing nervous system), no threshold levels can be discerned from the evidence. As was recognized at the time of the last review, estimating a threshold for toxic effects of Pb on the central nervous system entails a number of difficulties (CD, pp. 6-10 to 6-11). The task is made still more complex by support in the evidence for a nonlinear rather than linear relationship of blood Pb with neurocognitive decrement, with greater risk of decrement-associated changes in blood Pb at the lower levels of blood Pb in the exposed population (Section 3.3.7; CD, Section 6.2.13).

As the evidence cannot be used directly as the basis for selecting levels, we turn to somewhat more indirect uses of the evidence, such as the framework applied in the establishment of the standard. As discussed above (Sections 3.3 and 5.4.1), the body of evidence is much expanded from that available when the current standard was set. In the following discussion we have applied the 1978 framework to the currently available evidence.

With regard to the sensitive population, staff identifies young children, the same population identified in 1978, as the key sensitive population for Pb exposures. Our recognition

of young children as the sensitive population is based on the evidence summarized in Sections 3.3 and 5.4.1 and described in more detail in the CD.

As recognized in Section 5.4.1 above, the current evidence demonstrates the occurrence of adverse health effects, including those on the developing nervous system, associated with blood Pb levels extending well below 10 μg/dL to 5 μg/dL and possibly lower. Further, the current evidence does not indicate a threshold for the more sensitive health endpoints such as adverse effects on the developing nervous system (CD, pp. 5-71 to 5-74 and Section 6.2.13). This differs from the Agency's inference in the 1978 rulemaking of a threshold of 40 μg/dL blood Pb for effects of Pb considered clearly adverse to health, i.e., impairment of heme synthesis and other effects which result in anemia. Thus, the level of Pb in children's blood associated with adverse health effect has dropped by more than a factor of 8, from 40 μg/dL to less than 5 μg/dL, with some studies indicating Pb effects on intellectual attainment of young children at blood Pb levels ranging from 2 to 8 μg/dL (CD, Sections 6.2, 8.4.2 and 8.4.2.6), including a finding of similar Pb-related effects in a study of a nationally representative sample of children in which the mean blood Pb level was 1.9 μg/dL (CD, pp. 6-31 to 6-32; Lanphear et al., 2000).

As when the standard was set in 1978, we recognize that there remain today important contributions to blood Pb levels from nonair sources.[10] As discussed in Section 5.4.1, these contributions have been reduced since 1978, with estimates of reduction in the dietary component of 70 to 95 percent (CD, Section 3.4). The evidence is limited with regard to the aggregate reduction since 1978 of all nonair sources to blood Pb. However, the available evidence and some preliminary analysis led CASAC to recommend consideration of 1.0 to 1.4 μg/dL or lower as an estimate of the nonair component of blood Pb (Henderson, 2007a, Appendix D). The value of 1.4 μg/dL was the geometric mean blood Pb level derived from a simulation of current nonair exposures using the IEUBK model (Henderson, 2007a, pp. F-60 to F-61).

Regarding the relationship between air and blood, while the evidence demonstrates that airborne Pb influences blood Pb concentrations through a combination of inhalation and ingestion exposure pathways, estimates of the quantitative relationship (i.e., air-to-blood ratio) available in the evidence vary (USEPA, 1986a; Brunekreef, 1984) and there is uncertainty as to the values that pertain to current exposures. Studies summarized in the 1986 CD typically yield estimates in the range of 1:3 to 1:5, with some as high as 1:10 or higher (with regard to the air-

[10] It should be noted that deposition of airborne Pb is a major source of Pb in food (as is house dust, which may also be attributable to deposition of ambient air lead) (CD p. 3-54). Thus, although the risk assessment characterizes dietary Pb as "background," reductions in ambient air Pb have the potential to reduce exposures through dietary Pb as well.

influenced increase in blood Pb) (USEPA, 1986a; Brunekreef, 1984). Findings in a more recent study identified in the 2006 CD of blood Pb response to reduced air concentrations indicate a ratio on the order of 1:7 (CD, pp. 3-23 to 3-24; Hilts et al., 2003). A value of 1:5 has been used by the World Health Organization (2000).

Simply applying the framework relied upon in setting the standard in 1978 to the currently available information illustrates the need for a level for the standard that is appreciably lower than the current level. For example, replacement of the 1978 blood Pb target of 15 µg/dL for the child population geometric mean with a level of 2 µg/dL reflecting, not a recognized "safe" exposure level as was the case with 15 µg/dL in 1978, but some of the lowest population levels associated with adverse effect in the current evidence (e.g., CD, p. E-9)[11], and subsequent subtraction of 1 to 1.4 µg/dL, representing nonair sources, yields 0.6 to 1 µg/dL as a target for the air contribution to blood Pb. Division of the air target by 5, consistent with currently available information on the ratio of air Pb to blood Pb, yields a level of 0.1 to 0.2 µg/m^3. We note, however, that we cannot today identify a blood Pb level considered safe from all adverse health effects. Thus, putting the current evidence into the framework by which the current NAAQS was derived, and recognizing that today's evidence provides no evidence of a threshold for the most sensitive effects, indicates a level for the NAAQS that is lower than the current level by approximately an order of magnitude or more. The evidence, while indicating the potential for adverse effects at or below the level used here in application of the 1978 framework, does not provide specificity with regard to the public health implications associated with lower levels that might directly inform our consideration of the lower part of a range for the standard.

5.5.3.2 Exposure- and Risk-based Considerations

To inform staff judgments about a range of levels for the standard that could provide an appropriate degree of public health protection, in addition to considering the health effects evidence (see preceding section), staff has also considered the quantitative estimates of exposure and health risks attributable to policy-relevant Pb upon meeting specific alternative levels of alternative Pb standards and the uncertainties in the estimated exposures and risks. As discussed above (Section 5.4.2), staff has based this evaluation on the exposure and risk assessment results presented in Chapter 4, in which exposures have been estimated for children of less than 7 years of age in six case studies. We also estimated the risk of adverse neurocognitive effects in terms of IQ decrements associated with total and policy-relevant Pb exposures, including incidence of

[11] It is important to note that the 1978 target of 15 was described as the geometric mean level associated with a 99.5 percentile of 30 µg/dL which the Agency described as a "safe level" for an individual child, while current epidemiological evidence using a large national database has identified an association with IQ decrement of blood Pb levels for which the geometric mean blood Pb concentration was 2 µg/dL (Lanphear et al., 2000).

different levels of IQ loss in three of the six case studies (Chapter 4). In so doing, we are mindful of the important uncertainties and limitations that are associated with the exposure and risk assessments, as discussed in Chapter 4. For example, with regard to the risk assessment, important uncertainties include those related to estimation of blood Pb concentration-response functions, particularly for blood Pb concentrations at and below the lower end of those represented in the epidemiological studies characterized in the CD; these uncertainties are described in Section 4.2.1.

As discussed in Section 4.2.7, we recognize important limitations in the design of, and data and methods employed in, the exposure and risk analyses, and the associated uncertainties with regard to the results. For example, the available monitoring data for Pb, relied upon for estimating current conditions for the urban case studies is quite limited, in that we do not have monitors near some of the larger known Pb sources (Section 2.3.2.1), which provides the potential for underestimation of current conditions. Additionally, we are uncertain about the proximity of existing monitors to other Pb sources not represented in the NEI but with the potential to influence exposures, such as old urban roadways and areas where housing with Pb paint has been demolished. All of these limitations raise uncertainty as to whether these data adequately capture the magnitude of ambient Pb concentrations to which the target population is currently exposed. Additionally, we did not have sufficient information and tools to evaluate all relevant sensitive groups (e.g., adults with chronic kidney disease) or all Pb-related adverse health effects (e.g., neurological effects other than IQ decrement, immune system effects, adult cardiovascular or renal effects), and the scope of our analyses was generally limited to estimating exposures and risks in six case studies intended to illustrate a variety of Pb exposure situations across the U.S., with three of them focused on specific areas in three cities. Thus, it is clear that national-scale public health impacts of ambient Pb exposures associated with meeting the current or alternative standards are larger than the quantitative estimates of Pb-related incidence of IQ decrement summarized in Chapter 4.

As mentioned in Section 5.4.3 and summarized in Chapter 4, we recognize limitations in our ability to characterize the contribution of policy-relevant Pb to total Pb exposure and Pb-related health risk. For example, given various limitations of our modeling tools, blood Pb levels associated with air-related exposure pathways and current levels of Pb emitted to the air (including via resuspension) are likely to fall between the estimates for “recent air” and those for “recent” plus “past air”, and we recognize limitations associated with our indoor dust Pb models that affect our ability to discern differences in the recent air category among different alternate air quality scenarios.

With these limitations in mind, we first consider the estimates of IQ loss associated with policy-relevant Pb at air Pb concentrations near those currently occurring in urban areas as

illustrated by conditions in the three cities chosen for the location-specific urban case studies, e.g., 0.09 to 0.36 μg/m^3 as a maximum quarterly average or 0.17 to 0.56 μg/m^3 as a maximum monthly average. Recognizing, as described above, that estimates of IQ loss associated with air-related exposure pathways and current levels of Pb emitted to the air (including via resuspension) are likely to fall between the estimates for "recent air" and those for "recent" plus "past air", we consider ranges reflecting those two categories. Further, as in Section 5.4.2 and for reasons discussed in Sections 4.2.1, 4.2.7 and 4.4, we focus on risk estimates derived using the LLL concentration-response function.

As described in Section 5.4.2, the ambient air Pb related IQ loss (based on LLL function) associated with the median IQ loss for current conditions in the three location-specific case studies (see Table 5-9 of the Risk Assessment Report) is estimated to fall between the estimates for recent air (0.6-0.7 points) and those for recent plus past air (2.9 points). This range appears to be of a magnitude that may reasonably be judged to be highly significant from a public health perspective in that it overlaps with the range of 1-2 points in IQ loss (see Sections 5.4.2 and 5.4.3). Comparable estimates for the current conditions scenarios in the general urban case study are still more significant with median estimates for IQ loss for the general urban case study ranging from 1.3 to 1.8 points for recent air and from 3.2 to 3.6 points for recent plus past air. The estimates of IQ loss for the 95th percentile (for the current conditions scenario in all of the urban case studies) extends higher than those for the median, ranging from 1.2-3.1 (recent air) to 5.2-6.0 points (recent plus past).

As mentioned previously, a current conditions scenario was not assessed for the primary Pb smelter case study, in which current air Pb concentrations across the study area are generally near or greater than the current NAAQS. However, this case study illustrates the potential impact of alternative NAAQS levels in comparison to the current NAAQS. Accordingly, we compare total IQ loss estimates across air quality scenarios, while noting that the simulations of alternative NAAQS scenarios in the risk assessment involve changes only to the recent air category of policy-relevant pathways and, consequently, likely provide an underestimate of IQ loss associated with all policy-relevant Pb. In the subarea of this case study, where risks are driven by the adjacent point source, reductions in median IQ loss (based on the LLL function) of 1.0 point, 1.8 points, 2.6 points and 2.8 points are estimated for the alternative NAAQS scenarios for the 0.5, 0.2, 0.05 and 0.02 μg/m^3 levels (in terms of a maximum monthly average), respectively (Table 4-3). From this it can be seen that in a situation where risks are driven by air concentrations that just meet the standard across the area assessed (as compared to a situation where most of the area is well below the standard), an appreciable difference in risk is seen between the levels of 0.5 and 0.2 μg/m^3, and additionally between those levels and the lower alternative standard levels assessed. The difference between the alternative standard levels of

0.5 and 0.2 μg/m^3 is seen to a smaller degree in the general urban case study. The difference becomes quite small in the Cleveland case study because as mentioned above, few in the population of that case study reside in the area with the highest air concentrations. Rather, the vast majority of the population in that case study resides in areas of notably lower levels.

As recognized above, differences in total IQ loss for different air quality scenarios reflect changes in the risk assessment simulations only to the recent air category of policy-relevant pathways. We also looked directly at risk estimates for the recent air category, which as recognized in Chapter 4 are likely underestimates of risk contributed by ambient air-related Pb; we observe that estimates of "recent air" IQ loss (based on the LLL function) at the 95th percentile of population total IQ loss are greater than one point for current conditions scenarios in all three location-specific urban case studies (Risk Assessment Report, Table 5-10). As noted above, ambient air Pb levels in these case studies extend down to 0.09 μg/m^3 maximum quarterly average (0.17 μg/m^3 maximum monthly average). The estimates for recent air (for the LLL concentration-response function) associated with the 95th percentile total IQ loss in the two current conditions scenarios of the general urban case study are 2.2 and 3.1 points.

Next we consider the extent to which alternative standard levels below current conditions are estimated to reduce blood Pb levels and associated health risk in young children (Tables 4-1 through 4-4), turning first to the estimates of total blood Pb. In the general urban case study, blood Pb levels for the median of the population associated with the lowest alternative NAAQS (0.02 μg/m^3) are estimated to be reduced from levels in the two current conditions scenarios by 14% (0.3 μg/dL) and 24% (0.5 μg/dL), respectively. For the 95th percentile of the population, the estimated reductions are similar in terms of percentage, but are higher in absolute values (1.7 and 1.0 μg/dL). For the three location-specific urban case studies, median blood Pb estimates associated with the lowest alternative standard are reduced from those associated with current conditions by approximately 10% in the Chicago and Cleveland study areas and 6% in the Los Angeles study area; similar percent reductions are estimated at the 95th percentile total blood Pb. For the localized subarea of the primary Pb smelter case study, a 65% reduction in both median and 95th percentile blood Pb (3 and 8.1 μg/dL, respectively) is estimated for the lowest alternative NAAQS as compared to the current NAAQS.[12]

We next consider the extent to which specific levels of alternative Pb standards reduce the estimated risks in terms of IQ loss attributable to policy-relevant exposures to Pb (Tables 4-3 and 4-4). For the general urban case study, estimated reductions in median Pb-related IQ loss

[12] This can be compared to reductions in blood Pb, for the primary Pb smelter case study subarea estimated to be associated with a change in the level from the current standard to the 0.2 μg/m^3 level (either averaging time) which are approximately 45-50% for both the median and 95th percentile values.

associated with reduced exposures at the lowest alternative NAAQS level (0.02 μg/m^3) were 0.5 and 0.7 points (LLL function) for the two current conditions scenarios. Reductions at the 95th percentile were of a similar magnitude. Among the three location-specific case study areas, estimated reductions in median Pb-related IQ loss associated with reduced exposures at the lowest alternate NAAQS as compared to current conditions range from 0.4 to 0.6 points for the high-end concentration-response function to 0.1 to 0.2 points for the low-end concentration-response functions, with estimates for the LLL function ranging from 0.2 to 0.3 points. The reduction at the 95th percentile, based on the LLL function, is 0.3-0.4 points. Reduced exposures associated with the lowest alternative NAAQS in the primary Pb smelter case study subarea as compared with the current NAAQS (which is not currently met by this area) were more substantial, ranging from 2.8 points at the median and 3 points at the 95th percentile (based on LLL function).

In considering estimated reductions in Pb-associated IQ loss discussed above, we observe that estimates for the 95th percentile of the population are quite similar to (for the LLL concentration-response function) or smaller (for the high- and low-end concentration-response functions) than those at the median for all case studies. This is because of the nonlinear relationship between IQ decrement and blood Pb level such that relatively smaller IQ decrement is associated with changes in blood Pb at higher blood Pb levels.

As summarized in Section 4.4, reduction in air Pb concentrations from current conditions to meet the lower alternative NAAQS (0.02 and 0.05 μg/m^3, maximum monthly mean) is estimated to reduce the number of children having Pb-related IQ loss greater than one point by one half to one percent in each of the three location-specific urban case studies. More specifically, within the three study areas this corresponds to a range of approximately 100 to 3,000 fewer children having total IQ loss greater than 1.0 for an alternative standard of 0.02 μg/m^3, maximum monthly mean. Further, just meeting the lowest alternative standard in these three study areas is estimated to reduce the number of children having an IQ loss greater than seven points by one to two percent. This corresponds to a range of approximately 350 (for the Cleveland study area) up to 8,000 (for the Chicago study area) fewer children with total Pb-related IQ loss greater than 7.0.

In summary, in staff's view (as noted above), a population IQ loss of 1-2 points may reasonably be judged to be highly significant from a public health perspective, and is judged to be so by CASAC (Section 5.4.3). Estimates of IQ loss associated with policy-relevant Pb are of a magnitude that appears to fall near or within this range for air quality scenarios involving levels at or above 0.09 μg/m^3 (maximum quarterly mean, or 0.17 maximum monthly mean). Estimated reductions in risk associated with reducing air Pb concentrations from current conditions (in the urban case studies) to the two lower alternative levels evaluated (0.02 and 0.05 μg/m^3) appear to

range from a few tenths to just below one IQ point (for the LLL concentration-response function) (and up to 1.5 IQ points for the highest concentration-response function). In considering changes in risk across the population associated with the two lower alternative levels (as compared to current conditions), we estimate reductions in the number of children with total Pb-related IQ loss greater than 1 or greater than 7 on the order of hundreds to thousands of children in the three location-specific urban case studies.

In considering the exposure and risk information with regard to a level for the standard, staff notes that at the time the standard was set, the Agency recognized a particular blood Pb level as "safe". Today, current evidence does not support the recognition of a "safe" level. This is generally reflected in the concentration-response functions used in the risk assessment and in CASAC recommendations on these functions with regard to a lack of a threshold. We therefore have considered a different approach in this review.

In considering these risk estimates, we note our conclusion and CASAC's recommendation regarding the high public health significance of a population loss of 1 to 2 IQ points, our recognition in Sections 5.4.1 and 5.4.2 of other unquantified health effects, and the significant implications of potential shifts in the distribution of IQ for the exposed population summarized in Section 5.4.2. Based on these factors and the range of estimates summarized above for IQ loss associated with policy-relevant Pb for the current conditions scenarios of the location-specific case studies, staff concludes that reducing the NAAQS to a level of 0.1 to 0.2 $\mu g/m^3$ or less would provide appreciable improvement in the protection of public health from air-related ambient Pb relative to that afforded by the current standard.

In considering standard levels below 0.1 $\mu g/m^3$, staff has considered risk as well as blood Pb reduction that might be achieved. Notable reductions in blood Pb are estimated for the lower alternative standards as compared to the current conditions scenarios. As has been recognized previously (e.g., Sections 2.1 and 3.1), ambient air Pb is one of several sources of Pb exposure to children in the U.S. Accordingly, the NAAQS is one of several regulatory tools the Agency brings to the national task of eliminating blood Pb poisoning in the U.S.[13]

In considering the public health significance of IQ loss, we have started with our conclusion that a population loss of 1-2 IQ points may reasonably be judged to be highly significant from a public health perspective. We also note that some may judge that any IQ loss at the population level is of potential public health significance. That is, there is no amount of IQ loss at the population level that is clearly recognized as being of no importance from a public health perspective. Thus, the magnitude of IQ loss that could be allowed by a standard that

[13] The President's Task Force on Environmental Health Risks to Children (Executive Order 13045, as amended) has identified childhood blood Pb poisoning as a priority public health issue in the United States.

protects public health with an adequate margin of safety is clearly a public health policy judgment to be made by the Administrator.

In considering the magnitudes of IQ loss estimated in our assessment for the range of alternative levels that we considered, we focused on total IQ loss and also on the contribution to total IQ loss from policy-relevant pathways. In so doing, we recognize that an IQ loss of 1-2 points may reasonably be judged to be highly significant from a public health perspective and we also recognize that nonair contributions to total Pb-related IQ loss are estimated to reach and exceed that amount, with air Pb contributions generally of a much smaller magnitude. Thus, we recognize that it may be appropriate to consider smaller estimates of IQ loss (e.g., less than 1 point IQ loss) in identifying the appropriate target for the policy-relevant component.

Placing weight on incremental changes in policy-relevant Pb-related IQ loss of less than one point IQ would lead to consideration of the lower standard levels evaluated in the risk assessment as part of a judgment as to what standard would protect public health with an adequate margin of safety. Staff also recognizes, however, the significant uncertainties in the quantitative risk estimates and that uncertainty in the estimates increases with increasing difference of the air quality scenarios from current conditions (See Section 4.2.7). Thus, to the extent that incremental exposure reductions achieved through lowering the NAAQS are concluded to contribute to notable incremental reductions in children's blood Pb and to associated reductions in health effects, staff suggests that consideration of NAAQS levels below 0.1 $\mu g/m^3$ (e.g., the lower levels included in the risk assessment of 0.02 and 0.05 $\mu g/m^3$) may be appropriate to consider.

Thus, if the policy goal for the Pb NAAQS was to be defined so as to provide protection that limited estimates of IQ loss from policy-relevant sources to no more than 1-2 points IQ loss at the population-level, we note that standard levels in the range of 0.1 to 0.2 $\mu g/m^3$ may achieve that goal. We also note that even with lower levels of the standard evaluated, while the range of policy-relevant IQ loss estimates is lower, the upper end of the range still extends up to and in some cases above 1 point IQ loss. We note, however, appreciably greater uncertainty associated with these estimates that increases with increasing difference of the alternative standards from current conditions (See Section 4.2.7).

Alternatively, if the policy goal was to be defined so as to provide somewhat greater public health protection by limiting the air-related component of risk to somewhat less than 1 point IQ loss at the population level, this would suggest greater consideration for standards in the lower part of the range evaluated (0.02-0.05 $\mu g/m^3$). Such a goal might reflect recognition that nonair sources, in and of themselves, are estimated to contribute 1-2 points or more of IQ loss, such that the incremental risk for policy-relevant Pb is adding to a level of total Pb exposure that is already in a range that can be reasonably judged to be highly significant from a public health

perspective. We note, however that considering standards in this lower range places greater weight on the more highly uncertain risk estimates and thus would be more precautionary in nature.

5.5.3.3 CASAC Advice and Recommendations

Beyond the evidence- and risk/exposure-based information discussed above, in our consideration of the level for the NAAQS, we have also considered the advice and recommendations of CASAC, based on their review of the CD and the earlier draft of this document and the related technical support document, as well as comments from the public on earlier drafts of this document and the related technical support document. Comments from the public that pertained to the level of the standard recommended an appreciable reduction in the level, e.g., setting it at 0.2 $\mu g/m^3$ or less.

In their advice to the Agency during this review CASAC has recognized the importance of both the health effects evidence and the exposure and risk information in selecting the level for the standard (Henderson, 2007a,b). In two separate letters, CASAC has stated that it is the unanimous judgment of the CASAC Lead Panel that the primary NAAQS should be "substantially lowered" to "a level of about 0.2 $\mu g/m^3$ or less", reflecting their view of the health effects evidence (Henderson, 2007a,b). The CASAC Lead Panel also performed some preliminary calculations to provide input to the staff in terms of the range of alternate standards appropriate to consider in carrying out the risk assessment (Henderson, 2007a, Appendix D). The CASAC calculations included an approach relating air Pb levels to blood Pb levels using the framework employed in the setting of the current NAAQS in 1978, while another related air Pb levels to blood Pb levels, and then related blood Pb to IQ loss. The results of these calculations and subsequent advice (Henderson, 2007b) led us to include a range of alternate NAAQS levels from 0.2 to 0.02 $\mu g/m^3$.

The CASAC Pb Panel also provided advice regarding how the Agency should consider IQ loss estimates derived from the risk assessment in selecting a level for the standard. The Panel stated that they consider a population loss of 1-2 IQ points to be "highly significant from a public health perspective". Further they recommended that "the primary Pb standard should be set so as to protect 99.5% of the population from exceeding that IQ loss." We anticipate further advice from CASAC with regard to level at the time of their review of the ANPR[14].

[14] As described in Section 1.2.3, EPA plans to sign an ANPR for the Pb NAAQS around the end of November 2007 for publication in the Federal Register, consistent with the new NAAQS process. A public meeting of the CASAC Pb Panel is now being planned for mid-December.

5.5.3.4 Staff Conclusions and Recommendations

Staff's consideration of alternative levels for the primary Pb standard builds on our conclusion that the overall body of evidence clearly calls into question the adequacy of the current standard with regard to health protection afforded to at-risk populations (Section 5.4.4). We believe that the available information provides strong support for consideration of a range of standard levels that are appreciably below the level of the current standard in order to provide increased public health protection for these populations. The support for this conclusion is drawn from consideration of the evidence and also from consideration of the quantitative risk and exposure information.

Consideration of the health effects evidence (Section 5.5.3.1) leads the staff to conclude that it is reasonable to consider a range for the level of the standard, for which the upper part is represented by 0.1 to 0.2 $\mu g/m^3$. We note that such levels (in terms of monthly or quarterly averaging time) are currently seen in many urban areas across the U.S. The evidence, while indicating the potential for adverse effects at or below these levels, does not provide specificity with regard to the public health implications associated with lower levels that might directly inform our consideration of the lower part of a range for the standard. This evidence-based conclusion takes into account the wealth of evidence characterizing adverse effects, particularly those to young children, at much lower blood Pb levels than were indicated by the evidence when the standard was set, the lack of evidence today for a threshold associated with the effects of greatest public health concern, and the evidence for quantitative relationships between air Pb and blood Pb that support a higher contribution to blood Pb by air Pb. This conclusion is consistent with advice and recommendations from CASAC.

Having reached this evidence-based conclusion on the upper part of the range of levels appropriate for consideration, staff then considered the exposure and risk assessment (Section 5.5.3.2), first as to what extent it provided support for the evidence-based conclusions. In this assessment, population IQ loss of a magnitude that may reasonably be judged to be highly significant from a public health perspective was estimated to be associated with policy-relevant Pb in air quality scenarios involving levels at or above 0.17 $\mu g/m^3$ Pb with a maximum monthly averaging time and form (or levels at and above 0.09 $\mu g/m^3$ with a maximum quarterly averaging time and form). Thus, the exposure and risk information supports and extends the evidence-based conclusion by indicating that important reductions in blood Pb and in Pb-associated IQ loss may be gained or maintained by reducing the level of the standard (in conjunction with a monthly averaging time) by at least an order of magnitude to approximately 0.15 $\mu g/m^3$ or lower.

Staff then considered the exposure and risk assessment with regard to the lower part of the range of alternative levels for the standard. In looking at the lower standard levels evaluated

in the quantitative risk assessment, we recognize increasing uncertainty in both the total risk attributable to Pb exposure as well as that portion attributable to policy-relevant Pb. Further, we note that the extent to which the estimates of policy-relevant risks may reasonably be judged to be important from a public health perspective becomes less clear at these lower levels. Nonetheless, consideration of the risk estimates in a similar framework to that applied when the current standard was set recognizes the importance of incremental contributions from air-related sources to total blood Pb and the associated health risks. Thus, to the extent one places weight on risk estimates for the lower standard levels, we believe these risk results may suggest consideration of a range of levels that extend down to the lowest levels assessed in the risk assessment, 0.02 to 0.05 $\mu g/m^3$.

In conclusion, staff judges that a level for the standard set in the upper part of our recommended range (0.1-0.2 $\mu g/m^3$, particularly with a monthly averaging time) is well supported by the evidence and also supported by estimates of risk associated with policy-relevant Pb that overlap with the range of IQ loss that may reasonably be judged to be highly significant from a public health perspective, and is judged to be so by CASAC. A standard set in the lower part of the range would be more precautionary in nature in that it would place weight on the more highly uncertain range of estimates from the risk assessment.

To provide some perspective on the implications of alternative primary standards (within the range of levels recommended above and within the alternate averaging times and forms focused on in Section 5.5.2), staff analyzed the 2003-2005 Pb-TSP dataset described in Section 2.3 to estimate the percentage of counties, and the populations in those counties, that likely would not attain various Pb standards. We note that given the limitations of the current monitoring network recognized in Section 2.3.2.1, the estimates of percentage of counties are likely to be underestimates. This analysis, shown in Appendix 5.A for various forms and levels of the standards, was not considered as a basis for the above staff conclusions and recommendations.

5.5.4 Summary of Staff Conclusions and Recommendations on the Primary Pb NAAQS

Staff recommendations for the Administrator's consideration in making decisions on the primary standard for Pb, together with supporting conclusions from section 5.4 and 5.5, are briefly summarized below. In making these recommendations, staff is mindful that the Act requires standards to be set that, in the Administrator's judgment, are requisite to protect public health with an adequate margin of safety, such that the standards are to be neither more nor less stringent than necessary. Thus, the Act does not require that NAAQS be set at zero-risk levels, but rather at levels that avoid unacceptable risks to public health.

(1) Staff concludes that the currently available information does not support either delisting Pb as a criteria pollutant or revoking, and not replacing, the current Pb NAAQS, a conclusion that has also been reached by CASAC. Accordingly, we recommend that Pb not be delisted as a criteria pollutant and that the Pb NAAQS not be revoked.

(2) Staff concludes that the overall body of evidence clearly calls into question the adequacy of the current standard and provides strong support for consideration of a Pb standard that would provide an appreciable increase in health protection for sensitive groups, including most notably young children, against an array of effects, most importantly including effects on the developing nervous system. We also conclude that risks estimated to remain upon meeting the current standard, based on the exposure and risk assessment, are indicative of risks to sensitive groups that can reasonably be judged to be highly significant from a public health perspective, which reinforces our conclusion that consideration should be given to revising the level of the standard so as to provide increased public health protection. Accordingly, we recommend that the Administrator consider revision of the primary NAAQS for Pb.

(3) Staff concludes that it is appropriate to continue to use Pb-TSP as the indicator to address effects associated with exposure to Pb. Based on the available information, and with consideration of the views of CASAC and public commenters, we conclude that currently available information does not provide a basis for considering an alternative indicator at this time. Staff notes interest in obtaining data concerning relationships between Pb-TSP and PM_{10} that might facilitate consideration of this issue in the next review.

(4) Staff concludes that it is appropriate to consider changing the standard to a monthly averaging time or to retaining the quarterly averaging time. In considering a form for a monthly averaging time, staff concludes that a form of maximum or second maximum would be appropriate to consider. Further, staff concludes that it is appropriate to also consider changing the duration of the time period evaluated in considering attainment to a three-year period as is common practice for NAAQS for other pollutants (from the current single-year period).

(5) Staff concludes that it is appropriate for the Administrator to consider an appreciable reduction in the level of the standard, reflecting our judgment that a standard appreciably lower than the current standard could provide an appropriate degree of public health protection and would likely result in important improvements in protecting the health of sensitive groups. We recommend that consideration be given to a range of standard levels from approximately 0.1-0.2 $\mu g/m^3$ (particularly in conjunction with a monthly averaging time) down to the lower levels included in the

exposure and risk assessment, 0.02 to 0.05 μg/m^3. In so doing, staff recognizes the substantial complexity in the assessment of exposures and risks and the increasing uncertainty in the risk estimates at these lower levels.

5.6 SUMMARY OF KEY UNCERTAINTIES AND RESEARCH RECOMMENDATIONS RELATED TO SETTING PRIMARY STANDARD

Staff believes it is important to highlight key uncertainties associated with establishing standards for Pb. Such key uncertainties and recommendations for health-related 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. Staff has identified the following key uncertainties and research questions that have been highlighted in this review of the health-based primary standards:

- A critical aspect to the risk assessment conducted for this review is the concentration-response function for the relationship between blood Pb levels in children and neurological effects, specifically IQ decrement. The functions applied in the assessment are derived from a recent analysis of pooled datasets from a number of studies (Lanphear et al., 2005). A particular area of uncertainty in our application of this analysis to our assessment is with regard to the specification of the concentration-response relationship at the lower blood Pb levels, particularly below 5 μg/dL, where the pooled analysis was quite limited with regard to number of observations. Additional epidemiological research involving substantial populations with blood Pb levels in this lower range would help to reduce uncertainty in predicting IQ loss at these lower exposure levels.
- The prediction of blood Pb levels in children and other at-risk subgroups would benefit from research in a number of areas including:
    - Temporal scale associated with changes in blood Pb levels associated with changes in ambient air Pb;
    - Interindividual variability in blood Pb levels and methods for characterizing interindividual variability, including consideration of both empirical and mechanistic methods;
    - Apportionment of blood Pb levels with regard to exposure pathway contributions, particular distinctions pertinent to policy-relevant exposures and background sources;
    - Prediction of blood Pb levels for subgroups other than young children, including adults with consideration for the full period of exposure from childhood into adulthood; and
    - Model performance evaluation, with emphasis on applications pertaining to blood Pb response to ambient air-related pathways and responses to changes in exposures for those pathways.

- An important element in the Pb NAAQS risk assessment is the characterization of the relationship between indoor dust Pb levels and levels of Pb in the ambient air, particularly with regard to the influence of changes in ambient air Pb on indoor dust Pb. Research in this topic area generally, as well as in specific environments, and also with regard to aspects associated with mechanistic modeling (e.g., air exchange rates, home cleaning frequency and efficiency) would contribute to improved models and methods for use in subsequent reviews.
- The spatial and temporal characterization of ambient air Pb levels in urban residential areas is a key element of the exposure and risk assessment completed as part of the Pb NAAQS review. Current limitations in this area contribute uncertainty to our characterization of ambient air Pb levels and associated exposures. Research in the area of characterizing spatial variation in air Pb concentrations in different environments and related to different air sources would help to reduce this uncertainty. An examples of a particular aspects of interest include the potential for systematic trends in the relationship between ambient air Pb (in terms of both spatial and temporal patterns) and the distribution of urban residential populations (e.g., are there elevated ambient air Pb levels in the vicinity of older roads due to resuspension in the vicinity of higher-density residential populations?).
- An important aspect to this review is the relationship between ambient air Pb levels and soil Pb levels, including the temporal dynamics of that relationship and variation in that for different environments. Research to improve our understanding of these areas would contribute to reducing associated uncertainty with regard to characterization of the relationship between air and soil Pb and the impact of changes in air Pb on outdoor soil Pb levels over time.

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6 ASSESSMENT OF THE SECONDARY STANDARD

6.1 INTRODUCTION

This chapter presents information in support of the review of the secondary NAAQS for lead (Pb). The presentation of welfare effects information summarizes policy-relevant aspects of the assessment of welfare effects evidence contained in the CD. Staff conclusions and recommendations on the secondary standard are based on the assessment and integrative synthesis of the welfare effects evidence presented in the CD, staff analyses and evaluations presented in Chapter 2 and in this chapter, and the comments and advice of CASAC and interested parties who commented on an early draft of this document and on the pilot phase Risk Assessment Report (USEPA, 2006; ICF, 2006).

Welfare effects addressed by the secondary NAAQS include, but are not limited to, effects on soils, water, crops, vegetation, manmade 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. The presentation in this chapter recognizes several key aspects of the welfare evidence for Pb. Lead is persistent in the environment and accumulates in soils, aquatic systems (including sediments), and some biological tissues of plants, animals and other organisms, thereby providing long-term, multipathway exposures to organisms and ecosystems. Additionally, we recognize there have been a number of widespread uses of Pb especially as an ingredient in automobile fuel by also in other products such as decorative paints, lead-acid batteries, and some pesticides which have significantly contributed to widespread increases in Pb concentrations in the environment (e.g., CD, Chapters 2 and 3).

In this chapter, we first present key policy-relevant information on the welfare effects associated with exposure to ambient Pb in section 6.2. Next in Section 6.3, we summarize the screening level ecological risk assessment conducted in support of the review the details of which are presented in the pilot phase Risk Assessment Report.[1] In Section 6.4, we assess the secondary standard, draw conclusions and present recommendations for the Administrator to consider in deciding whether the existing secondary Pb standard should be revised and if so, what revision is appropriate.

[1] As recognized in the December 2006 draft Staff Paper, a full-scale ecological risk assessment has not been performed for this review.

6.2 WELFARE EFFECTS

In this section we present a summary of the policy-relevant welfare effects evidence that is presented in detail in the CD. Key effects and concentration responses for other criteria pollutants are much more fully understood than for Pb. In the case of Pb, it is difficult to generalize effects due to the nature of the data and the general lack of community or population level information on the effects of Pb. Therefore, in this section we describe the effects of Pb on ecosystems by grouping known effects into categories of organisms and summarizing the limited available information for broader ecosystem effects of Pb.

6.2.1 Effects in Terrestrial Ecosystems

Ecosystems near smelters, mines and other industrial sources of Pb have demonstrated a wide variety of adverse effects including decreases in species diversity, loss of vegetation, changes to community composition, decreased growth of vegetation, and increased number of invasive species. Apportioning these effects between Pb and other stressors is complicated because these point sources also emit a wide variety of other heavy metals as well as SO_2 which may cause toxic effects. There are no field studies which have investigated effects of Pb additions alone but some studies near large point sources of Pb have found significantly reduced species composition and altered community structures. While these effects are significant, they are spatially limited: the majority of contamination occurs within 20 to 50 km of the emission source (CD, AX7.1.4.2).

By far, the majority of Pb found in terrestrial ecosystems was deposited in the past during the use of Pb additives in gasoline. This gasoline-derived Pb was emitted predominantly in small size particles which were widely dispersed and transported across large distances. The evidence indicates that many sites receiving Pb predominantly through such long-range transport have accumulated large amounts of Pb in soils (CD, pl AX7-98). There is little evidence that sites exposed as a result of this long range transport of Pb have experienced significant effects on ecosystem structure or function (CD, AX7.1.4.2, p. AX7-98). Strong complexation of Pb by soil organic matter may explain why few ecological effects have been observed (CD, p. AX7-98). Studies have shown decreasing levels of Pb in vegetation which seems to correlate with decreases in atmospheric deposition of Pb resulting from the removal of Pb additives to gasoline (CD, AX 7.1.4.2). Little work, however, has been done investigating the effect of residual long term, low-level metal concentration on species diversity.

As stated in the CD (Section 7.1), terrestrial ecosystems remain primarily sinks for Pb but amounts retained in various soil layers vary based on forest type, climate, and litter cycling. Once in the soil, the migration and distribution of Pb is controlled by a multitude of factors

including pH, precipitation, litter composition, and other factors which govern the rate at which Pb is bound to organic materials in the soil (CD, Section 2.3.5).

Like most metals the solubility of Pb is increased at lower pH. However, the reduction of pH may in turn decrease the solubility of dissolved organic material (DOM). Given the close association between Pb mobility and complexation with DOM, a reduced pH does not necessarily lead to increased movement of Pb through terrestrial systems and into surface waters. Studies have shown that in areas with moderately acidic soil (i.e., pH of 4.5 to 5.5) and abundant DOM, there is no appreciable increase in the movement of Pb into surface waters compared to those areas with neutral soils (i.e., pH of approximately 7.0). This appears to support the theory that the movement of Pb in soils is limited by the solubilization and transport of DOM. In sandy soils without abundant DOM, moderate acidification appears likely to increase outputs of Pb to surface waters (CD, AX 7.1.4.1).

Forest harvesting and management practices have significant and lasting effects on organic matter cycling in forest ecosystems. Clear cutting, as well as other methods of tree removal, leads to decreased organic matter for several years after harvesting and organic matter remaining in soils is exposed to higher temperatures and moisture which tend to increase rates of decomposition. Despite these effects, studies have shown very little to no mobilization of Pb from soils to surface waters following clear cutting. On possible explanation for this is that mineral soils (those below the biologically active, organic layer of soil) are efficient in capturing and retaining mobilized Pb. Loss of Pb in particulate form due to runoff and erosion in clear cut areas remains a potential source of Pb to surface waters (CD, Section 7.1.5 and associated Annex).

As described in Chapter 2 (Sections 2.6 to 2.8) and in the CD (Chapter 7 and the Chapter 7 Annex), Pb emitted anthropogenically into the atmosphere accumulates in surface soils and vegetation throughout the United States as a result of wet and dry deposition. The following discussion relies heavily on information presented in Chapters 2, 7, 8 of the CD and the Chapter 7 Annex of the CD.

6.2.1.1 Pathways of Exposure

The main pathways of exposure to Pb for animals are inhalation and ingestion. Inhalation exposures, which would be limited to areas immediately surrounding point sources, are not thought to be common and little information is available about inhalation in wildlife. Ingestion constitutes the main pathway of exposure for most organisms whether by incidental ingestion or prey contamination. For higher organisms which may ingest either contaminated plants or soils/sediments, the form and species of Pb ingested influences uptake and toxicity as

does the presence of other heavy metals. The relative toxicity of metal mixtures and their effects on Pb toxicity is complex and varies greatly by species and metal.

For plants, direct deposition onto surfaces and uptake of dissolved Pb by roots is the main exposure route (CD, Section 7.1.3). While the migration and biological uptake of Pb in ecosystems is relatively low compared to other metals, there are many factors that may affect the mobility of Pb, including elevation and climate, vegetation type, acidity, and soil composition. The bioavailability and accessibility of Pb to plants is determined largely by the soil pH, chemical form of Pb, presence of other metals, and source of the Pb in the ecosystem. Low pH soils enhance bioavailability to plants and Pb chlorides and acetates are more bioavailable than Pb oxides. These factors directly relate to the ability of Pb complexes to enter pore water in soils and sediments and thereby enter root tissues.

6.2.1.2 Effects of Lead on Energy Flow and Biogeocycling

Lead in soils and leaf litter can have a significant adverse effect on energy flow in terrestrial ecosystems through reducing the rate of litter decomposition and by decreasing photosynthetic rates in plants, both of which alter the ecosystem carbon cycling and may reduce the ability of trees and other plants to obtain nutrients from the soil (CD, AX7.1.4.3). Recent studies have associated high Pb concentrations in soils, such as those found near point sources, with reduced fungal and bacterial activity. This can lead to interruptions in various metabolic pathways by either reducing symbiotic relationships between the roots of some types of plants and fungi and/or bacteria or by tying up nutrients needed for plant growth (CD, AX7.1.4.3).

In less contaminated areas removed from point sources, there is little evidence that Pb represents a threat to energy flow or carbon cycling or that large pulses of Pb are likely to enter surface waters. Recent studies have shown that atmospheric deposition of Pb has decreased dramatically (>95%) over the last three decades and residence times in soils (the time for Pb to move out of the biologically active layers of soil) can varying greatly, for example from about 60 years in deciduous forests to 150 years in coniferous stands (CD, AX7.1.2.2).

6.2.1.3 Tools for Identifying Ecotoxicity in Terrestrial Organisms

In recognition of a need by EPA's Superfund Program to identify the potential for adverse effect from various pollutants in soils to ecosystems, a multi-stakeholder group, consisting of federal, state, private sector, and academic participants developed Ecological Soil Screening Levels (Eco-SSLs) for various pollutants including Pb. Eco-SSLs describe the concentrations of contaminants in soils that would result in little or no measurable effect on ecological receptors (USEPA, 2005a). They are intentionally conservative in order to provide confidence that contaminants that could present an unacceptable risk are not screened out early

in the evaluation process (intended to be a specific site under consideration of the Superfund Program). At or below these levels, adverse effects are considered unlikely. These values are defined in the *Ecological Soil Screening Levels for Lead* **(**USEPA, 2005a) as "concentrations of contaminants in soil that are protective of ecological receptors that commonly come into contact with soil or ingest biota that live in or on soil." They were derived separately for four general categories of ecological receptors: plants, soil invertebrates, birds, and mammals.

In the case of plants and soil invertebrates, Eco-SSLs are expressed as concentration of Pb in soil (mg Pb /kg soil) and were developed with consideration of characteristics affecting bioavailability (e.g., pH, organic content, etc). The development of Eco-SSLs for avian and mammalian wildlife involved a two step process: 1) derivation of a toxicity reference value (TRV) in mg contaminant per kg body weight per day from available literature, and 2) application of the TRV with information on soil intake, foraging habits, diet, contaminant uptake by prey for a single species to derive an Eco-SSL in mg Pb per kg soil. In general for avian and mammalian wildlife categories, a single TRV was developed (e.g., the reference dose for the most sensitive of the adverse ecological effects on birds) for all species in each category. However, default assumptions regarding incidental soil ingestion, foraging techniques, contaminant intake by prey, and overall diet composition generally resulted in different Eco-SSL values, expressed as soil concentrations, for the different species in each receptor category. The receptor category Eco-SSL was then set equal to the lowest species-specific Eco-SSL (USEPA, 2005a; ICF, 2006). The Eco-SSLs for Pb, as developed by EPA Superfund Program, for terrestrial plants, birds, mammals, and soil invertebrates are 120 mg/kg, 11 mg/kg, 56 mg/kg and 1700 mg/kg, respectively. Section 2.7 discusses current concentrations of Pb in soils. Values range from 40 to 100 mg Pb/kg soil in remote forests where historic deposition of Pb from gasoline would be presumed to be the major source to hundreds to tens of thousands of mg/kg near point sources.

By comparing known or modeled soil concentrations of Pb to the Eco-SSL value derived for each receptor group, Eco-SSL values can be used to identify locations for which further analyses are warranted to determine adverse effects from Pb. Soil screening values, including Eco-SSLs, were used in this way in the ecological screening analyses conducted for this assessment.

6.2.1.4 Effects on Plants

As discussed in Section 7.3.1 of the CD, atmospheric deposition of Pb onto vegetation is the primary route of exposure to plants from atmospheric Pb. Lead enters plant tissues primarily through direct transport, whether by surface deposition or through the soil. There is some uptake

through root cell walls via pore water but little Pb is translocated to other parts of the plant by this mechanism. Most Pb that does enter plant tissues is deposited in the roots.

Toxicity to plants occurs over a broad range of soil Pb concentrations (tens to thousands of mg/kg) due in part, to the interaction between various soil processes and the bioavailability of Pb to plants (CD, Section 7.1.4). Laboratory studies have shown great variation in toxicity to plants based on the route of exposure and the form of Pb to which the plants are exposed. Two main factors make it very difficult to determine concentration responses for plants in the field: 1) the large number of confounding factors that need to be controlled for, and 2) the lack of good field sites without multiple metal exposures. The 1986 CD (USEPA, 1986) indicated that most plants experience reduced growth when Pb concentrations in pore water exceed 2 to 10 mg/kg and when soil concentrations exceed 10,000 mg/kg under conditions of low bioavailability (e.g., high pH, oxide rather than acetate forms, etc.) Under increased bioavailability, Pb would cause reduced growth at much lower levels (e.g. <100 mg/kg). More recent studies have indeed indicated effects at much lower levels than 10,000 mg/kg in the laboratory. For example, at 2,800 mg Pb/kg dry weight of soil, adverse effects on growth were found for radish shoots when exposed to Pb chloride in mildly acidic sandy loams and at 12,000 mg/kg for shoots under similar exposures to Pb oxide (CD, Section AX7.1.4). Root cell elongation, another indicator of growth, was inhibited in ryegrass at <2.5 mg/kg Pb chloride and absence of root growth was observed at 5 mg/kg. Elevated toxicity was also found for red spruce and ryegrass when exposed to Pb under low pH conditions (CD, Section AX7.1.3.1). There is a wide breadth of studies discussed in the CD for various plants in the laboratory which indicate that Pb in concentrations found in soils near point sources could reduce plant growth. Despite this information, there are very few reports of phytotoxicity from Pb exposure under field conditions. Indeed two studies cited in Section AX7.1.3.2 of the CD found no indication of toxicity in plants exposed to high soil concentrations of Pb and other heavy metals near mining sites despite relatively high concentrations of Pb in the vegetation (4000 µg/g in Leita et al., 1989). Overall, the phytotoxicity of Pb is considered relatively low because little Pb enters plants from soil and what Pb does enter into plant tissue is deposited in roots where it is either detoxified or sequestered.

6.2.1.5 Effects on Birds and Mammals

The primary source of Pb exposure to birds and mammals is through dietary intake of both contaminated food items and incidental ingestion of soils/sediments. Direct inhalation of Pb rarely accounts for more than 10 to 15% of daily exposures and drinking water exposure is not a significant source of Pb for most organisms (CD, AX7.1.3.1).

Physiological effects from Pb exposure in birds and mammals include increased lipid peroxidation (fat breakdown) and effects on blood component production (CD, Section

AX7.1.2.5). Lipid peroxidation and fatty acid changes have been linked to changes in immune system response and bone formation. Other adverse effects may include changes in juvenile growth rates; delay of reproductive maturity; behavioral effects, such as decreased predator avoidance or lack of balance and coordination; and mortality. This cascade of effects has the potential to influence populations by reducing the number of organisms and the rate at which they are replaced, as well as altering food web composition.

Toxic effects to birds from Pb exposure have been observed over a wide range of doses in laboratory studies, usually measuring reproductive success, but little to no data are available on field populations. Studies have found few significant effects in birds below doses of 100 mg/kg in the diet and there is evidence that wide ranges of effects levels may be expected. Even in studies focused on reproductive effects in the same species, effects from doses ranging from <1 to >100 mg Pb/kg bw/day have been observed (CD, AX7.1.3.5). This variation is also true for other effects (e.g. behavioral and physiological effects) which have been observed at lower doses. As described in Section AX7.1.3.3 of the CD, no data are available on inhalation exposures of birds and very little research has been done since the 1986 CD on toxicity from Pb to birds not exposed to sediment (waterfowl).

Soil Pb concentrations and potential toxicity to birds has been considered in the development of Eco-SSLs by EPA's Office of Solid Waste and Emergency Response (USEPA, 2005b). A soil Pb concentration of 11 mg/kg dry weight of soil was derived as the Eco-SSL for birds (woodcock) (CD, Section AX7.1.4). This concentration is commonly exceeded in many areas including those not influenced by point sources (CD, Sections 3.2 and AX7.1.2.3).

Toxic effects to mammals from Pb exposure have also been observed over a wide range of doses in laboratory studies with little information available for field populations or exposures. Recent studies indicate that effects on wildlife survival would likely occur at higher doses than the 2 to 8 mg/kg-day reported in the 1986 CD. Several studies have recently reported no observed adverse effect levels (NOAELs) for survival ranging from 3.5 to as high as 3200 mg/kg-day (CD, AX7.1.3.3). No inhalation studies were found to evaluate endpoints in mammals and in those studies used to develop toxicity endpoints, organisms were dosed using either ingestion or gavage (tube feeding) which may not necessarily simulate exposure levels in the field.

A Pb Eco-SSL has been derived for mammals (shrews) at 56 mg/kg dry weight of soil based in part on toxicity reference values established for reproductive and growth effects (USEPA, 2005b). Soil concentrations exceeding 56 mg Pb/kg are not uncommon in urban/industrial locations or near major roadways and may indeed also occur in areas influenced by deposition of gasoline derived Pb without current Pb emission sources (CD, Section 3.2 and AX7.1.2.3).

Several behavioral and physiological processes seem to alter the toxicity of Pb in birds and mammals. Nutritionally deficient diets, especially those low in calcium, lead to increased uptake of Pb from the diet. Studies have also shown that younger animals and females are generally more sensitive to Pb, insectivorous animals may be more highly exposed than herbivores, and higher trophic level organism are less exposed than lower trophic level organisms.

6.2.1.6 Effects on Decomposers and Soil Invertebrates

Elevated concentrations of Pb in soils can lead to decreased decomposition rates either by direct toxicity to specific groups of decomposers, by deactivating enzymes excreted by decomposers to break down organic material or by binding with organic matter and making it resistant to the action of decomposers. Direct adverse effects to invertebrates, such as earthworms and nematodes, include decreased survival, growth and reproduction. Toxicity has been observed in soil invertebrates and microorganisms at concentrations of hundreds to thousands of mg Pb/kg soil with significant variation due to soil parameters such as pH and amount of organic matter (CD, Section AX 7.1.2).

As discussed in CD Section 7.1.4, an Eco-SSL of 1700 mg/kg dry weight of soil has been derived for soil invertebrates (USEPA, 2005). This concentration does not appear to be commonly exceeded in areas not directly influenced by point sources (CD, Sections 3.2 and AX7.1.2.3).

Several physiological mechanisms for reducing Pb toxicity have been found among invertebrates and microorganisms. These include enzyme mediated detoxification in two species of spider, Pb storage in waste nodules in earthworms and storage as an inert compound, pyromorphite, in nematodes. Avoidance of contaminated substrates and reduced feeding has also been observed in invertebrates.

6.2.1.7 Summary

Lead exists in the environment in various forms which vary widely in their ability to cause adverse effects on ecosystems and organisms. Current levels of Pb in soil also vary widely depending on the source of Pb but in all ecosystems Pb concentrations exceed what is thought to be natural background levels. The deposition of gasoline-derived Pb into forest soils has produced a legacy of slow moving Pb that remains bound to organic materials despite the removal of Pb from most fuels and the resulting dramatic reductions in overall deposition rates. For areas influenced by point sources of air Pb, concentrations of Pb in soil may exceed by many orders of magnitude the concentrations which are considered harmful to laboratory organisms. Adverse effects associated with Pb include neurological, physiological and behavioral effects

which may influence ecosystem structure and functioning. Eco-SSLs have been developed for Superfund site characterizations to indicate concentrations of Pb in soils below which no adverse effects are expected to plants, soil invertebrates, birds and mammals. Values like these may be used to identify areas in which there is the potential for adverse effects to any or all of these receptors based on current concentrations of Pb in soils.

6.2.2 Effects in Aquatic Ecosystems

Atmospheric Pb enters aquatic ecosystems primarily through the erosion and runoff of soils containing Pb and deposition (wet and dry). While overall deposition rates of atmospheric Pb have decreased dramatically since the removal of Pb additives from gasoline, Pb continues to accumulate and may be re-exposed in sediments and water bodies throughout the U.S (CD, Section 2.3.6).

Several physical and chemical factors govern the fate and bioavailability of Pb in aquatic systems. A significant portion of Pb remains bound to suspended particulate matter in the water column and eventually settles into the substrate. Species, pH, salinity, temperature, turbulence and other factors govern the bioavailability of Pb in surface waters (CD, Section 7.2.2).

6.2.2.1 Tools for Identifying Ecotoxicity in Aquatic Organisms

Ambient Water Quality Criteria (AWQC) have been developed by U.S. EPA to provide guidance to states and tribes to use in adopting water quality standards. AWQC values are available for freshwater and marine environments and for chronic and acute exposures. These values vary with water hardness and are based on the amount of dissolved Pb in the water column. They are derived from toxicity testing on aquatic organisms, including fish, invertebrates and algae and are considered to be values below which no adverse effect is anticipated (USEPA, 1993). Therefore these values are useful in identifying locations for which there is the potential for adverse effect from Pb. Section 4.4 describes how these criteria were used in the risk characterization for the ecological analyses that accompany this review.

A number of sediment ecotoxicity screening values have been developed to identify the concentration of Pb in sediment at which the potential for adverse effects occur. EPA has recently published an equilibrium partitioning method for sediment which incorporates the bioavailability of Pb and allows for mixtures of metals but may not account for ingestion of sediment by sediment dwelling organisms. There are other alternative approaches for deriving sediment criteria which are based more directly upon comparisons between concentrations of Pb in sediment and associated effects from toxicity tests. These methods do not account for bioavailability or metal mixtures but are compatible with data available from current water quality databases.

6.2.2.2 Effects in Marine/Estuarine Ecosystems

This section gives a brief overview of the information available for Pb in marine and estuarine systems. Most Pb in marine systems is in the inorganic form, complexed with chloride and carbonate ions. Increasing salinity increases the amount of Cl^- and CO_3^{2-} complexation and reduces concentration of free Pb^{2+} thereby producing compounds with lower bioavailability. There is less data available for the effects of Pb on saltwater organisms and ecosystems but studies indicate lower concentrations of Pb in oceans and large lakes. Toxicity data as expressed in both the AQWC guidelines (USEPA, 1993) and CD, AX7.2.2, indicate a much higher threshold for effects in saltwater environments.

6.2.2.2.1 Pathways of Exposure

Sources of Pb to marine and estuarine ecosystems include runoff from contaminated watersheds, direct atmospheric deposition and turnover of contaminated sediment in areas of high turbulence. Lead is primarily found in the open ocean in the dissolved form and is available in sediment in a variety of complexed forms. Lead concentrations in oceans were found to be much lower than those measured in freshwater lotic environments and studies with estuarine organisms have also shown reduced toxicity with increasing salinity, most likely due to increased complexation with Cl^- ions thereby reducing bioavailability. Studies in the Pacific Ocean near Hawaii have found concentrations of total Pb between 5-11 ng/kg (CD, Section 7.2.2).

6.2.2.2.2 Effects on Organisms and Communities

Hematological and neurological responses, including red blood cell destruction, enzyme inhibition and spinal curvature, were the most commonly reported effects in aquatic vertebrates. Demonstrated effects in invertebrates include alteration of reproduction rates and reduced growth.

Studies with marine protozoa indicate that at water column concentrations of 0.02 to 1.0 mg Pb/L, abundance, biomass and diversity are reduced. In an estuarine community, Pb was found to affect species abundance when sediment concentrations reached 1343 mg/kg dry weight. Inhibition of embryo development in commercial shellfish has been documented at water concentrations of 50µg/L (CD, AX 7.2.4.3).

The toxicity of Pb in the marine or estuarine environment is highly dependent on salinity. A study of mysid shrimp reported a lethal concentration for 50% of the test organisms (LC_{50}) of 1140 µg/L at a salinity of 5% and an LC_{50} of 4274 µg/L at 25 % salinity. There is also some evidence of gender sensitivity in that male copepods were more sensitive to Pb in sediment than females. Smaller fish have been shown to be more sensitive than larger fish of the same species. Studies on invertebrates have also shown that deposit feeders were most affected by elevated substrate concentrations.

6.2.2.3 Effects in Freshwater Ecosystems

This section gives a brief overview of information available for Pb in freshwater systems. Most Pb in freshwater systems is in the inorganic form. Speciation is important in bioavailability and is dependent upon factors such as pH, temperature and water hardness. In freshwater, Pb typically forms strong inorganic complexes with OH^- and CO_3^{2-} and weak complexes with Cl^-. Organic Pb compounds in freshwater, which may increase bioavailability, arise from both natural and anthropogenic sources. Concentrations of various forms of organic Pb complexes are largely dependent on pH and water hardness.

6.2.2.3.1 Pathways of Exposure

The bioavailability and accessibility of Pb to aquatic organisms is determined largely by the species of Pb that forms in the ecosystem. In an acidic environment (pH<4) the ionic form, which is the more toxic form, of most metals generally predominates. As pH increases, carbonate, oxide, hydroxide, and sulfide complexes usually predominate and tend to be less toxic. Water hardness also influences toxicity by providing competition in the form of calcium and magnesium to Pb binding sites on biological membranes. Therefore, Pb is least toxic in neutral to basic pH levels and at increased water hardness. A further discussion of speciation and toxicity can be found in Section AX7.2.2.1 of the CD.

The U.S. Geological Survey (USGS) has developed the National Water Quality Assessment (NAWQA) program which is a nationwide water quality monitoring program. In the NAWQA program, data have been collected on Pb concentrations in surface water, bulk sediment, and fish tissue in many watersheds throughout the U.S. While the data are not representative of the entire U.S., the NAWQA database is the most comprehensive national database available. The mean concentration of Pb in U.S. surface waters sampled in the NAWQA program between 1991 and 2003 was 0.66 µg/L (ranging from 0.04 to 30) and in bulk sediment was 120.11µg/g (ranging from 0.5 to 12,000 µg/g) (CD, AX7.2.2.2 and Section 2.2).

6.2.2.3.2 Effects at an Ecosystem Level

Aquatic ecosystems near point sources such as smelters, mines and other industrial sources of Pb have demonstrated a wide variety of effects including reduced species diversity, abundance and richness; decreased primary productivity, and alteration of nutrient cycling. Apportioning these effects between Pb and other stressors is problematic since these point sources also emit a wide variety of other heavy metals which may cause toxic effects in aquatic systems.

Lead exposure may adversely affect organisms at different levels of organization, i.e., individual organisms, populations, communities, or ecosystems. Generally, however, there is insufficient information available for single contaminants in controlled studies to permit

evaluation of specific impacts on higher levels of organization (beyond the individual organism). Potential effects at the population level or higher are, of necessity, extrapolated from individual level studies. Available population, community, or ecosystem level studies are typically conducted at sites that have been contaminated or adversely affected by multiple stressors (several chemicals alone or combined with physical or biological stressors). Therefore, the best documented links between Pb and effects on the environment are with effects on individual organisms.

However, several recent studies have attributed the presence of Pb to reduced primary productivity, increased respiration, and alterations of community structure. Specifically, dissolved Pb at concentrations from 6 to 80 mg/L (concentrations higher than those found in the NAWQA database) was found to reduce primary productivity and increase respiration in an algal community. Laboratory microcosm studies have indicated reduced species abundance and diversity in protozoan communities exposed to 0.02 to 1 mg Pb/L (CD, Section AX 7.2.5). Field studies have associated the presence or bioaccumulation of Pb with reductions in species abundance, richness, or diversity, particularly in sediment-dwelling communities (CD, Section AX7.2.5). Most of the available data for Pb effects in aquatic ecosystems comes from either laboratory studies which focused on only a few aspects of the natural system thereby neglecting some of the factors known to influence bioavailability of Pb, or from complex natural systems with many stressors and various sources of anthropogenic Pb, particularly direct mining waste inputs (CD, AX7.2.5.2). Thus, the effects of atmospheric Pb on aquatic ecological condition remain to be defined.

There is a paucity of data in the general literature that explores the effects of Pb in conjunction with all or several of the various components of ecological condition as defined by the EPA (Young and Sanzone, 2002). Recent studies have attributed the presence of Pb to adverse effects on biotic conditions such as abundance, diversity, reduced primary productivity, and alteration of community structure (CD, Section 7.2.5). It is difficult to apportion effects between Pb ands other stressors, however, and these studies did not generally account for modifying factors that may mediate or exacerbate Pb effects.

Lead concentrations in sediment vary with depth and are attributable to increased anthropogenic inputs over the last few decades. Several studies have been undertaken to identify regional sources of Pb in eastern North America and the Great Lakes and have found positive correlations between Pb isotope ratios in the Great Lakes and known aerosol emissions from current and historic industrial sources in Canada and the U.S. These studies seem to indicate that current emissions are contributing somewhat to Pb in sediments (CD, AX7.2.2.3). Resuspension of historically deposited Pb in sediments may also constitute a source of Pb in some systems for the foreseeable future (CD, AX 7.2.2.3).

6.2.2.3.3 Effects on Algae and Aquatic Plants

As primary producers in aquatic systems, algae and aquatic plants are vital to ecosystem function and provide the foundation upon which the food web depends. Therefore impacts to these organisms can create a chain of effects that impacts the entire ecosystem. Algae and aquatic plants are exposed to Pb by either uptake from the water column or sediment. Pb is most bioavailable in the divalent form (Pb^{2+}) and as such is adsorbed onto cell walls and accumulates in the cell wall or surface of the plasma membrane of aquatic plants and algae (CD, AX7.2.3.1). Bioconcentration of Pb, the accumulation of Pb inside an organism, may be quite high for both algae and aquatic plants and have made them effective in the remediation of contaminated areas. In aquatic plants as in terrestrial plants, Pb tends to be sequestered (bound and stored) in roots much more than in shoots although some wetland plants have been found to accumulate high levels of Pb in shoots as well. Within the plants the sequestered Pb tends to be metabolically unavailable until a certain concentration is reached which appears to be species specific.

Growth inhibition is exhibited by algae and aquatic plants over a broad range of Pb concentrations in water (1000 to >100,000 µg/L) due in part, to the interaction between various biochemical factors and the bioavailability of Pb to these organisms (CD, AX7.2.3.1). Clinical signs of Pb toxicity in algae include deformation and disintegration of cells, shortened exponential growth phase, and inhibition of pigment synthesis which may ultimately lead to cell death. As reported in the CD (Section AX7.2.3.1), studies have shown growth inhibition of *Closterium acerosum*, a freshwater algae, at concentrations of 1,000 µg/L Pb nitrate exposure and an effects concentration for 50% of the test population (EC_{50}) for growth inhibition of *Scenedesmus quadricauda* has been reported at 13,180µg/L. Other species of algae such as *Synechococcus aeruginosus* were much more tolerant and required concentrations in excess of 82,000 µg/L to elicit significant growth inhibition. In aquatic plants, toxicity studies have focused on the effects of Pb on plant growth, chlorophyll concentration and protein content. An EC_{50} of 1,100 µg/L was reported for growth inhibition for *Azolla pinnata*, an aquatic fern, when exposed to Pb nitrate for 4 days. Studies with duckweed, *Lemna gibba*, have reported an EC_{50} of 3,750µg/L under the same conditions. These studies indicate the possibility of adverse impacts to algae and aquatic plants at concentrations which may be found in the vicinity of direct discharges from point sources but which would not be expected from ambient deposition.

There are two main mechanisms by which algae and plants may moderate Pb toxicity: sequestration in roots or cell walls, and production of enzymes which complex Pb to make it metabolically inactive. Studies have shown phytochelatins, polypeptides which chelate heavy metal ions and make them biologically unavailable to the organism, may be synthesized in response to exposure to heavy metals (CD, AX7.1.2.4).

6.2.2.3.4 Effects on Invertebrates

Aquatic invertebrates serve an important role in aquatic ecosystems as both consumers of detrital material and as a prey source for many other organisms. Therefore, adverse impacts to invertebrates can dramatically alter or reduce ecosystem function. Invertebrates may accumulate Pb in tissue through ingestion of food and water and adsorption from water. Dietary Pb may contribute significantly to the chronic toxicity of Pb through ingestion of food which has accumulated Pb or by incidental ingestion of sediments. Studies which relate the effects of dietary exposure and toxic effects in aquatic systems are rare; however, it may be assumed that both dietary and waterborne exposures are important to overall Pb toxicity (CD, AX7.2.4.3).

Exposure to Pb can result in reduction of growth rates and reproductive rates as well as cause increased mortality. As discussed in Section 4.3.2.1 of this document, both acute and chronic toxicity of Pb can be significantly influenced by water hardness and pH. A study by Borgmann et.al (2005) with *Hyalella azteca*, a freshwater amphipod, showed a 23-fold increase in acute toxicity in soft water (18 mg $CaCO_3$/L) compared to hard water (124 mg $CaCO_3$/L). The influence of pH on Pb toxicity varies between invertebrate species. Studies have reported increasing mortality with decreasing pH in some bivalves, cladocerans, amphipods, gastropods and mayflies while some crustaceans and gastropods have shown no relationship between pH and mortality under identical conditions. For the amphipod *H. azteca*, the lowest observed effect concentration (LOEC) for survival in hard water at pH 8.27 was 192 µg/L as dissolved Pb and 466 µg/L as total Pb leading to the conclusion that both waterborne and dietary Pb contributed to this reduced survival (CD, AX7.2.4.3). Overall, adverse effects for the most sensitive invertebrates studied, amphipods and waterfleas, occurred at concentrations ranging from 0.45 to 8,000 µg/L. Exposures to Pb in sediment can also produce toxic effects in sediment dwelling invertebrates. Acute effects in the water flea, *Daphnia magna*, included reduced mobility after exposure to 7,000 mg Pb/kg dw for 48 hours while chronic exposure of midges to sediments containing 31,900 mg Pb/kg dw resulted in 100% mortality over 14 days (CD, AX7.2.4.3).

Based on recorded Pb concentrations in the NAWQA database, there are some surface waters and sediments in the U.S where effects on sensitive invertebrates would be expected but apportioning these concentrations between air and nonair sources has not been done.

There are several mechanisms by which invertebrates detoxify Pb. Lead may be concentrated in some invertebrates by formation of granules which may be eventually excreted, sequestered within the exoskeleton and glandular cells, or bound to membranes in gills and other tissues. Avoidance behaviors have been documented for the aquatic snail, *Physella columbiana*, but few studies were found that reported avoidance behaviors in invertebrates.

6.2.2.3.5 Effects on Fish and Waterfowl

Both the ingestion of contaminated sediment and prey items as well as direct absorption from water contributes to fish exposures to Pb. Dietary effects of Pb are not well studied in fish but evidence supports that higher tissue concentrations have been found in fish with direct contact with sediment. Gale et al. (2002) found a good correlation between sediment concentration and tissue concentrations in suckers and small sunfish, which feed directly from the sediment, but not in smallmouth bass, which feed at a higher trophic level. Bioconcentration does occur in freshwater fish and bioconcentration factors (BCFs) for brook trout and bluegill of 42 and 45, respectively, have been reported (CD, AX7.2.3.1). Studies have also shown that fish accumulate Pb more rapidly in low pH environments and when diets are calcium deficient.

Lead has been observed to have adverse effects on the production of some enzymes which affect locomotor function as well as adverse blood chemistry effects in some fish. Symptoms of Pb toxicity in fish include the production of excess mucous, spinal deformity, anemia, darkening of the dorsal region, degeneration of the caudal fin, destruction of spinal neurons, enzyme inhibition, growth inhibition, renal pathology, reproductive effects, and mortality (CD, AX7.2.4.3). As in other organisms, Pb speciation, water pH and water hardness play an important role in the toxicity of Pb. Spinal deformities were found to occur at much lower Pb concentrations in soft water than in hard water. Maximum acceptable threshold concentrations (MATC), the maximum concentrations at which no adverse effects were seen, have been reported (CD, AX7.2.4.3) for rainbow and brook trout in soft water as 4.1 to 7.6 µg/L Pb and 58 to 118µg/L Pb respectively. A LC_{50} of 810 µg/L was found using fathead minnows at a pH of 6-6.5 while at the same water hardness the LC_{50} was >5,400 µg/L at a pH range of 7 – 8.5. Other studies have shown alterations in blood chemistry in fish from chronic and acute exposures ranging from 100 to 10,000 µg/L Pb (CD, Section AX8.2.3.3). Therefore, given the concentrations of Pb found in surface waters in the NAWQA database, there are likely adverse effects to fish populations in some locations of the U.S. It is not clear what the ambient air contributions of Pb are at these locations.

There are several physiological and behavioral mechanisms by which fish reduce exposure and absorption of Pb. While the avoidance response to Pb in fish has not been well studied, it is known for other metals and is thought likely for Pb (CD, AX7.2.3.2). As in other organisms, gender and age are important variables in determining the adverse effects of Pb with females and young fish being more sensitive to Pb.

Incidental ingestion of contaminated sediment is the primary route of exposure for waterfowl. Studies by Beyer et al. (2000) in the Coeur d'Alene watershed near mining and smelting activity have shown a range of effects for waterfowl based on sediment concentrations and corresponding blood Pb levels. This study suggested that a NOAEL blood concentration of

0.20 mg/kg wet weight Pb corresponded to a sediment concentration of 24 mg/kg Pb. Subclinical poisoning (LOEL) occurred in swans when sediment concentration was 530 mg/kg Pb which corresponded to a 0.68 mg/kg blood Pb level. Some mortality may occur with sediment concentrations as low as 1800 mg/kg Pb and an LC_{50} was found in swans at 3,600 mg/kg Pb in sediment. While these values are somewhat site specific and are dependent on the bioavailability of the Pb as well as the overall health and diet of the animals, the correlation between blood Pb levels and effects should be applicable irrespective of location-specific variables. Given current concentrations of Pb in sediment, it is likely that some adverse effects are occurring in waterfowl exposed to point sources of Pb, whether through deposition or direct discharge.

6.2.2.4 Summary

Lead exists in the aquatic environment in various forms and under various chemical and physical parameters which determine the ability of Pb to cause adverse effects either from dissolved Pb in the water column or Pb in sediment. Current levels of Pb in water and sediment also vary widely depending on the source of Pb. Conditions exist in which adverse effects to organisms and thereby ecosystems may be anticipated given experimental results. It is unlikely that dissolved Pb in surface water constitutes a threat to ecosystems that are not directly influenced by point sources. For Pb in sediment, the evidence is less clear. It is likely that some areas with long term historical deposition of Pb to sediment from a variety of sources as well as areas influenced by point sources have the potential for adverse effects to aquatic communities. The long residence time of Pb in sediment and its ability to be resuspended by turbulence make Pb likely to be a factor for the foreseeable future. Criteria have been developed to indicate concentrations of Pb in water and sediment below which no adverse effects are expected to aquatic organisms. These values may be used to identify areas in which there is the potential for adverse effects to receptors based on current concentrations of Pb in water and sediment.

6.3 SCREENING LEVEL RISK ASSESSMENT

A screening level ecological risk assessment was performed in the pilot phase of the risk assessment for this review. The screening level assessment is described in detail in *Lead Human Exposure and Health Risk Assessments and Ecological Risk Assessment for Selected Areas, Pilot Phase* (ICF, 2006). Funding constraints have precluded performance of a full-scale ecological risk assessment. The discussion here is focused on the screening level assessment performed in the pilot phase (ICF, 2006) and takes into consideration CASAC recommendations with regard to interpretation of this assessment (Henderson, 2007a, b).

6.3.1 Overview of Analyses

The screening level risk assessment involved multiple case studies and a national-scale surface water and sediment screen. The case studies included areas surrounding a primary Pb smelter and a secondary Pb smelter, as well as a near roadway nonurban location. An additional case study for an ecologically vulnerable location was identified and described, but schedule constraints precluded risk analysis for this location (ICF, 2006).

The case study analyses were designed to estimate the potential for ecological risks associated with exposures to Pb emitted into ambient air. Soil, surface water, and/or sediment concentrations were estimated from available monitoring data or modeling analysis, and then compared to ecological screening benchmarks to assess the potential for ecological impacts from Pb that was emitted into the air (Figure 6-1). Results of these comparisons are not definitive estimates of risk, but rather serve to identify those locations at which there is the greatest likelihood for adverse effect. Similarly, the national-scale screening assessment evaluated the potential for ecological impacts associated with the atmospheric deposition of Pb at surface water and sediment monitoring locations across the United States.

Figure 6-1 illustrates the use of information and models in each phase of the analysis. Table 6-1 specifies the information types and models used for each case study, and for the national-scale screen. The reader is referred to the pilot phase Risk Assessment Report (ICF, 2006) for details on the use of this information and models in the screening assessment. As indicated in these exhibits, the specific approach for each case study differed based on the nature of the case study (e.g., type of source, land use) and the site-specific measurements available. In cases where the available measurements were not sufficient to characterize the study area (e.g., due to insufficient spatial coverage), these data were used for performance evaluation of modeling tools.

Figure 6-1. Overview of Ecological Screening Assessment.

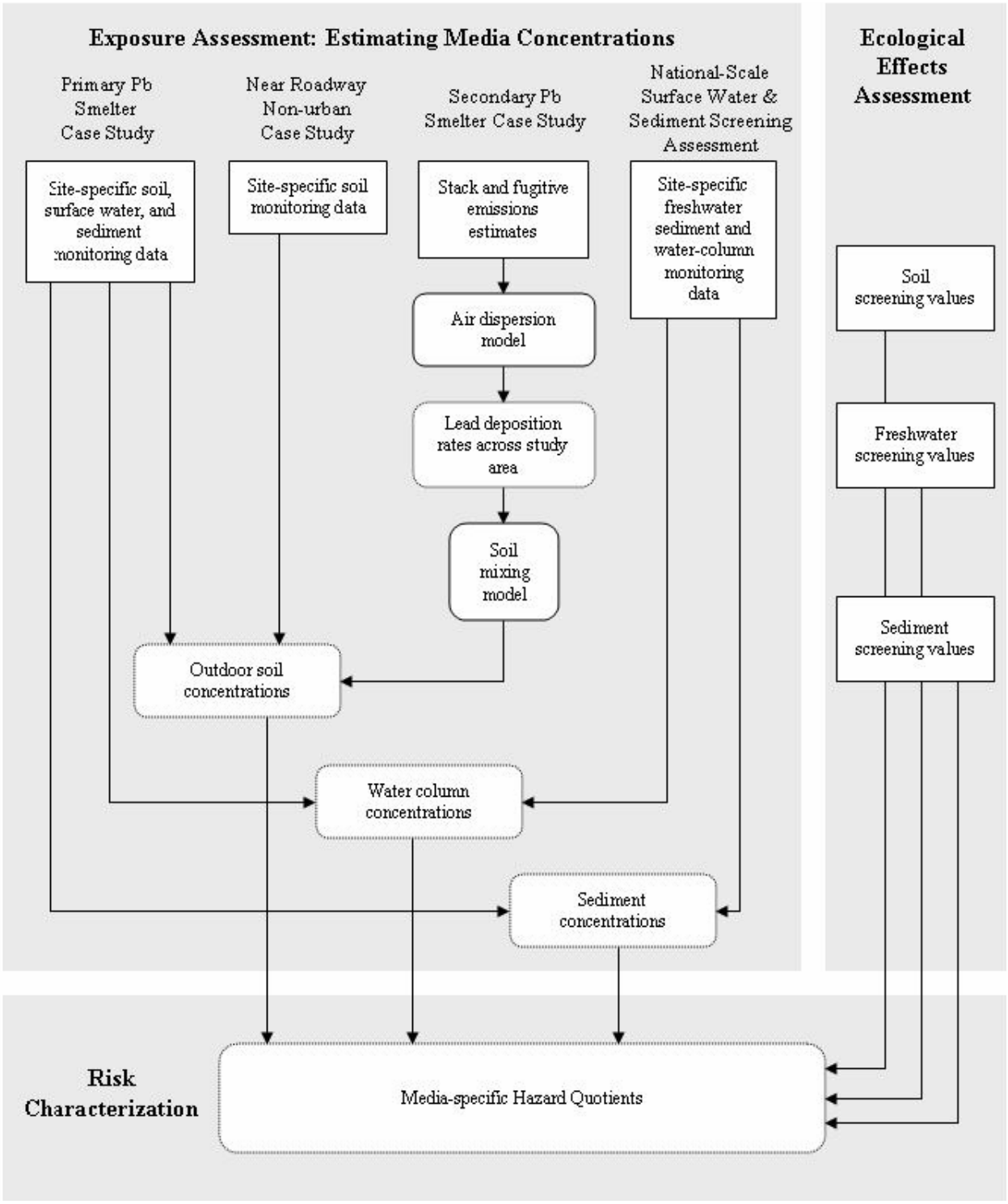

Table 6-1. Models and Measurements Used for Ecological Risk Screening Assessment.

		Primary Pb Smelter	Secondary Pb Smelter	Near Roadway Non-Urban	National-Scale Aquatic Screen
Location		Missouri	Alabama	Corpus Christi, Texas Atlee, Virginia	Surface water bodies in the U.S.
Spatial extent and resolution		Approximately 6 km diameter, centered on point source	U.S. Census blocks	Corpus Christi: single transect perpendicular to road; 0.5 - 4 m from road Atlee: 140 m section of road; 2 - 30 m from road	47 basin study units from all regions of U.S., covering approx. 50 percent of U.S. land base
Exposure Assessment: Estimating Media Concentrations					
Deposition to soil	Models	n/a	AERMOD[a]	n/a	n/a
	Measure-ments	n/a	n/a	n/a	n/a
Soil conc.	Models	n/a	MPE[b]	n/a	n/a
	Measure-ments	Site-specific conc. of total Pb in soil samples (26 locations)	n/a	Site-specific conc. of total Pb in soil samples (Corpus Christi: 2 locations; Atlee: 26 locations)	n/a
Surface water conc.	Models	n/a	n/a	n/a	n/a
	Measure-ments	Site-specific conc. of dissolved Pb in water column samples from eight water bodies/drainage areas (30 locations)	n/a	n/a	Site-specific conc. of dissolved Pb in surface water samples (430 samples)
Sediment conc.	Models	n/a	n/a	n/a	n/a
	Measure-ments	Site-specific conc. of total Pb in sediment samples from five water bodies /drainage areas (69 sites)	n/a	n/a	Site-specific or nearby water body conc. of total Pb in sediment samples (15 sites)
Ecological Risk Assessment					
Screening ecotoxicity benchmarks	Soil	Soil screening values			n/a
	Freshwater – water column	U.S. EPA Pb freshwater AWQC for aquatic life derived based on site-specific measured water hardness (conc. Of $CaCO_3$)[c]	n/a	n/a	U.S. EPA Pb freshwater AWQC for aquatic life derived based on site-specific measured water hardness (conc. of $CaCO_3$)[c]
	Freshwater – sediment	Sediment screening values based on MacDonald et al. (2000) sediment quality assessment guidelines	n/a	n/a	Sediment screening values based on MacDonald et al. (2000) sediment quality assessment guidelines

a American Meteorological Society/EPA Regulatory Model (AERMOD) (USEPA, 2004)
b Multiple Pathways of Exposure (MPE) (USEPA, 1998)
c These screening values are based on measured ecotoxicity data

6.3.2 Measures of Exposure and Effect

The measures of exposure for these analyses are total Pb concentrations in soil, dissolved Pb concentrations in fresh surface waters (water column), and total Pb concentrations in freshwater sediments. These exposure concentrations were estimated for the three case studies and the national-scale screening analyses as described below:

- For the primary Pb smelter case study, measured concentrations of total Pb in soil, dissolved Pb in surface waters, and total Pb in sediment were used to develop point estimates for sampling clusters thought to be associated with atmospheric Pb deposition, rather than Pb associated with nonair sources, such as runoff from waste storage piles.
- For the secondary Pb smelter case study, concentrations of Pb in soil were estimated using fate and transport modeling based on EPA's MPE methodology (USEPA, 1998) and data available from similar locations.
- For the near roadway nonurban case study, measured soil concentration data collected from two interstate sampling locations, one with fairly high-density development (Corpus Christi, Texas) and another with medium-density development (Atlee, Virginia), were used to develop estimates of Pb in soils for each location.
- For the national-scale surface water and sediment screening analyses, measurements of dissolved Pb concentrations in surface water and total Pb in sediment for locations across the United States were compiled from available databases. Air emissions, surface water discharge, and land use data for the areas surrounding these locations were assessed to identify locations where atmospheric Pb deposition may be expected to contribute to potential ecological impacts. The exposure assessment focused on these locations.

The Hazard Quotient (HQ) approach was used to compare estimated Pb media concentrations with ecotoxicity screening values for soils, surface waters, and sediments around the primary Pb smelter, for soils only around the secondary Pb smelter case study location and the near roadway non-urban case study locations, and for surface water and sediment in the national-scale screen. The HQ is calculated as the ratio of the media concentration to the ecotoxicity screening value. The HQ is represented by the following equation:

$$HQ = (\text{estimated Pb media concentration}) / (\text{ecotoxicity screening value})$$

For each case study, HQ values were calculated for each location where either modeled or measured media concentrations were available. Separate soil HQ values were calculated for each ecological receptor group for which an ecotoxicity screening value has been developed (i.e., birds, mammals, soil invertebrates, and plants). HQ values less than 1.0 suggest that Pb concentrations in a specific medium are unlikely to pose significant risks to ecological receptors whereas HQ values greater than 1.0 indicate that the expected exposure exceeds the ecotoxicity screening value.

6.3.3 National-Scale Screen and Case Studies

This section provides an overview of the study locations included in the ecological screening risk assessment performed in support of the NAAQS review. A national-scale screen was conducted to look at current Pb concentrations in freshwaters and sediments throughout the U.S. and three case study locations were selected: 1) near a primary Pb smelter, 2) near secondary Pb smelter and 3) a near roadway non-urban location. The primary and secondary Pb smelter case studies represent large and moderate sized point source scenarios, respectively, while the near roadway non-urban location represents a more ubiquitous exposure from historic emissions of gasoline Pb along major roadways.

6.3.3.1 National-Scale Screen

A national-scale assessment was performed using the NAWQA database to identify locations in the U.S. in which concentrations of Pb in surface water and/or sediment exceed established screening values and for which ambient air Pb is likely to be a major factor. These locations were identified using the methodology described below and in the risk assessment report (ICF, 2006).

6.3.3.1.1 Fresh Surface Waters

A screening-level ecological risk assessment for aquatic ecosystems was conducted for Pb concentrations in fresh surface waters of the United States to identify areas in which there are concentrations in excess of EPA recommended national ambient water quality criteria (AWQC), both chronic and acute, for the protection of freshwater aquatic life. In this assessment, we identified locations at which Pb concentrations exceeded the EPA AWQC and for which air sources are likely to be the major contributor to the Pb concentrations in the water (i.e., there are no other obvious sources of Pb to the water).

As the geographic coverage achieved in this surface water screen is based entirely on the geographic coverage of available measurements of dissolved Pb in the selected database, it was important that the most appropriate dataset be used. It was concluded that of the commonly available databases, including NWIS, STORET and NAWQA, the NAWQA data set is most

appropriate for a nationwide aquatic risk screen for several reasons. The inclusion of dissolved Pb as an analyte is limited in all of the databases (total Pb is measured more often). None of the databases provide the co-located measurements of water hardness in the same records as the measurements of dissolved Pb. STORET and NWIS include samples from more locations in the United States than does the NAWQA data set, but the sampling and reporting protocols represented in STORET and NWIS are less consistent from site to site. Data for dissolved Pb in NWIS are predominantly from the 1980s, and therefore do not represent current conditions. The NAWQA data set, on the other hand, provides representative (though not complete) coverage of the United States. The portion of the database containing samples through 2004 is what has been used in this assessment (USGS, 2004). The NAWQA data set also provides a consistent approach to sampling and analysis of the elements using consistent quantitation limits across the country. Given the sampling design for NAWQA and the consistency of the data across the country, it is considered to be more appropriate for a national-scale aquatic risk screen than the other two data sets and was therefore used for this screen.

6.3.3.1.2 Lead in Sediments

Possible risks to sediment dwelling organisms were also examined at locations identified in the surface water screen by comparing total Pb concentrations in sediments to ecotoxicity benchmarks for sediments, generally referred to as sediment quality criteria. The preferred approach for sediment data was to obtain them from surface water sampling locations in the NAWQA database. Sediment sampling data were not always available at the same locations as surface water samples. Therefore, some of the sites of interest do not have sediment samples available from the same location. Where an exact match was not found, a nearby sampling location was identified on the basis of latitude, and longitude, and name of the site location.

6.3.3.2 Ecologically Vulnerable Location

A literature search was conducted to identify an acidified forest or non-urban acidified watershed ecosystem to which the following criteria could be applied:

- Potential for increased bioavailability of Pb due to soil and water acidification;
- Relatively distant from point sources of Pb emissions;
- Relatively high elevation which may be subject to comparatively higher deposition of Pb due to wind speed and precipitation as well as longer residence time; and
- Availability of data on trends (temporal, elevation, etc.) of Pb concentration in various environmental media.

Based on these criteria, we selected the Hubbard Brook Experimental Forest (HBEF) in the White Mountains of New Hampshire for the ecologically vulnerable case study. The HBEF was established by the USDA Forest Service in 1955 as a major center for hydrologic research in New England. The USDA proposed to use the small watershed approach at Hubbard Brook to study linkages between hydrologic and nutrient flux and cycling in response to natural and human disturbances, such as air pollution, forest cutting, land-use changes, increases in insect populations and climatic factors. The first grant was awarded by the National Science Foundation (NSF) to Bormann and Likens in 1963 to support the research at the HBEF. Since that time there has been continuous support from the NSF and the U.S. Department of Agriculture (USDA) Forest Service. In 1988 the HBEF was designated as a Long-Term Ecological Research (LTER) site by the NSF. On-going cooperative efforts among diverse educational institutions, private institutions, government agencies, foundations and corporations have resulted in one of the most extensive and longest continuous data bases on the hydrology, biology, geology and chemistry of natural ecosystems in the United States. This historical record makes HBEF uniquely suited to the purpose of this review. Findings with regard to atmospheric deposition and Pb mobility in soil at this location are described in the CD (CD, pp. AX7-98). As discussed earlier, an assessment of Pb related ecological risks for this case study location is not presented in this document.

6.3.3.3 Primary Pb Smelter Case Study

The primary Pb smelter case study location is one of the largest primary Pb smelters in the world, is the only remaining operating Pb smelter in the United States, and is also the longest operating smelter in the world, sustaining nearly continuous operation since 1892. Further information on the surroundings and demographics in the vicinity of the primary Pb smelter can be found in the risk report (ICF, 2006). Portions of this study area comprise an active Superfund site and are subject to ongoing evaluation under the Superfund program administered by the EPA Office of Solid Waste and Emergency Response. Methods used in conducting ecological risk assessment for the analysis have been selected to address policy questions relevant to the Pb NAAQS review and consequently, may differ from those used by the Superfund program.

The primary Pb smelter property is bordered on the east by the Mississippi River, on the southeast by Joachim Creek, on the west and north-northwest by residential areas, and on the south-southwest by a slag pile. A large part of the slag pile is located in the floodplain wetlands of Joachim Creek and the Mississippi River.

Ecological features near the facility include the Mississippi River, streams, emergent and scrub-shrub wetlands, and successional and mature bottomland hardwood forest tracts (ELM, 2005). Bottomland hardwood forests and agricultural fields are present to the west, south, and

east of the characterization area between the smelter's slag storage area and Joachim Creek. The most mature bottomland hardwood forest is adjacent to Joachim Creek. Immediately south of the facility is a mixture of floodplain forest, emergent marsh, and scrub-shrub wetland habitat that is populated by willow trees. Throughout much of the year, migratory birds such as the red-tailed hawk, belted kingfisher, and great blue herons utilize the habitat near the facility. The federally threatened bald eagle has been spotted on facility property, which is known to be within the habitat for the bird. The facility is also within the habitat of the Indiana bat, which is on the federal and state endangered species lists. In addition, the state and federally endangered pallid sturgeon has been identified in the Mississippi River adjacent to and downstream of the facility. The pink mucket, scaleshell, and gray bat also occur in Jefferson County and are on both the state and federal endangered lists.

6.3.3.4 Secondary Pb Smelter Case Study

The secondary Pb smelter location falls within the Alabama Coastal Plain in Pike County, Alabama. It is located in an area of disturbed forests, and is less than 2 km from Big Creek, which is part of the Pea River watershed. Big Creek is located approximately 0.5 m south southeast from the center of the facility. The surrounding area includes emergent and scrub shrub wetlands, forests, freshwater creeks, ponds, rivers, croplands, pastureland, and developed urban areas. The Pea River watershed drains into the Gulf of Mexico. The watershed is underlain by coastal plain sediments, including sand, clay, and limestone; and the topography can be characterized as gentle to moderate rolling hills (CPYRWMA 2006). Diversity of terrestrial and aquatic animal species is relatively high. The Choctawhatee and Pea River basins, in which the secondary Pb smelter is located, contain 43 species of marine, estuarine, and freshwater fish species (Cook and Kopaska-Merkel, 1996). Anadromous fish species (i.e., saltwater fish that must spawn in freshwater) found in the Pea River basin include the following: the threatened Gulf sturgeon, Alabama shad, striped bass, and skipjack herring. The Pea River basin also provides habitat for 20 species of freshwater mussels (Cook and Kopaska-Merkel, 1996), as well as numerous species of snails, snakes, and other invertebrates. Terrestrial species supported in this region include a variety of birds, mammals, invertebrates, and vascular plants. Other terrestrial fauna found in the region include migratory birds, small mammals and invertebrate species. A total of 34 vascular flora from Pike County are listed by the Alabama Natural Heritage Inventory Program as state endangered, threatened, or of special concern (Alabama Natural Heritage Inventory 2001). According to NatureServe and the U.S. Fish and Wildlife Service (USFWS), no species in Pike County are on the federal endangered species list (Outdoor Alabama, 2003). A few species, however, are recognized as candidates for the federal list.

6.3.3.5 Near Roadway Nonurban Case Study

The Houston, Texas near roadway urban case study for the human health risk assessment used surrogate soil Pb concentration data measured at a sampling location in downtown Corpus Christi, Texas (Turer and Maynard, 2003). For the ecological screening assessment, nonurban case study locations that provide soil concentration data were sought with the expectation that ecological receptors would be more likely to occur along roads in less developed areas compared to the downtown location evaluated in the human health risk assessment. Terrestrial wildlife may forage in Pb contaminated soils alongside highways, particularly on roads traversing undeveloped areas.

From the literature search for studies of Pb in near roadway soils, two nonurban sites for which soil Pb levels are available were identified for use in the ecological risk assessment. These locations are: (1) Interstate 37 near oil refineries in Corpus Christi, Texas (Turer and Maynard, 2003) and (2) Interstate 95 in Atlee, Virginia, which connects to a moderately traveled, two-lane road (Speiran, 1998).

Land cover data from 1992 within 1 mile of the Corpus Christi, Texas study location showed 59 percent industrial, 10 percent low intensity residential, and 25 percent high intensity residential (Vogelmann et al., 2001). The remaining 5 percent of the surrounding area includes shrubland, row crops, pasture, grasses, and forested upland, including evergreen forest and deciduous forest.

The 1992 land cover data within 1 mile of the Atlee, Virginia study locations showed 26 percent developed: 2 percent low-intensity residential and commercial and 24 percent industrial and transportation. The remaining 74 percent included 25 percent deciduous forest, 14 percent woody wetlands, and 12 percent pasture (Vogelmann et al., 2001). Smaller proportions of mixed forest, evergreen forest, row crops, and transitional (barren) areas were also found.

6.3.4 Screening Values

The following is a brief summary of specific ecological screening values selected for use in the risk assessment. The main discussion of the development and derivation of these tools can be found in Section 6.2.1.3 of this document and in the risk report (ICF, 2006). This discussion summarizes the ways in which the tools were used for this assessment to identify potential for effect from Pb exposure to specific ecological receptor groups in either the case studies or the national-scale screening assessment using the NAWQA monitoring database.

6.3.4.1 Soil Screening Values

In developing soil screening values for use in this assessment, assumptions inherent in the derivation of the Superfund Eco-SSLs were examined, and as appropriate, augmented or

replaced with current species-specific information. For example, the assumptions employed for deriving the Eco-SSLs for avian and mammalian wildlife from the corresponding TRVs were examined (ICF, 2006). Soil screening values were derived for this assessment using the Eco-SSL methodology (described in Section 6.2.1.3) with the TRVs for Pb (USEPA, 2005b) and consideration of the inputs on diet composition, food intake rates, incidental soil ingestion, and contaminant uptake by prey. The soil screening values shown in Table 6-2 for plants and soil invertebrates are the Eco-SSL values (USEPA, 2005a) while the screening values for birds and mammals are based on the Eco-SSL methodology but with modified inputs specific to this assessment (see Section 7.1.3.1 and Appendix L, of ICF, 2006).

Table 6-2. Soil Screening Values for Pb for Ecological Receptors

Ecological Receptor	Soil Concentration (mg Pb/kg soil, dry weight)
Plants [1]	120
Soil Invertebrates [1]	1700
Birds [2]	38
Mammals [2]	112
[1] Values obtained from Ecological Soil Screening Levels for Lead, Interim Final (USEPA, 2005a). [2] Values obtained by refinement described in risk report (ICF, 2006).	

6.3.4.2 Surface Water Screening Values

As described in Section 7.1.3.2 of the pilot phase Risk Assessment Report (ICF, 2006, hardness-specific surface water screening values were calculated from the EPA recommended AWQC (1984). Values were derived for the primary Pb smelter case study location and the national-scale screen based on site-specific water hardness data. AWQC values for chronic exposures are called the criterion continuous concentration (CCC) and for acute exposures are called the criterion maximum concentration (CMC), and they are available for freshwater and marine environments. A CCC is generally considered to be exceeded when a 4-day average water concentration exceeds the CCC more than once every three years (USEPA, 1984).

6.3.4.3 Sediment Screening Values

This risk screen uses sediment criteria developed by MacDonald et al (2000) which focus on total Pb concentrations in sediment. These criteria for Pb include a threshold effect concentration (TEC) and a probable effect concentration (PEC) of 35.8 mg/kg dry weight and

128 mg/kg dry weight, respectively. The methodology for these sediment criteria is described more fully in section 7.1.3.3 and Appendix M of the pilot phase Risk Assessment Report (ICF, 2006).

6.3.5 Results for Case Studies and Comparison to Screening Value

To identify locations in which Pb concentrations in soil, water and/or sediment may be potentially harmful to resident biota, each case study location was assessed using either empirical data or model estimated Pb concentrations for each medium. These concentrations were then compared to screening values as described in Section 6.4.2. The HQ approach was then used to compare estimated exposures for geographic areas around the case study sites with ecotoxicity benchmark values for each of three media: soil, surface water, and sediment. HQs less than or equal to one suggest that ecological risks are negligible. HQs greater than one indicate a potential for adverse effects and a more refined analysis of sensitive receptors would be needed to more completely assess risk

6.3.5.1 National-scale Surface Water Screen

Based on EPA's reevaluation of AWQC for metals (USEPA, 1993), the CCC for relatively soft water (50 mg/L $CaCO_3$) is 1.2 µg/L, and higher for waters of greater hardness. Therefore, the initial screen of dissolved Pb concentrations in surface water looked for measurements equal to or greater than 1.2 µg/L. This resulted in 42 sampling locations for which one or more measurements exceeded that screening value. The individual dissolved Pb measurements at these stations are provided in the risk assessment report (ICF, 2006b). For a more refined risk assessment, a given location would not usually be represented with a single sampling measurement of dissolved Pb. However, for purposes of this risk screen, given the limited analyses for dissolved Pb, all 42 sampling locations were retained for analysis. Next, the location-specific CCC and CMC values were determined based on water hardness for those locations. A review of the data on water hardness in the NAWQA data set for 1994 to 2004 indicated that the initial screening value of 1.2 µg/L was too high to identify all locations for which dissolved Pb concentrations exceeded the CCC for the protection of aquatic life. Many waters in the United States are softer than anticipated (i.e., measured $CaCO_3$ concentrations down to 1 mg/L).

A second screen was therefore conducted in which dissolved Pb measurements greater than the quantitation limit (QL) but less than 1.2 µg/L were reviewed. In the second screen, for each sampling location with one or more dissolved Pb measurements above the QL but less than 1.2 µg/L co-located measurements of $CaCO_3$ were used to calculate a site-specific CCC as described above. To attempt to isolate those locations where air derived Pb is the major source

of Pb to water, land-use information was obtained from the NAWQA database for each location in which the derived HQ was greater than 1.0. The available categories of land use in the dataset separated mining sites but did not separate other activities which are likely to produce Pb (e.g. smelting sites were included in the industrial category). While some mining activities produce air emissions of Pb (see Table 2-5), the data are lacking to apportion Pb between air and nonair sources at mining sites. Therefore, results for locations with mining as the land use category were separated from the other land use types.

Table 6-3 summarizes the HQs for the 15 non-mining sites for which the chronic HQs exceed 1.0, indicating potential for adverse effect. These locations are in areas classified in the NAWQA database as urban and mixed, but also include forest, rangeland, and a "reference" site in Alaska. The HQ for the Alaska reference site is based on one measurement of dissolved Pb and one hardness measurement. Thus, the uncertainty associated with this HQ is high (ICF, 2006).

Table 6-3. Results of Aquatic Risk Screen - Locations at which Dissolved Pb Measurements Exceed AWQC, Excluding Mining Sites. [a]

Basin ID		State	Station ID	Land Use	Lead CCC (ug/L)	Pb Measurements		Hazard Quotient		
						No. > CCC / Total N	No. < QL, which is > CCC	Mean [Pb] / CCC	Max [Pb] / CCC	Max [Pb] / CMC
45	RIOG	NM	8331000	Mixed	2.9	1/12	0	**1.03**	**1.03**	0.04
44	UCOL	CO	3.85E+14	Other/Mixed	0.89	1/4	2	**1.09**	**1.09**	0.04
2	CONN	CT	1127000	Mixed	0.36	3/22	14	**1.13**	**1.31**	0.05
46	NROK	WA	12422500	Urban	0.99	4/28	24	**1.14**	**1.25**	0.05
46	NROK	WA	12422000	Urban	0.99	2/20	18	**1.17**	**1.17**	0.05
46	NROK	ID	12392155	Forest	0.17	4/17	10	**1.32**	**1.54**	0.06
47	GRSL	UT	4.05E+14	Rangeland	5.8	1/2	0	**1.45**	**1.45**	0.06
2	CONN	CT	1119375	Mixed	0.18	5/20	13	**1.68**	**2.09**	0.08
31	OZRK	MO	7018100	Forest	3.7	1/2	0	**1.89**	**1.89**	0.07
58	OAHU	HA	16212700	Mixed	0.17	1/2	1	**1.98**	**1.98**	0.08
1	NECB	RI	1112900	Mixed	0.44	3/3	0	**2.51**	**3.53**	0.14
2	CONN	CT	1124000	Mixed	0.30	11/23	9	**2.53**	**3.33**	0.13
46	NROK	ID	12419000	Mixed	0.37	2/26	16	**2.69**	**4.27**	0.17
31	OZRK	AR	7050500	Mixed	2.6	1/8	0	**3.46**	**3.46**	0.14
31	COOK	AK	6.01E+14	Reference	0.11	1/1	0	**14.91**	**14.91**	0.61

a In order of increasing Hazard Quotient for the mean CCC aquatic toxicity benchmark. Additional information characterizing these locations is provided in the risk report (ICF, 2006).

When the 15 sampling locations in Table 6-3 are compared to NEI data, only three appear to be near facilities emitting relatively large quantities of Pb to the atmosphere (i.e., more than 1 ton per year): one is in Oahu, Hawaii, one in Jewett City, Connecticut, and one in Manville, Rhode Island. An additional two sampling locations appear to be within 50 km of facilities emitting relatively large quantities of Pb, both in Connecticut; however, whether these facilities are close enough to influence the Pb concentrations in the water column at these sampling sites is unknown. Of the three sampling locations within 20 km of facilities emitting more than 1 ton of Pb per year, the location in Rhode Island might also be receiving a portion of its Pb from upstream discharges from metal ore processing facilities; there are six such discharges out of 14 National Pollutant Discharge Elimination System (NPDES) permitted facilities upstream of this sampling location. More information on emissions for these 15 locations can be found in the risk report (ICF, 2006).

In summary, the national-scale screen of surface water data identified 15 locations with water column levels of dissolved Pb that were greater than hardness adjusted chronic criteria for the protection of aquatic life (with one location having a HQ of 15), indicating a potential for adverse effect if concentrations were persistent over chronic periods; acute criteria were not exceeded at any of these locations. The extent to which air emissions of Pb have contributed to these surface water Pb concentrations is unclear.

6.3.5.2 National-scale Sediment Screen

Sediment characterization for the 15 sites identified in the AWQC screen was performed using the hazard quotient method, where measures of total Pb concentrations in sediments were compared with the sediment TEC and PEC values for the protection of sediment dwelling organisms. The first step involved attempting to find matching sediment sampling locations in the NAWQA database. It was not always possible to find co-located sediment and surface water samples. It was expected, therefore, that some of the 15 sites of interest would not have sediment samples available from the same location. Where an exact match was not found, a nearby sampling location on the same water body was identified.

Table 6-4 shows the HQs for measured total Pb concentrations in sediments at 12 of the 15 surface water locations for which data were available. The HQs are calculated by dividing sediment concentrations by the sediment screening values, which as described in Section 6.3.4.3, are the TEC and PEC for sediment dwelling organisms from the consensus-based approach to sediment quality criteria (MacDonald et al., 2000).

Table 6-4. Concentrations of Total Pb in Sediments at Locations Near or Matching the 15 Sites at which Dissolved Pb Concentrations Exceeded the AWQC, Excluding Mining Sites.

Basin ID		State	Land Use	Match	Total [Pb] (mg/kg dry sediment)	SW HQ [a]: max [Pb]/CCC	Pb Emissions (tons/year) [(b)]		No. Upstream NPDES permits for metals	Sediment Hazard Quotients	
							Fac < 20 km	Fac < 50 km		[Pb]/ TEC	[Pb]/PEC
45	RIOG	NM	Mixed	Yes	23	1.03	0.068	0.095	0	0.64	0.18
2	CONN	CT	Mixed	Near	68	1.13	6.1	7.0	0	**1.9**	0.53
46	NROK	WA	Urban	Near	47.3	1.14	0.39	0.43	0	**1.3**	0.37
46	NROK	ID	Forest	Yes	24.9	1.32	0.0	0.0	1	0.70	0.19
47	GRSL	UT	Rangeland	Yes	2900	1.45	0.0	0.36	1	**81**	**23**
31	OZRK	MO	Forest	Yes	2300	1.89	0.0	0.34	ND	**64**	**18**
58	OAHU	HA	Mixed	Yes	59	1.98	4.9	4.9	ND	**1.6**	0.46
1	NECB	RI	Mixed	Yes	240	2.51	4.1	11.7	6	**6.7**	**1.9**
2	CONN	CT	Mixed	Near	68	2.53	0.081	11.3	0	**1.9**	0.53
46	NROK	ID	Mixed	Yes	1620	2.69	0.34	0.43	4	**45**	**13**
31	OZRK	AR	Mixed	Yes	28	3.46	0.0	0.01	0	0.78	0.22
31	COOK	AK	Reference	Yes	239	14.91	0.0	0.0	0	**6.7**	**1.9**

a Data collected for corresponding surface water locations

Abbreviations:

[Pb] = total Pb concentration in sediments (mg/kg dry sediment). CCC = Criterion Continuous Concentration (or chronic AWQC). TEC = threshold effect concentration, and PEC = probable effect concentration, both from the consensus-based sediment quality criteria approach published by MacDonald et al. (2000; 2003).

Table 6-4 presents the HQs for sediments at the 9 matching and 3 nearly matching locations at which dissolved Pb concentrations in the water column exceeded the CCC (i.e., chronic AWQC) for the protection of aquatic organisms in surface waters. Nine of the TEC-based HQs exceeded 1.0, and three were less than 1.0. The three sites with HQs less than 1.0 are unlikely to pose risks to benthic aquatic communities based on the available data. Lead concentrations at these three sites were considered to be less likely to be affected by current air emissions of Pb from point sources (i.e., Pb emissions were less than 0.07 tons per year at all three locations).

Five of the PEC-based HQs exceeded 1.0, indicating probable adverse effects to sediment dwelling organisms. None of these locations were likely to be dominated, however, by current air emissions. One location in Idaho was downstream from several NPDES-permitted discharges of metals to surface waters (10th entry). Two other locations were found in Utah and Montana and it is possible that these concentrations reflect historical sediment contamination from mining operations.

Of the locations for which air emissions of Pb appear to be more likely to be contributing to ongoing Pb contamination of surface water and sediments (i.e. locations in Connecticut, Hawaii, and Rhode Island, respectively), only one, the Blackstone River in Manville, Rhode Island, is also likely to receive significant current Pb inputs from upstream NPDES-permitted sites. In addition to Pb contamination of sediment through deposition of current air emissions to surface waters, sediment at these three locations may have received Pb from atmospheric deposition in the past as well as from current and past erosion of soils containing current and historic deposits of Pb, particularly from leaded gasoline. The Quienebaug River in Connecticut (a near match between the Jewett City and Clayville locations) and the water body at Waikakalaua Street near Wahiawa, Oahu, Hawaii, had no other obvious inputs of Pb within 20 km than the air point sources. Both of those locations are in "mixed" urbanized areas, and may also have historic Pb deposition from leaded gasoline and ongoing inputs of Pb to sediments from erosion of soils contaminated by leaded gasoline. A further discussion of methodology for the sediment screen can be found in the risk assessment report (ICF, 2006).

In summary, sediment Pb concentrations at some sites are high enough that the likelihood that they would cause adverse effects to sediment dwelling organisms may be considered "probable". However, the contribution of air emissions to these concentrations is unknown.

6.3.5.3 Primary Pb Smelter Case Study

A Characterization Area Investigation (CAI) was performed at the primary Pb smelter facility by ELM Consulting in 2005. The investigation area included the smelter, slag areas, and several haul roads within a 2.1 km radius from the facility as well as two "reference areas", presumed to be outside the area of influence of the smelter, 6 to 7 km south of the facility. The area was evaluated for the potential for ecological impacts to soil, sediment, and surface water from Pb originating from the facility. Data collected as part of the CAI were used here.

To develop soil concentrations for this assessment, surface soil data were grouped into 3 geographic clusters: the west bank of Joachim Creek and two "reference areas": Crystal City and Festus Memorial Airport. Surface water and sediment samples were taken from backwater and low flow areas along Joachim Creek both upstream and downstream of the facility 800 m, 1.6 km and 3.2 km from the smelter. Additional samples were taken from the Mississippi River and a nearby pond. Details on the sampling methods used by ELM can be found in the risk assessment report (ICF, 2006).

HQs calculated for each of the sampling clusters developed for this case study are provided here: soil results are listed in Table 6-5, surface water results are presented for Table 6-6, and sediment results are presented in Table 6-7. HQs equal to or greater than 1.0 are bolded. All three of the soil sampling clusters (including the "reference areas") had HQs that exceeded

1.0 for birds. The west bank of the Joachim Creek samples had HQs greater than 1.0 for plants and mammals also. The surface water sampling clusters all had HQs less than 1.0 as results were all below the detection limit of 3.0µg/L. However, three sediment sample clusters in Joachim Creek (1, 2, and 3), the U-shaped pond, and one drainage area had HQs greater than 1.0.

Table 6-5. HQs for Soils for Primary Pb Smelter Case Study.

Location of Sample Cluster	HQ for Plants	HQ for Soil Invertebrates	HQ for Birds	HQ for Mammals
1 - West Bank of Joachim Creek	3.55	0.25	11.19	3.80
2 - Crystal City [1]	0.54	0.04	1.70	0.58
3 - Near Festus Airport [1]	0.40	0.03	1.28	0.43
1 Control samples taken outside perceived influence of the smelter.				

Table 6-6. HQs Calculated for Surface Waters for Primary Pb Smelter Case Study.

Sample Location and Cluster ID	HQ using CCC (Chronic)	HQ using CMC (Acute)
Joachim Creek		
Cluster 1	0.39	0.02
Cluster 2	0.40	0.02
Cluster 3	0.39	0.02
Cluster 4	0.41	0.02
Cluster 5	0.41	0.02
Mississippi River		
Upstream	0.54	0.02
Near Facility	0.49	0.02
Downstream	0.48	0.02
Emission Deposition		
Cluster 1	0.69	0.03
CHRDDP	0.24	0.01
RRDP-02	0.47	0.02
DAMUP	0.40	0.02

Table 6-7. HQs Calculated for Sediments in Surface Waters for Primary Pb Smelter Case Study.

Location and Cluster Sample ID	Hazard Quotient
Joachim Creek	
Cluster 1	1.0
Cluster 2	1.6
Cluster 3	2.2
Cluster 4	0.84
Cluster 5	0.96
Mississippi River	
Upstream	0.41
Near Facility	0.84
Downstream	0.34
Pond and Drainage Areas	
U-shaped Pond Cluster	4.8
ED1	3.1
ED2	0.41

In summary, the concentrations of Pb in soil and sediments exceed screening values for these media indicating potential for adverse effects to terrestrial organisms (plants, birds and mammals) and to sediment dwelling organisms. While the contribution to these Pb concentrations from air as compared to nonair sources is not quantified, air emissions from this facility are substantial (see Appendix D, USEPA 2007; ICF 2006).

6.3.5.4 Secondary Pb Smelter Case Study

For the secondary Pb smelter case study, as described in Section 6.2.3, two sets of model-predicted average Pb soil concentrations were used as exposure estimates for the pilot phase screening ecological risk assessment. The first set of concentrations was estimated using the MPE methodology (ICF, 2006). These estimated soil concentrations for the secondary Pb smelter were compared to empirical data obtained from a surrogate location. Based on this comparison, which suggested that modeled soil Pb concentrations for this case study might be significantly underestimated, we included a second characterization of soil concentrations. Specifically, measurements from a surrogate secondary Pb smelter location were used to "scale" up the modeled surface generated for this case study location to more closely match the empirical data obtained from the surrogate location (at specified distances from the facility). The

averages for 1-, 5-, or 10-km interval distances from the secondary Pb smelter facility and the associated soil HQs calculated for each interval are presented in Table 6-8.

Distance Range (m)		Modeled Soil Concentration Datasets (mg/kg)	HQ for Plants	HQ for Soil Invertebrates	HQ for Birds	HQ for Mammals
		Total Pb Soil Conc. with Background				
0	1000	86.6	0.72	0.05	**2.28**	0.77
1000	2000	20.7	0.17	0.012	0.54	0.18
2000	3000	17.3	0.14	0.010	0.46	0.15
3000	4000	16.3	0.14	0.010	0.43	0.15
4000	5000	15.8	0.13	0.009	0.42	0.14
5000	10000	15.4	0.13	0.009	0.41	0.14
10000	20000	15.1	0.13	0.009	0.40	0.13
		Scaled 3x Total Pb Soil Conc. with Background				
0	1000	260	**2.17**	0.15	**6.84**	**2.32**
1000	2000	62.3	0.52	0.037	**1.64**	0.56
2000	3000	51.8	0.43	0.030	**1.36**	0.46
3000	4000	48.9	0.41	0.029	**1.29**	0.44
4000	5000	47.3	0.39	0.028	**1.24**	0.42
5000	10000	46.2	0.39	0.027	**1.22**	0.41
10000	20000	45.4	0.38	0.027	**1.19**	0.41

[a] HQ values greater than 1.0 are highlighted in bold type.

Table 6-8. HQs Calculated for Soils for Secondary Pb Smelter Case Study.[a]

The modeled soil concentrations within 1 km of the facility showed HQs of greater than 1.0 for avian wildlife. All soil concentrations for locations greater that 1 km from the facility were associated with HQs less than 1.0 for this dataset. The three-times-higher-scaled soil concentration dataset, developed based on soil data from similar locations, resulted in avian HQs greater than 1.0 for all distance intervals evaluated, including the farthest interval modeled, 10 to 20 km from the facility. The scaled soil concentrations within 1 km of the facility also showed HQs greater than 1.0 for plants, birds, and mammals.

In summary, the estimates of Pb concentration in soils associated with the secondary Pb smelter case study were associated with HQs above 1 for plants, birds and mammals, indicating potential for adverse effect to those receptor groups.

6.3.5.5 Near Roadway Nonurban Case Study

Table 6-9 presents the HQ calculated for the Corpus Christi, Texas, near roadside soil concentration data. HQs for birds were greater than 1.0 at all but one of the distances from the road. Mammalian HQs also were greater than 1.0 at the 2 m sampling distance from the roadway. Finally, plants also had HQs ranging from 2.83 and 5.42 at the 2 m distance. However at the further distance from the roadway (4 m), birds and mammals still had HQs greater than 1.

In summary, HQs above one were concluded for plants, birds and mammals, indicating potential for adverse effect to these receptor groups.

Table 6-9. HQs Calculated for Soils Near Roadway Nonurban Case Study.

Sample location – distance from roadway	Sample depth	Total Pb concentration (mg/kg)	HQ for Plants	HQ for Soil Invertebrates	HQ for Birds	HQ for Mammals
2 m	2.5 cm	340	2.83	0.20	8.95	3.04
2 m	10 cm	650	5.42	0.38	17.1	5.80
2 m	20 cm	15	0.13	0.019	0.395	0.13
4 m	2.5 cm	140	1.17	0.082	3.68	1.25

6.3.6 Discussion

The screening-level ecological risk assessment briefly described above is described in detail in the pilot phase Risk Assessment Report and appendices (ICF, 2006). The results for the ecological screening assessment for the three case studies and the national-scale screen for surface water and for sediment indicate a potential for adverse effect from ambient Pb to multiple ecological receptor groups in terrestrial and aquatic locations. The screening assessment did not provide clear categorization of contributions from air and nonair sources although air Pb emissions are estimated to be substantial in some locations assessed. More refined analyses, which were beyond the funding limitations for the current Pb NAAQS review, would be necessary in order to more completely characterize risk to various receptors from ambient Pb, and to more completely characterize locations as to contributions from air and nonair sources of Pb.

6.3.7 Uncertainty and Variability

This section briefly summarizes uncertainties and limitations associated with the primary Pb smelter case study, the secondary Pb smelter case study, the near roadway non-urban case study and associated with the national-scale surface water and sediment screens that are

presented in the pilot phase Risk Assessment Report and appendices (ICF, 2006). Note that some limitations for the ecotoxicity screening values are described where they are introduced in Section 6.2.2.1.

Uncertainties that apply across case studies include, but are not limited to, the following:

- The ecological risk screen is limited to specific case study locations and other locations for which dissolved Pb data were available and evaluated in the national-scale surface water and sediment screens. Efforts were made to ensure that the Pb exposures assessed were attributable to airborne Pb or naturally occurring Pb and not dominated by nonair sources. However, there is uncertainty as to whether other sources might have actually contributed to the Pb exposure estimates.
- A limitation to using the selected ecotoxicity screening values is that they might not be sufficient to identify risks to some threatened or endangered species or unusually sensitive aquatic ecosystems (e.g., CD, p. AX7-110).
- The methods and database from which the surface water screening values (i.e., the AWQC for Pb) were derived is somewhat dated and new data and approaches may now be available to estimated the aquatic toxicity of Pb (CD, Section AX7.2.1.2). For example, EPA is evaluating whether pH may be a better indicator of bioavailability compared to water hardness (CD, Section AX7.2.1.3).
- No adjustments were made for sediment-specific characteristics that might affect the bioavailability of Pb in sediments in the derivation of the sediment quality criteria used for this ecological risk screen (CD, Sections 7.2.1 and AX7.2.1.4; Appendix M, ICF, 2006). Similarly, characteristics of soils for the case study locations were not evaluated for measures of bioavailability.
- Although the screening value for birds used in this analysis is based on reasonable estimates for diet composition and assimilation efficiency parameters, it was based on a conservative estimate of the relative bioavailability of Pb in soil and natural diets compared with water soluble Pb added to an experimental pellet diet. A recent site-specific determination of a soil concentration protective of birds that consume soil invertebrates suggested that the values of 38 mg/kg or even 83 mg/kg are still overly conservative. This is possibly because the assimilation efficiency of Pb in soils and natural foods compared with the assimilation efficiency of Pb acetate added to pelleted diets is much less than 50 percent (Appendix L, ICF, 2006).

6.3.7.1 Primary Pb Smelter Case Study

The ELM *Sampling and Analysis Plan* (ELM, 2003) was designed to investigate possible ecological risks from all sources of Pb (and other contaminants) attributable to the primary Pb smelter without a need to attribute the source of Pb in ecologically sensitive areas (ELM, 2003; ELM, 2005). For purposes of the Pb NAAQS review, it is important to distinguish areas impacted primarily from current or historic air deposition of Pb from areas impacted primarily from nonair sources (e.g. erosion of mining waste piles, surface runoff from exposed mining ores, direct waste discharges to water). While those areas impacted from these nonair sources are likely to be impacted from air deposition as well, availability of data and tools limited our ability to apportion Pb among these sources in these areas. Therefore, these analyses attempt to focus on those areas where it may be possible to identify effects from policy relevant sources.

The soil sampling locations within a 2.1 km radius were all in areas that might have been subject to Pb inputs from Joachim Creek during flooding events. As such, the stations might not represent the concentrations of Pb in soils that result from air emissions from the smelter. This limitation may overstate the risks from deposition of Pb emitted from the facility.

6.3.7.2 Secondary Pb Smelter Case Study

The ecological risk screen used modeled rather than measured media concentration data because measured data were not available for the case study location. Data were available for similar locations and these data were compared to the modeled results. These results appeared to vary three-fold; therefore, scaled modeled data is also reported in this assessment. A full discussion of the modeling steps and the fate and transport modeling limitations and uncertainties are described in the risk assessment report (ICF, 2006).

6.3.7.3 Near Roadway Nonurban Case Study

Few measurements were available to evaluate ecological impacts of contaminated soils near roadways in less developed areas where ecological receptors may be anticipated to occur. The measured soil data for the Corpus Christi, Texas location 2 m from the roadway ranged from 15 mg Pb/kg at 20-cm depths to 650 mg Pb/kg at 10-cm depths. The Pb concentrations selected at the Atlee, Virginia location ranged from 17 mg/kg 15 m from the roadway to 540 mg/kg 2 m from the roadway; both samples were collected from 7.5-to 15-cm soil depths. It is uncertain how representative of other roadways these data are.

The assessment did not address surface water ecosystem impacts of Pb from near roadway runoff of Pb contaminated soils. This may underestimate risks to aquatic receptors via this exposure pathway.

6.3.7.4 National-scale Surface Water Screen

The analysis revealed only two or three NAWQA water column sampling locations nationwide where there appear to be risks to the aquatic community from Pb that may have originated from atmospheric deposition. However, this is likely to be a large underestimate of the true number of such sites for several reasons:

- The NAWQA Study Units cover less than 50 percent of the land area of the United States.
- Dissolved Pb was an analyte at only 16 percent of all NAWQA sampling locations.
- Dissolved Pb was measured only once or twice at many locations.
- For waters with a hardness of less than 47 mg/L as CaCO3, the CCC for dissolved Pb is less than the quantitation limit for dissolved Pb that was used until the fall of 2000 (i.e., 1 µg/L).
- Fewer than 15 percent of samples analyzed for dissolved Pb between 1994 and 2004 were assessed with the lower quantitation limit of 0.08 µg/L, which is a value that is sufficiently low to match the CCC for waters with a hardness as low as 4.7 mg/L CaCO3.

The first two points above suggest that the number of such sites nationwide might easily be at least ten times higher than what was represented in the NAWQA database. In addition, where the land use around a sampling location was classified as "mining," no investigation was conducted to determine whether air emissions from a nearby smelter might also be contributing to the Pb in the water. Emissions information summarized in Section 2.2 indicates that such contribution is likely.

There are a variety of sources of uncertainty in the results presented for the sampling locations for which there were some data, including the following:

- Many sampling locations are represented by only one or two measurements of dissolved Pb.
- The water hardness for some sampling locations was not measured or is represented by only one or two measurements.
- Where there are multiple measures of both dissolved Pb and water hardness at a given location, no attempt was made to match sampling dates and times to develop time-specific CCC values.

- The water hardness measured at some locations was less than the lowest value of 20 mg/L of CaCO3 used to develop the equation to calculate a CCC. The CCC equation is not necessarily valid at values less than 20 mg/L CaCO3.
- It is not known how quickly dissolved Pb concentrations changed at any of the locations.
- The database supporting the current AWQC for Pb is over 20 years old; new AWQC for Pb may be available in the near future.

6.3.7.5 National-scale Sediment Screen

This analysis was limited to those 15 locations from the NAWQA database at which dissolved concentrations of Pb in surface waters exceeded the chronic AWQC for Pb. Those 15 locations are believed to represent a small fraction of surface waters in the U.S. for reasons given above. Results of this analysis cannot conclusively link any of the locations with probable adverse effects of Pb in sediments on benthic communities to ongoing air emissions of Pb. Evidence from sediment cores of historical trends, however, illustrates the influence of airborne Pb on sediments (Section 2.8).

An additional limitation is that where the land use around a sampling location was classified as “mining”, no investigation was conducted to determine whether air emissions from a nearby smelter might also be contributing to the Pb in the water and sediments. It was assumed that direct runoff and erosion from the mining sites to the surface waters would have contributed to the bulk of the Pb in sediments.

Further limitations accrue from the sediment sampling data. There were only nine exact matches and three near matches between the 15 surface water sampling locations of interest and locations at which sediment samples also were analyzed. Furthermore, there was a single sediment sample at each of the locations of interest, some of which were taken in the early 1990s.

Finally, no adjustments were made for sediment-specific characteristics that might affect the bioavailability of Pb in sediments in the derivation of the sediment quality criteria used for this risk screen.

6.4 THE SECONDARY LEAD NAAQS

6.4.1 Introduction

This section and subsections present staff conclusions and recommendations for the Administrator to consider in deciding whether the existing secondary Pb standard should be revised and, if so, what revision is appropriate. The staff conclusions and recommendations presented here are based on the assessment and integrative synthesis of information presented in the CD, staff analyses and evaluations presented in Chapter 2 and the preceding sections of this chapter, and the comments and advice of CASAC who commented on an early draft of this document and the related Risk Assessment Report.

In recommending policy options for the Administrator's consideration, we note that the final decision on retaining or revising the current secondary Pb standard is largely a public policy judgment to be made by the Administrator. The Administrator's final decision should draw upon scientific information and analyses about welfare effects, exposure and risks, as well as judgments about the appropriate response to the range of uncertainties that are inherent in the scientific evidence and analyses. In evaluating whether it is appropriate to consider retaining the current secondary Pb standard, or whether consideration of revisions is appropriate, we intend to focus on the extent to which a broader body of scientific evidence is now available that would inform such decisions.

Section 109 of the Clean Air Act requires the Administrator to establish a secondary standard that, in the judgment of the Administrator, are requisite to protect the public welfare from any known or anticipated adverse effects associated with the presence of the pollutant in the ambient air. In so doing, the Administrator seeks to establish standards that are neither more nor less stringent than necessary for this purpose. The Act does not require that secondary standards be set to eliminate all risk of adverse welfare effects, but rather at a level requisite to protect public welfare from those effects that are judged by the Administrator to be adverse.

We discuss the background for the current standard in Section 6.4.2, and the general approach used in considering the adequacy of the current standard and alternatives in Section 6.4.3. Further, staff conclusions and recommendations for the Administrator to consider in deciding whether the existing secondary Pb standard should be revised and, if so, what revised standard is appropriate is discussed in Section 6.4.4, and consideration of the elements of the standard is discussed in Section 6.4.5.

6.4.2 Background on the Current Standard

The current standard was set in 1978 to be identical to the primary standard(1.5 μg Pb/m^3, as a maximum arithmetic mean averaged over a calendar quarter), the basis for which is

summarized in section 5.2.1. At the time the standard was set, the Agency concluded that the primary air quality standard would adequately protect against known and anticipated adverse effects on public welfare, as the Agency stated that it did not have evidence that a more restrictive secondary standard was justified. In the rationale for this conclusion, the Agency stated that the available evidence cited in the 1977 CD indicated that "animals do not appear to be more susceptible to adverse effects from lead than man, nor do adverse effects in animals occur at lower levels of exposure than comparable effects in humans" (43 FR 46256). The Agency recognized that Pb may be deposited on the leaves of plants and present a hazard to grazing animals. With regard to plants, the Agency stated that Pb is absorbed but not accumulated to any great extent by plants from soil, and that although some plants may be susceptible to Pb, the Pb is generally present in a form that is largely nonavailable to them. Further the Agency stated that there was no evidence indicating that ambient levels of Pb result in significant damage to manmade materials and Pb effects on visibility and climate are minimal.

The secondary standard was subsequently considered during the 1980s in development of the 1986 CD (USEPA, 1986) and the 1990 Staff Paper (USEPA, 1990). In summarizing staff conclusions and recommendations at that time, staff stated that a qualitative assessment of available field studies and animal toxicological data suggested that "domestic animals and wildlife are as susceptible to the effects of lead as laboratory animals used to investigate human lead toxicity risks." Further, in a departure from the conclusions when the standard was set, staff highlighted concerns over potential ecosystem effects of Pb due to its persistence. In the 1990 Staff Paper, staff stated the following:

> *The available data raise concerns about the continued accumulation of lead in soil and sediment reservoirs. Due to the persistence of lead in the environment, such accumulations are expected to continue as long as inputs exceed outputs. Thus, even at relatively low deposition rates, lead could affect the ecosystem over time.*
>
> *In summary, while the available data are limited and do not provide clear quantitative relationships, they generally support the need for limiting lead emissions to protect against potential ecosystem effects. Indications are that the emission reductions achieved since promulgation of the current standards in 1978, particularly when coupled with reduction achieved by the phasedown of lead in gasoline … may have mitigated or delayed the potential risk for lead-induced ecosystem effects occurring in many areas of the country. In urban centers, along roadsides, and in the immediate vicinity of major stationary sources that have experienced a long-term historical accumulation of lead, and where the natural soil sinks for lead may be approaching or have exceeded their capacity to bind lead, the more sensitive components of the ecosystem (e.g., soil microbes) may remain at some risk that is difficult to quantify at present.*

Accordingly, the staff concluded that pending development of a stronger database that more accurately quantifies ecological effects of different lead concentrations, consideration

should be given to retaining a secondary standard at or below the level of the then-current secondary standards of 1.5 $\mu g/m^3$.

6.4.3 Approach for the Current Review

In developing conclusions and identifying options for the Pb standard in this review, staff has taken into account both evidence-based and risk-based approaches. A series of general questions frame our approach to reaching conclusions about the adequacy of the current standard and identifying options for consideration by the Administrator as to whether consideration should be given to retaining or revising the current secondary Pb standard. Examples of questions that we address in our review include the following:

- To what extent has evidence of new effects and/or sensitive organisms/ecosystems become available since the last review and to what extent are we able to characterize these effects?
- To what extent does newly available information support or call into question any of the basic elements of the current standard?
- Is there evidence of associations, especially likely causal associations, in areas that meet the current standard? What are the important uncertainties associated with that evidence?

Our approach to considering these questions recognizes that the available welfare effects evidence generally reflects laboratory-based evidence of toxicological effects on specific organisms exposed to concentrations of Pb at which scientists generally agree that adverse effects are likely to occur. It is widely recognized, however, that environmental exposures are likely to be at lower concentrations and/or accompanied by significant confounding factors (e.g., other metals, acidification), which increases our uncertainty about the likelihood and magnitude of the organism and ecosystem response.

6.4.4 Adequacy of the Current Standard

In considering the adequacy of the current standard, staff has considered the environmental effects evidence presented in detail in the CD and summarized above in Section 6.2, the screening-level risk assessment summarized above in Section 6.3, and CASAC advice and recommendations contained in their letters (Appendices A and B) and voiced during their public meetings on the first draft staff paper (February 6-7, 2007) and on the second draft lead human exposure and health risk assessment (August 28-29, 2007).

6.4.4.1 Evidence–based Considerations

In considering the welfare effects evidence with respect to the adequacy of the current standard, we consider not only the array of evidence newly assessed in the CD but also that assessed in the 1986 CD and summarized in the 1990 Staff Paper. As discussed extensively in the latter two documents, there was a significantly improved characterization of environmental effects of Pb in the ten years after the Pb NAAQS was set. And, in the subsequent nearly 20 years, many additional studies on Pb effects in the environment are now available. These studies are described in detail in the CD, and summarized in Section 6.2. Some of the more significant aspects of the evidence available since the standard was set that are relevant to our consideration of the adequacy of the current standard include the following.

- A more quantitative determination of the mobility, distribution, uptake, speciation, and fluxes of atmospherically delivered Pb in terrestrial ecosystems. These studies show that the binding of Pb to organic materials in the soil slows its mobility through soil and may prevent uptake by plants (CD, Sections 7.1.2, 7.1.5, AX7.1.4.1, AX7.1.4.2, AX7.1.4.3 and AX7.1.2). Therefore, while atmospheric deposition of Pb has decreased, Pb may be more persistent in some ecosystems than others and may remain in the active zone of the soil, where exposure may occur, for decades (CD, Sections 7.1.2, AX7.1.2 and AX7.1.4.3).
- Plant toxicity may occur at lower levels than previously identified as determined by data considered in development of Eco-SSLs (CD, pp. 7-11 to 7-12, AX7-16 and Section AX7.1.3.2), although the range of reported soil Pb effect levels is large (tens to thousands of mg/kg soil).
- Avian and mammalian toxicity may occur at lower levels than those previously identified (e.g., in the 1986 Lead CD) based on data considered in the development of Eco-SSLs, although the range of Pb effect levels is large (<1 to >1,000 mg Pb/kg bw-day) (CD, p. 7-12, Section AX7.1.3.3).
- An expanded understanding of the fate and effects of Pb in aquatic ecosystems and of the distribution and concentrations of Pb in surface waters throughout the United States (CD, Section AX7.2.2).
- The availability of new methods for assessing the toxicity of metals to water column and sediment-dwelling organisms and data collection efforts, such as the USGS NAWQA program, that monitor Pb in many U.S. aquatic ecosystems (CD, Sections 7.2.1, 7.2.2, AX7.2.2, and AX7.2.2.2). Findings indicate that in some estuarine

systems Pb deposited during historic usage of leaded gasoline may remain in surface sediments for decades (CD, p. 7-23).

- A larger dataset of aquatic species assessed with regard to Pb toxicity, and findings of lower effect levels for previously untested species (CD, p. AX7-176 and Section AX7.2.4.3).
- Currently available studies have also shown effects on community structure, function and primary productivity, although some confounders have not been well addressed (CD, Section AX7.1.4.2).
- A consideration of ecosystem critical loads for Pb. Such analysis had not been previously considered in review of the Pb NAAQS. Current information on Pb critical loads is lacking for many processes and interactions involving Pb in the environment and work is ongoing (CD, Section 7.3).

Given the full body of current evidence, it may be concluded that despite wide variations in Pb concentrations in soils throughout the country, Pb concentrations are in excess of concentrations expected from geologic or other non-anthropogenic forces. In particular, the deposition of gasoline-derived Pb into forest soils has produced a legacy of slow moving Pb that remains bound to organic materials despite the removal of Pb from most fuels and the resulting dramatic reductions in overall deposition rates (CD, Section AX7.1.4.3). We note that in areas influenced by point sources of air Pb that meet the current standard, concentrations of Pb in soil may exceed by orders of magnitude the concentrations which are considered harmful to laboratory organisms (CD, Sections 3.2 and AX7.1.2.3).

We recognize, however, that major difficulties arise in attempting to quantify the role of current ambient Pb in the environment. For example, some Pb deposited before the standard was enacted is still present in soils and sediments and historic Pb from gasoline continues to move slowly through systems as does current Pb derived from both air and nonair sources. Additionally, the evidence of adversity in natural systems is very sparse due in no small part to the difficulty in determining the effects of confounding factors such as multiple metals or factors influencing bioavailability in field studies. Nonetheless, the evidence summarized above and described in detail in the CD leads us to the following conclusions based on Pb in the environment today and evidence of environmental Pb exposures of potential concern.

Conditions exist in which adverse effects to aquatic organisms and thereby ecosystems may be anticipated given experimental results. While the evidence does not indicate that dissolved Pb in surface water constitutes a threat to those ecosystems that are not directly influenced by point sources, the evidence regarding Pb in sediment is less clear (CD, Sections

AX7.2.2.2.2 and AX7.2.4). It is likely that some areas with long term historical deposition of Pb to sediment from a variety of sources as well as areas influenced by point sources have the potential for adverse effects to aquatic communities. The long residence time of Pb in sediment and its ability to be resuspended by turbulence make Pb likely to be a factor for the foreseeable future. Accordingly, staff concludes that the evidence suggests the occurrence of environmental levels of Pb allowed by the current standard, set nearly thirty years ago, that may pose risk of adverse environmental effect.

6.4.4.2 Risk-based Considerations

In addition to the evidence-based considerations discussed above, staff has also considered the findings of the screening level ecological risk assessment taking into account key limitations and uncertainties associated with the analyses.

The screening level risk assessment involved a comparison of estimates of environmental media concentrations of Pb to ecological screening levels to assess the potential for ecological impacts from Pb that was emitted into the air. Results of these comparisons are not considered to be definite predictors of risk, but rather serve to identify those locations at which there is greatest likelihood for adverse effect. Similarly, the national-scale screening assessment evaluated the potential for ecological impacts associated with the atmospheric deposition of Pb released into ambient air at surface water and sediment monitoring locations across the United States. However, as noted above, funding constraints have precluded conducting a full-scale ecological risk assessment for this review.

The ecological screening levels employed in the screening level risk assessment for different media are drawn from different sources, as summarized above and described in more detail in Section 7.1.3 of the pilot phase Risk Assessment Report. Consequently there are somewhat different limitations and uncertainties associated with each. In general, their use here recognizes their strength in identifying media concentrations with the potential for adverse effect and their relative nonspecificity regarding the magnitude of risk of adverse effect.

As discussed in the previous section, as a result of its persistence, Pb emitted in the past remains today in aquatic and terrestrial ecosystems of the Unites States. Consideration of the environmental risks associated with the current standard is complicated by the environmental burden associated with air Pb concentrations, predominantly in the past, that exceeded the current standard. For example, the primary Pb smelter case study involves a facility that has been emitting Pb for many decades, and as for much of that time there was no Pb NAAQS, air concentrations associated with the facility may have been well above the current NAAQS.

Concentrations of Pb in soil and sediments associated with the primary Pb smelter case study exceeded screening values for those media indicating potential for adverse effect to

terrestrial organisms (plants, birds and mammals) and to sediment dwelling organisms. While the contribution to these Pb concentrations from air as compared to nonair sources has not been quantified, air emissions from this facility are substantial (see Appendix D, USEPA 2007; ICF 2006). Additionally, estimates of Pb concentration in soils associated with the secondary Pb smelter case study were also associated with HQs above 1 for plants, birds and mammals, indicating potential for adverse effect to those receptor groups. The industrial facility in this case study is much younger than the primary Pb smelter and apparently became active less than ten years prior to the establishment of the current standard.

The national-scale screens, which are not focused on particular point source locations, indicate the ubiquitous nature of Pb in aquatic systems of the U.S. today. Further the magnitude of Pb concentrations in several aquatic systems, nationally, exceeded screening values In the case of the national-scale screen of surface water data, 15 locations were identified with water column levels of dissolved Pb that were greater than hardness adjusted chronic criteria for the protection of aquatic life (with one location having a HQ as high as 15), indicating a potential for adverse effect if concentrations were persistent over chronic periods Further, sediment Pb concentrations at some sites in the national-scale screen were high enough that the likelihood that they would cause adverse effects to sediment dwelling organisms may be considered "probable".

A complicating factor in interpreting the findings for the national-scale screening assessments is the lack of clear apportionment of Pb contributions from air as compared to nonair sources, such as industrial and municipal discharges. While the contribution of air emissions to the elevated concentrations has not been quantified, documentation of historical trends in the sediments of many water bodies has illustrated the sizeable contribution that airborne Pb can have on aquatic systems. This documentation also indicates the greatly reduced contribution in many systems as compared to decades ago (presumably reflecting the banning of Pb-additives from gasoline used by cars and trucks). We note, however, that the timeframe for removal of Pb from surface sediments into deeper sediment varies across systems, such that Pb remains available to biological organisms in some systems for much longer than in others (Section 2.8; CD, pp.AX7-141 to AZX7-145).

The case study locations included in the screening assessment, with the exception of the primary Pb smelter site, are currently meeting the current Pb standard, yet Pb occurs in some locations, particularly in soil and aquatic sediment, at concentrations above the screening levels, indicative of a potential for harm to some terrestrial and sediment dwelling organisms. While the role of airborne Pb in determining these Pb concentrations is unclear, the historical evidence indicates that airborne Pb can create such concentrations in sediments. Further, whether such concentrations may be related to emissions prior to establishment of the current standard is also

unclear. The staff suggests that such concentrations appear to indicate a potential for harm to ecological receptors under the current standard.

6.4.4.3 CASAC Advice and Recommendations

In the CASAC letter transmitting advice and recommendations pertaining to the review of the First Draft Pb Staff Paper and Draft Pb Exposure and Risk Assessments, the CASAC Pb panel provided recommendations regarding the need for a Pb NAAQS, and the adequacy of the current Pb NAAQS, as well as comments on the draft documents. With regard to the need for a Pb NAAQS and adequacy of the current NAAQS, the CASAC letter said:

> *The unanimous judgment of the Lead Panel is that lead should not be delisted as a criteria pollutant, as defined by the Clean Air Act, for which primary (public health based) and secondary (public welfare based) NAAQS are established, and that both the primary and secondary NAAQS should be substantially lowered.*

Specifically with regard to the secondary NAAQS, the CASAC Pb Panel stated that the December 2006 draft documents presented "compelling scientific evidence that current atmospheric lead concentrations and deposition – combined with a large reservoir of historically deposited lead in soils, sediments and surface waters – continue to cause adverse environmental effects in aquatic and/or terrestrial ecosystems, especially in the vicinity of large emissions sources." The Panel went on to state that "These effects persist in some cases at locations where current airborne lead concentrations are below the level of the current primary and secondary lead standards" and "Thus, from an environmental perspective, there are convincing reasons to both retain lead as a regulated criteria air pollutant and to lower the level of the current secondary standard."

In making this recommendation, the CASAC Pb Panel cites the persistence of Pb in the environment, the possibility of some of the large amount of historically deposited lead becoming resuspended by natural events, and the expectation that humans are not uniquely sensitive among the many animal and plant species in the environment. In summary, with regard to the recommended level of a revised secondary standard, the CASAC panel stated that:

> *Therefore, at a minimum, the level of the secondary Lead NAAQS should be at least as low as the lowest-recommended primary lead standard.*

The March 2007 CASAC letter also encouraged the Agency to "identify the necessary funds to support needed continuing research on the ecological effects of airborne lead pollution and to consider developing alternative secondary standards such as critical loads for lead, which may be different from primary standards in indicator, averaging time, level, or form."

CASAC provided further advice and recommendations on the Agency's consideration of the secondary standard in this review in their letter of September 2007 (Henderson, 2007b). In

that letter, they restated their recommendation from the March letter that "at a minimum, the secondary Lead NAAQS should be at least as low as the lowest-recommended primary lead standard". They also recognized the role of the secondary standard in influencing the long-term environmental burden of Pb and a need for environmental monitoring to assess the success of the standard in this role:

> *The large environmental burden of historically-deposited lead is currently decreasing. Accordingly, the goal should be to set the secondary Lead NAAQS such that there is no reversal of the current downward trend in lead concentrations in the environment. The limited funds available for monitoring environmental lead should be focused on this critical task.*

6.4.4.4 Staff Conclusions and Recommendations

In considering the adequacy of the current standard, staff first considered, for reasons discussed in Section 5.4, whether it is appropriate to maintain a NAAQS for Pb or to retain Pb on the list of criteria pollutants. Given the persistence of Pb, continued emissions of Pb by many and varied sources (Section 2.2), and the harmful environmental effects associated with Pb (Section 6.2; CD, Chapter 7 and Appendix AX7), staff concurs with CASAC that Pb should not be delisted as a criteria pollutant and the secondary standard should not be revoked.

In addressing whether, in view of the information now available, the secondary standard should be revised to provide requisite protection from Pb-related adverse effects on public welfare, staff has considered evidence described in the CD and summarized above in Section 6.2 and Chapter 2 as well as information gained from the screening-level risk analyses summarized above in Section 6.3.

In summary, environmental Pb levels that exist today reflect atmospheric Pb concentrations and associated deposition, in combination with a large reservoir of historically deposited Pb in environmental media. As discussed above in Section 6.4.4.1, the information presented in detail in the CD and summarized in Section 6.2 indicates that there is evidence, largely qualitative, that suggests the potential for adverse environmental impacts under the current standard. Given the limited data on Pb effects in ecosystems, it is necessary to look at evidence of Pb effects on organisms and extrapolate to ecosystem effects. Therefore, by looking to laboratory studies and current media concentrations in a wide range of areas, it seems likely that adverse effects are occurring, particularly near point sources, under the current standard. While the role of current airborne emissions is difficult to apportion, it is conclusive that deposition of Pb from air sources is occurring and that this ambient Pb is likely as persistent in the environment as historically deposited Pb has been, although location-specific dynamics of Pb in soil lead to differences among locations as to the timeframe for Pb to be retained in surface

soils or sediments where it may be available to ecological receptors (see Section 2.7 and 2.8 and USEPA, 2007, section 2.3.3). In addition, as noted by CASAC in alluding to the low levels of human exposure currently associated with adverse health effects, it is reasonable to expect that many animals and plant species in the environment may have sensitivities to Pb that are similar to those in humans. The screening-level risk information (Section 6.4.4.2), while limited and accompanied by various uncertainties, also suggests occurrences of environmental Pb concentrations existing under the current standard that suggest the potential for adverse environmental effects.

For the reasons discussed above, staff concurs with CASAC with regard to consideration of Pb in the environment today and the adequacy of the current standard. Accordingly, we suggest that there is a need for the Administrator to consider a revision of the current standard to provide increased protection against reasonably anticipated adverse environmental effects.

6.4.5 Elements of the Standard

The four elements of the standard– indicator, averaging time, form and level serve to define the standard and must be considered collectively in evaluating the welfare protection afforded by the standards. In the previous section, staff suggested that the Administrator consider a revision to the current standard. In considering a revision to the current standard, we consider the four elements of the standard, the information available, and advice and recommendations from CASAC, regarding how the elements might be revised to provide a secondary standard for Pb that protects against adverse environmental effect.

With regard to the pollutant indicator for use in a secondary NAAQS that provides protection for public welfare from exposure to Pb, the staff makes note of the evidence that Pb is a persistent pollutant to which ecological receptors are exposed via multipathways. While the evidence indicates that the environmental mobility and ecological toxicity of Pb are affected by various characteristics of its chemical form, and the media in which it occurs, information is insufficient to identify an indicator other than Pb-TSP that would provide protection against adverse environmental effect in all ecosystems nationally. Accordingly, staff concludes that Pb-TSP should be retained as the pollutant indicator for use in a secondary NAAQS.

With regard to averaging time, the staff recognizes that the evidence demonstrates that Pb is a cumulative pollutant with environmental effects that can last many decades. In considering the appropriate averaging time for such a pollutant the concept of critical loads may be useful (CD, Section 7.3). However, information regarding critical loads is currently insufficient for such use in this review. We conclude that there is not a basis at this time for recommending any revision to the current averaging time.

With regard to level, staff notes that there is a general lack of data that would indicate a level of Pb in environmental media below which Pb-related adverse ecosystem effects would not occur. In addition, the cumulative and persistent nature of Pb requires protection over long periods of time even from accumulations resulting from small amounts of deposition. Staff also notes the influence of airborne Pb on Pb in aquatic systems, as demonstrated by historical patterns in sediment cores from lakes and Pb measurements (Section 2.8.1; CD, Section AX7.2.2; Yohn et al., 2004; Boyle et al., 2005). In its advice to the Administrator, the CASAC Pb panel indicated that a significant change to current air concentrations (e.g., via a significant change to the standard) is likely to have significant beneficial effects on the magnitude of lead exposures in the environment and lead toxicity impacts on natural and managed terrestrial and aquatic ecosystems in various regions of the U.S., the Great Lakes and also U.S. territorial waters of the Atlantic Ocean (Henderson, 2007a, Appendix E).

Staff concurs with CASAC's conclusion that the Agency lacks the relevant data to provide a clear, quantitative basis for setting a secondary Pb NAAQS that differs from the primary standard in indicator, averaging time, form, or level. CASAC further advised that:

> *To collect such data for the next Lead NAAQS review cycle, the EPA needs to initiate new measurement activities in rural areas —including those that are remote, close to urban and other sources, and located at high elevations — which quantify and track changes in lead concentrations in the ambient air, soils, deposition, surface waters, sediments and biota, along with other information as may be needed to calculate and apply a critical loads approach for assessing environmental lead exposures and risks in the next review cycle.*

The CASAC Pb Panel also recommended that the Agency "identify the necessary funds to support needed continuing research on the ecological effects of airborne lead pollution and to consider developing alternative secondary standards such as critical loads for lead, which may be different from primary standards in indicator, averaging time, level or form."

In conclusion, staff concludes that in the absence of information on which to base independent recommendations for the secondary standard, a reduction in the level of the secondary standard consistent with a reduction in the level of the primary standard (Section 5.5.4) would provide increased protection against adverse environmental effect. Accordingly, staff recommends that the Administrator consider a revised secondary standard set identical to the indicator, averaging time, form, and level of a revised primary standard.

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ATTACHMENT A

Clean Air Scientific Advisory Committee Letter

(March 27, 2007)

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON D.C. 20460

OFFICE OF THE ADMINISTRATOR
SCIENCE ADVISORY BOARD

March 27, 2007

EPA-CASAC-07-003

Honorable Stephen L. Johnson
Administrator
U.S. Environmental Protection Agency
1200 Pennsylvania Avenue, NW
Washington, DC 20460

Subject: Clean Air Scientific Advisory Committee's (CASAC) Review of the 1st Draft Lead Staff Paper and Draft Lead Exposure and Risk Assessments

Dear Administrator Johnson:

The Clean Air Scientific Advisory Committee (CASAC or Committee), augmented by subject-matter-expert Panelists — collectively referred to as the CASAC Lead Review Panel (Lead Panel) — completed its review of the Agency's 1st Draft Lead Air Quality Criteria Document (AQCD) in September 2006 (EPA-CASAC-06-010). On December 7, 2006, Mr. Marcus Peacock, the EPA Deputy Administrator, issued a memorandum providing his final decisions on revisions to the process by which the National Ambient Air Quality Standards (NAAQS) are reviewed. In this memo, Deputy Administrator Peacock directed that this revised NAAQS review process should begin with the current, ongoing review of the NAAQS for lead. (See URLs: http://www.epa.gov/ttnnaaqs/memo_process_for_reviewing_naaqs.pdf and http://www.epa.gov/ttnnaaqs/naaqs_process_report_march2006_attachments.pdf).

On February 6–7, 2007, the CASAC's Lead Panel conducted a peer review of EPA's *Draft Review of the National Ambient Air Quality Standards for Lead: Policy Assessment of Scientific and Technical Information* (1st Draft Lead Staff Paper, December 2006) and a related draft technical support document, *Lead Human Exposure and Health Risk Assessments and Ecological Risk Assessment for Selected Areas: Pilot Phase, Draft Technical Report* (Draft Lead Exposure and Risk Assessments, December 2006). In addition, on March 9, 2007, the Lead Panel held a public teleconference to review the CASAC's draft letter to the Administrator resulting from its February meeting. The CASAC roster is found in Appendix A of this report, and the Lead Panel roster is attached as Appendix B. The charge questions provided to the Lead Panel by EPA staff are contained in Appendix C to this report, and examples of population-based approaches to lead risk assessments for the primary Lead NAAQS are found in Appendix D. A discussion of issues related to setting of the secondary Lead NAAQS is attached as Appendix E, and Panelists' individual review comments are provided in Appendix F.

At the February 6–7 public meeting, the Lead Panel expressed serious concerns both about the EPA documents to be reviewed and the Agency's proposed rulemaking schedule for the Lead NAAQS, as follows:

- 1st Draft Lead Staff Paper had no staff-derived options for keeping or altering the current Lead NAAQS.
- The Draft Lead Exposure and Risk Assessments document did not have a full discussion of the risk associated with different options for keeping or altering the Lead NAAQS. The Lead Panel judges that, while the latter document represented a good first effort, it was nowhere near completion.
- Under the Agency's new NAAQS review process, EPA's Staff Paper for lead will no longer be prepared but will be replaced by a Policy Assessment (PA) for lead, to be issued in the form of an Advance Notice of Proposed rulemaking (ANPR). However, the Agency's proposed schedule for the Lead NAAQS review calls for completion of the Lead Exposure and Risk Assessments document *after* the PA for lead is issued via the ANPR. Thus, it was not planned for the CASAC to be given an opportunity to review a more fully-developed, second-draft version of the Risk/Exposure Assessment (RA) prior to the ANPR, so that the PA would not be informed by the science assessments of the Lead Panel.

Subsequent to the February 6–7 meeting of the CASAC Lead Review Panel, Agency officials, managers and staff held administrative discussions with the chartered members of the CASAC to learn directly from these seven members their specific concerns with the schedule for review of the lead standards and the revised NAAQS review process in general. The Lead Panel is pleased to have been briefed by Agency staff during the Panel's March 9 teleconference that EPA has modified its timeline both for the generic NAAQS review process and the current Lead NAAQS review in particular, such that the Lead Panel will now review the 2nd draft of the Agency's Lead Risk/Exposure Assessment this summer, prior to the issuance of the associated PA document in the ANPR.

The CASAC Lead Review Panel used the scientific information found in the Agency's Final Lead AQCD, which was also reviewed by the Lead Panel, in its review of EPA's 1st Draft Lead Staff Paper and the Draft Lead Exposure and Risk Assessments document. The Lead Panel's recommendations and the associated scientific basis for these recommendations are presented below. *The unanimous judgment of the Lead Panel is that lead should not be de-listed as a criteria air pollutant, as defined by the Clean Air Act, for which primary (public-health based) and secondary (public-welfare based) NAAQS are established, and that both the primary and secondary NAAQS should be substantially lowered.* It is also recommended that future monitoring of lead exposure be conducted with low-volume PM_{10} samplers rather than with total suspended particulate (TSP) samplers, and that the averaging time be decreased from quarterly to monthly.

The reasons for these recommendations are given below.

Introduction

Over the past three decades, blood lead (PbB) concentrations in the U.S. population have plummeted (1). This decline was largely due to the elimination of leaded gasoline (2). In 1976, the Consumer Products Safety Commission restricted the allowable amount of lead in residential paints to 0.06 percent (600 ppm) (3). Lead solder used in canned foods was also decreased — from over 90% in 1978 to less than 5% in 1988 (4). Finally, there was a decrease in the abundance of residential housing in which lead-based paints had been used (5). Although it is difficult to quantify the extent of decrease in blood lead concentrations attributable to specific sources, the 1978 NAAQS for lead were undoubtedly among the major reasons for the rapid and widespread decrease in PbB levels in the U.S. population (6).

Despite the dramatic decrease in environmental lead exposure, lead toxicity remains a major public health problem. Environmental lead exposure in children has been associated with increased risks for reading problems, school failure, Attention Deficit Hyperactivity Disorder (ADHD), delinquency, and criminal behavior (6–10). Among U.S. children, eight to fifteen years old, those in the highest quintile (> 2 µg/dl) of lead exposure were four times more likely to have doctor-diagnosed ADHD (11). Moreover, there is no evidence of a threshold for the adverse consequences of lead exposure; studies show that the decrements in intellectual (cognitive) functions in children are proportionately greater at PbB concentrations < 10 µg/dl, the concentration considered acceptable by the Centers for Disease Control (11–14).

Lead's effects extend beyond childhood. In adults, lead exposure is a risk factor for some of the most prevalent diseases or conditions of industrialized society, including cardiovascular disease and renal disease (16–20). There is also compelling evidence that the risks for mortality from stroke and myocardial infarction are increased at PbB concentrations below 10 µg/dl, which is considerably lower than those considered acceptable for adults (19). Finally, although less definitive, there is also evidence that lead exposure during pregnancy is a risk factor for spontaneous abortion or miscarriage at PbB concentrations < 10 µg/dl (21). (It should be noted that references 11 and 19 above were not cited in EPA's Final Ozone AQCD.)

Scientific Basis for Continuing or De-listing the Lead NAAQS

The CASAC Lead Review Panel considered the implications of present scientific understanding regarding the need for protection of public health and public welfare from exposure to lead in the environment. One of these implications relates to the question of whether the current science continues to support the need for lead to be listed as a criteria air pollutant for which a NAAQS is established, or might warrant the de-listing of lead, as presented as a policy option in the 1st Draft Lead Staff Paper. In addressing this question, the Lead Panel examined several scientific issues and related public health and public welfare issues that are essential in determining whether or not a pollutant such as lead should be de-listed or maintained as a criteria air pollutant.

1. *Does new scientific information accumulated since EPA's promulgation of the current primary Lead NAAQS of 1.5 µg/m^3 in 1978 suggest that science previously overstated the toxicity of lead*? Here, the Lead Panel's answer clearly is No. The data accumulated over the past three decades make it apparent that adverse health effects on both humans and

other species appear at blood lead concentrations and environmental exposures well below those previously thought to pose important risks. Indeed, if anything, this improved scientific understanding indicates that scientific studies previously underestimated the toxicity of lead.

2. *Have past regulatory and other controls on lead decreased PbB concentrations in human populations so far below levels of concern as to suggest there is now an adequate margin of safety inherent in those PbB levels*? Again, the Panel's answer here is No. The Nation can take great pride in the extent to which exposures to lead have been decreased, leading to laudable decreases in PbB concentrations to an average approaching 2 µg/dl. However, there remains a significant segment of the population with blood-lead concentrations above 5 µg/dl — and some even above 10 µg/dl — and scientific evidence supports the contention that these PbB concentrations do not provide an adequate margin of safety. In fact, this evidence suggests these blood lead concentrations below 5 µg/dl are associated with unacceptable adverse effects.

3. *Have the activities that produced emissions and atmospheric redistribution of lead in the past changed to such an extent that society can have confidence that emissions will remain low even in the absence of NAAQS controls*? Here, the Lead Panel concludes that the answer, once again, is No. While there have been major decreases in emissions of lead from use of leaded gasoline, industrial and other activities, even the current air emissions from some lead mining and reprocessing facilities produce considerable environmental exposures once the concentrations of lead in environmental media equilibrate. The Lead Panel concludes that past success in decreasing PbB concentrations in human populations are due in part to NAAQS controls, and that in the absence of such controls, there will be a significant possibility that blood-lead concentrations would begin to rise again.

4. *Are airborne concentrations and amounts of lead sufficiently low throughout the United States that future regulation of lead exposures can be effectively accomplished by regulation of lead-based products and allowable amounts of lead in soil and/or water?* Lead Panel concludes that the answer to this question is No. While airborne lead concentrations have been decreased throughout much of the United States, airborne lead remains a primary vehicle for movement of lead between different environmental compartments. While control of airborne lead is not sufficient by itself to control exposure to lead, it is an essential component of a successful control strategy. Maintaining appropriate Lead NAAQS is considered by the Lead Panel to be an essential component of a national program to decrease the ongoing adverse effects of lead in children, adults, and in both terrestrial and aquatic ecosystems.

5. *If lead were de-listed as a criteria air pollutant, would it be appropriately regulated under the Agency's Hazardous Air Pollutants (HAP) program?* The Panel's answer is again No. The HAP program, which regulates according to use of maximum achievable control technology (MACT), followed by an analysis of residual risk, is appropriate for point sources. However, the most widespread source of airborne lead throughout the

nation is the historically-deposited lead along roadways. Thus, this source of airborne lead could not be regulated under the HAP program.

As a result of the CASAC Lead Review Panel's own answers to these scientific and public health issues, the Panel concludes that the existing state of science is consistent with continuing to list ambient lead as a criteria air pollutant for which fully-protective NAAQS are required.

Additional Analyses to Inform Decisions About a Primary (Health-Based) NAAQS for Lead

Despite the dramatic decreases in amounts of airborne lead exposures and human-population blood-lead concentrations following the phase-out of leaded gasoline, lead toxicity remains a major public health problem. As discussed above, there is increasing evidence of lead-induced toxicity at the lowest contemporary exposures to lead — resulting in significant IQ deficits in children (11–14), and increased frequency of ADHD (11) and cardiovascular disease (16–19). Although less definitive, there is evidence that lead exposure is a risk factor for spontaneous abortion and renal disease (20–21).

Although relatively few counties in the United States are out of compliance, the greatest benefit to public health will be realized by broad decreases in airborne lead concentrations across the U.S. population because:

1. The adverse consequences are proportionately greater at the lowest increments of lead exposure;
2. Lead exposure is cumulative; and
3. Airborne lead exposure, in contrast with exposure to lead-based paint, is more widely dispersed. Thus, reducing exposure from air lead will broadly reduce population blood lead levels.

In 1978, EPA established a primary Lead NAAQS of 1.5 $\mu g/m^3$ to ensure that 99.5% of the public did not exceed a blood-lead concentration of 30 µg/dl, with the 99.5% figure being the Agency's risk management (*i.e.*, policy) choice at that time. In addition to the separate Federal regulations that had been adopted in 1973 that requiring the phase-out of leaded gasoline, the 1978 Lead NAAQS was instrumental in helping to produce the dramatic decreases in air lead and blood-lead concentrations over the last 30 years. However, these primary and secondary Lead NAAQS are totally inadequate for assuring the necessary decreases of lead exposures in sensitive U.S. populations below those current health hazard markers identified by a wealth of new epidemiological, experimental and mechanistic studies.

Consequently, *it is the CASAC Lead Review Panel's considered judgment that the NAAQS for Lead must be decreased to fully-protect both the health of children and adult populations.*

The EPA pilot-phase human health risk assessment focused on three case study locations (*i.e.*, primary lead smelter, secondary lead smelter, and near-roadway urban). While the case study approach undertaken in the risk assessment is enlightening and provides a potentially

useful framework for understanding lead exposure for some discrete populations within the U.S., there are some additional considerations and analyses that the Lead Panel strongly feels could help inform a scientifically-defensible NAAQS for lead. In particular, the Panel believes that the risk assessment would be better informed with a *"population-based"* risk assessment to supplement the current case study approach. A population-based risk assessment would typically include two key components:

1. A quantitative description of the relationship between concentrations of lead in national ambient air and distributions of resulting blood lead concentrations; and
2. A quantitative description of the relationship between blood lead concentrations and impacts on IQ.

There are multiple ways in which EPA could conduct a population-based analysis, and the Panel illustrates some possibilities in Appendix D attached to this letter. Please note that this work does not represent a complete analysis on the part of the Lead Panel; rather, it is meant to illustrate the Panel's thinking in this area. It will be important for EPA to consider these approaches and to fully evaluate their pros, cons, and associated uncertainties. An adequately comprehensive analysis should characterize the uncertainty, preferably in a quantitative manner, in two key areas: (1) the relationship between a change in the NAAQS for lead and the distribution of population blood lead concentrations; and (2) the relationship between blood lead concentrations and the risk of adverse health effects. This type of review by the Agency will be necessary to highlight the strengths and weaknesses of the available approaches and help to elucidate a primary Lead NAAQS that is scientifically-defensible and adequately-protective of public health.

As described in Appendix D, the Lead Panel considered three separate, but related, population-based analytical approaches as examples to be considered in deriving an acceptable range of alternative levels, on the basis of the scientific evidence, for setting a new level for the primary Lead NAAQS, as follows: Approach A relates air lead levels to blood lead levels using the approach used in previous lead NAAQS calculations; Approach B uses an epidemiologic approach to derive an adjusted slope factor relating air lead levels to blood levels taking into account all exposure pathways between air lead and blood lead; and Approach C relates air lead levels to blood lead levels and then to IQ loss in children. In addition, *the CASAC Lead Review Panel considers that a population loss of 1-2 IQ points is highly significant from a public health perspective (22). Therefore, the primary lead standard should be set so as to protect 99.5% of the population from exceeding that IQ loss.*

The three approaches provide comparable results. Given the Panel's assumptions and preliminary analysis conducted for the three approaches, *the resulting analyses indicate to the CASAC that there is a need for a substantial reduction in the primary Lead NAAQS, to a level of about 0.2 µg/m^3 or less*. CASAC recognizes that these preliminary calculations are dependent upon the results of EPA's forthcoming uncertainty analyses and the current risk management choice for the percentage of the population left at risk, as well as acceptable blood levels, IQ loss and slope factor — the appropriateness of which all depend on certain scientific assumptions and the risk management criteria that are chosen. Imposing more stringent criteria would result in a lower (that is, more stringent) range of primary Lead NAAQS levels, whereas less stringent

criteria would result in the calculation of a higher (*i.e.*, less stringent) range of primary lead standards.

Possible Revision to Lead Indicator from TSP to Low-Volume PM_{10}

As revisions to the level, form, and averaging time of the Lead NAAQS are considered, CASAC also recommends that EPA revise the indicator. Currently, Lead NAAQS monitoring is predominantly based on atomic absorption analysis of fiberglass filters run on hi-volume total suspended particulate (TSP) samplers. Most other TSP sampling was discontinued after PM_{10} standards were promulgated in 1987. TSP samplers capture particles with an imprecise and variable upper particle cut size in the range of approximately 30 to 50 microns on fiberglass filters which are not well-suited for analysis by inexpensive, multi-elemental surface beam techniques like particle-induced X-ray emission (PIXE) or X-ray fluorescence (XRF). Consequently TSP sampling by imprecise samplers is primarily conducted only for lead analysis and these filters are rarely analyzed for other species.

If Lead NAAQS monitoring was based on (low-volume) PM_{10} sampling on Teflon filters, the resulting data would be correlated with TSP lead, as suggested by limited data in the 1st Draft Lead Staff Paper, but would have substantially improved sampling precision. The Lead Panel recognizes that either monitoring system would be subject to variability based on location, particularly near sources. Other advantages of low-volume PM_{10} sampling include:

1. Focus on those biologically-relevant particles that, when inhaled, are deposited in the thoracic region;
2. Larger spatial-scale representativeness for population exposures to monitored particles which remain airborne longer;
3. Could utilize more widespread PM_{10} and "air toxics" metals sampling networks, leading to collection of more data at lower costs;
4. Potential for inexpensive multi-elemental analysis by XRF or PIXE would provide useful supplemental metals information for health effects studies and source apportionment;
5. Potential for automated sequential PM_{10} samplers (not available for TSP) would be especially useful if sampling frequency is increased from once every six days; and
6. Weighing filters would provide useful information on PM_{10} mass; and, if collocated with $PM_{2.5}$ Federal Reference Methods (FRM), could provide needed information on $PM_{10-2.5}$ mass and speciation.

Reasons for retaining the current TSP lead indicator include: preservation of a long-term historical record at some sites; and inclusion of very coarse (> 10 micron particle) lead which may deposit in upper regions of the respiratory tract and ultimately be ingested, or which may deposit on surfaces and be ingested via hand-to-mouth activity of children. Some such coarse particles might be missed by PM10 samplers. Presumably a downward scaling of the level of the Lead NAAQS could accommodate the loss of very large coarse-mode lead particles, and some short period of concurrent PM_{10} and TSP lead sampling could help develop site-specific scaling factors at sites with highest concentrations where long-term historical records are important.

Given the advantages of using PM_{10}, the CASAC Lead Review Panel recommends that the Agency revise the lead indicator to utilize low-volume PM_{10} sampling, and also develop equivalent analytical methods to allow use of XRF and Inductively-Coupled Plasma Mass Spectrometry (ICP-MS) analysis.

Possible Revision to Averaging Time Used for the Lead NAAQS

A second change that should be considered with a change in the Lead NAAQS is possible use of a different averaging time. Currently, quarterly averaging is used. However, studies suggest that blood lead concentrations respond at shorter time scales than would be captured completely by quarterly values. Here, the Lead Panel recommends that the Agency conduct *monthly* averaging instead of quarterly.

One consideration involved in using a shorter averaging period is sampling frequency. Currently, many of the samplers operate with sampling frequencies less than once per day, and as infrequently as every sixth day. In the most extreme case, as few as four samples may be involved in determining a monthly average (assuming no samples are considered invalid). This may make the average susceptible to anomalously-high events. On the other hand, this may motivate more frequent sampling in those areas whose air concentrations are near the level of the Lead NAAQS, which would increase the protection of public health and significantly decrease the impact of a single high lead exposure event. One could also consider having the lead standards based on the second highest monthly average, a form that appears to correlate well with using the maximum quarterly value.

The CASAC Lead Review Panel recommends adopting monthly averaging as being more protective of human health in light of the response of blood lead concentrations that occur at sub-quarterly time scales, and further recommends that the most protective form would be the highest monthly average in a year. An area could choose to increase sampling frequency to make the monthly average less susceptible to more extreme events. Such a change is consistent with either using TSP or PM_{10} sampling.

Secondary (Welfare-Based) NAAQS for Lead

An extended discussion of issues related to setting the secondary Lead NAAQS can be found in Appendix E. Chapter 6 of the 1st Draft Lead Staff Paper and Chapter 7 of the "Pilot Phase" Draft Lead Exposure and Risk Assessments technical support document present compelling scientific evidence that current atmospheric lead concentrations and deposition — combined with a large reservoir of historically-deposited lead in soils, sediments and surface waters — continue to cause adverse environmental effects in aquatic and/or terrestrial ecosystems, especially in the vicinity of large emission sources. These effects persist in some cases at locations where current airborne lead concentrations are below the levels of the current primary and secondary lead standards.

Thus, from an environmental perspective, there are convincing reasons to both retain lead as a regulated criteria air pollutant and to lower the level of the current secondary standard.

Since concentrations of historically deposited lead in soils throughout the U.S. (averaging 0.5 to 4 g/m^2 of land area) are changing only slowly — with a half-life exceeding a century — these concentrated deposits of lead are expected to remain accessible for exchange with the atmosphere and the rest of the biosphere into the foreseeable future. Fires, changes in land use, or climatic events such as regional dust storms could mobilize significant quantities of lead that would be harmful both to human health and ecosystems downwind. This potential for harm is not adequately recognized in the 1st Draft Lead Staff Paper and the Draft Lead Exposure and Risk Assessments technical support document, but is a concern that warrants careful continued monitoring in the future.

In addition, while neither the 1st Draft Lead Staff Paper nor the Draft Lead Risk/Exposure Assessments document provide a clear quantitative basis for identifying a specific lower level at which a more protective secondary (welfare- or environmental-based) Lead NAAQS should be set, there are no reasons to expect that humans are uniquely sensitive to lead pollution among the millions of animal and plant species.

Therefore, at a minimum, the level of the secondary Lead NAAQS should be at least as low as the lowest-recommended primary lead standard. The EPA is also encouraged to identify the necessary funds to support needed continuing research on the ecological effects of airborne lead pollution and to consider developing alternative secondary standards such as critical loads *for lead, which may be different from primary standards in indicator, averaging time, level or form.*

The CASAC continues to be pleased to provide advice to you concerning the scientific basis for the setting of the primary and secondary Lead NAAQS. In addition, the CASAC looks forward to continued dialog with Agency officials and staff aimed at improving EPA's NAAQS review process in a manner that enhances the efficiency of the process while maintaining its integrity and adherence to the stipulations of the Clean Air Act. Finally, the Committee also looks forward to reviewing the 2nd draft of the Agency's Lead Risk/Exposure Assessment this summer. As always, we wish Agency staff well in this important task.

Sincerely,

/Signed/

Dr. Rogene Henderson, Chair
Clean Air Scientific Advisory Committee

Appendix A – Roster of the Clean Air Scientific Advisory Committee

Appendix B – Roster of the CASAC Lead Review Panel

Appendix C – Agency Charge to the CASAC Lead Review Panel

Appendix D – Population-Based Approaches to Risk Assessment Analyses for the Primary Lead NAAQS

Appendix E – Issues Related to the Setting of the Secondary Lead NAAQS

Appendix F – Review Comments from Individual CASAC Lead Review Panel Members

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Appendix A – Roster of the Clean Air Scientific Advisory Committee

U.S. Environmental Protection Agency
Science Advisory Board (SAB) Staff Office
Clean Air Scientific Advisory Committee (CASAC)

CHAIR

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Appendix B – Roster of the CASAC Lead Review Panel

U.S. Environmental Protection Agency
Science Advisory Board (SAB) Staff Office
Clean Air Scientific Advisory Committee (CASAC)
CASAC Lead Review Panel

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* Members of the statutory Clean Air Scientific Advisory Committee (CASAC) appointed by the EPA Administrator

Appendix C – Agency Charge to the CASAC Lead Review Panel

Charge to the CASAC Pb Panel

Within each of the main sections of the first draft Staff Paper, questions that we ask the Panel to focus on in their review include the following:

Ambient Pb information and analyses (Chapter 2):

1. To what extent are the emissions and air quality characterizations and analyses clearly communicated, appropriately characterized, and relevant to the review of the primary and secondary Pb NAAQS?

2. Does the information in Chapter 2 provide a sufficient ambient Pb-related basis for the exposure, human health and environmental effects, health risk assessment, and environmental assessment presented in later chapters?

Pb-related health effects (Chapter 3):

1. To what extent is the presentation of evidence from the health studies assessed in the Pb AQCD and the integration of information from across the various health-related research areas drawn from the Pb AQCD technically sound, appropriately balanced, and clearly communicated?

2. What are the views of the Panel on the appropriateness of staff's discussion and conclusions in Chapter 3 on key issues related to quantitative interpretation of epidemiologic study results, including, particularly, the form of a blood Pb-response function for neurocognitive effects, and the form of the associated blood Pb metric?

3. What are the Panel's views on the adequacy and clarity of the discussion of potential thresholds in concentration-response relationships presented in Chapter 3?

Human Exposure and Health Risk Analysis, Pilot-Phase (Chapter 4):

1. To what extent are the assessment, interpretation, and presentation of the results of the pilot exposure analysis, including characterization of Pb concentrations in media, the modeling of multi-pathway Pb exposure and application of biokinetic blood Pb models, as presented in Chapter 4 technically sound, appropriately balanced, and clearly communicated?

2. Are the methods used to conduct the pilot exposure analysis, including the modeling of population-level distributions of total blood Pb levels and the pathway-apportionment of those blood Pb levels (*e.g.*, air-inhalation, versus soil-ingestion versus dust-ingestion, versus

background) technically sound? Does the Panel have any suggestions for improvements in the methods used?

3. What are the Panel's views on the staff interpretation of the performance evaluation completed for the pilot analysis (and described in Chapter 4) with regard to the representativeness of individual modeling steps completed for the analysis (*e.g.*, characterization of ambient air and outdoor soil Pb levels and the estimation of blood Pb levels for specific case studies)?

4. In general, are the concentration-response functions and blood Pb metrics (*i.e.*, lifetime average, concurrent blood lead) used in the pilot analysis appropriate for this review?

5. Are the methods used to conduct the pilot health risk assessment, including the application of the cutpoints in relation to the concentration-response functions employed, technically sound? Does the Panel have any suggestions for improvements in the methods used?

6. To what extent does the sensitivity analysis completed for the pilot analysis (and described in Chapter 4) identify key sources of uncertainty and provide an assessment of their impact on risk results?

7. As part of the NAAQS review, there is interest in attempting to differentiate Pb exposure and health risk impacts for modeled populations between: (a) historically-deposited Pb (*e.g.*, near-roadway dust/soil lead from leaded gasoline); and (b) newly-emitted Pb. Does the Panel have specific recommendations regarding approaches that might be employed in the full-scale assessment for this purpose?

8. What are the Panel's views on the most important issues to be addressed in the subsequent full-scale human exposure and health assessment that will be presented in the revised documents?

<u>The Primary Pb NAAQS (Chapter 5)</u>

1. What are the Panel's views on the adequacy and clarity of the presentation of the basis for the existing standard and conclusions reached in the last review?

2. Based on the information contained in the first draft Staff Paper, as well as the AQCD, does the Panel have recommendations with regard to specific aspects of the standard to be considered in developing policy alternatives? For example, considering the prominence of the soil and dust pathways for ambient Pb exposures, and the evidence regarding environmental response times, is there reason to give more emphasis to consideration of an alternative (shorter or longer) averaging time; and, how might this be considered in the full-scale risk assessment given current capabilities?

Pb-related welfare effects and screening level ecological risk assessment (Chapter 6):

1. To what extent is the presentation of evidence from the ecological studies assessed in the Pb AQCD and the integration of information from across the various ecologically-related research areas drawn from the Pb AQCD technically sound, appropriately balanced, and clearly communicated?

2. Given the lack of quantitative information on Pb-related ecosystem effects, what are the Panel's views on the presentation of this topic in chapter 6?

3. What are the Panel's views of the data sources and models used to estimate current levels of Pb in soil, freshwater, and sediment for the case study locations?

4. To what extent are the methods used to conduct the exposure assessment and the interpretation and presentation of the results technically sound, appropriately balanced, and clearly communicated?

5. What are the Panel's views of the approach for addressing uncertainty in apportionment of Pb contributions in the national-scale screen by factoring out those locations with known non-air sources (*e.g.*, mining, point discharges)?

6. To what extent are the assessment, interpretation, and presentation of the results of the screening-level risk analysis, including characterization of lead concentrations in media and the comparisons to ecological screening values, as presented in Chapter 6 and the risk assessment report technically sound, appropriately balanced, and clearly communicated?

7. Does the Panel feel that adequate screening criteria (ecotoxicity screening values) were selected for each of the media?

8. What are the Panel's views on the derivation of the soil screening values for birds and mammals (*i.e.*, using the Eco-SSL methodology)? Do the resultant values adequately reflect current information on exposure characteristics of these organisms?

9. To what extent are the uncertainties associated with the exposure analysis clearly and appropriately characterized in Chapter 6 and the risk assessment report?

Appendix D – Population-Based Approaches to Risk Assessment on Analyses for the Primary Lead NAAQS

The CASAC Lead Review Panel considered three separate, but related, population-based analytical approaches as examples to be considered in deriving an acceptable range of alternative levels, on the basis of the scientific evidence, for setting a new level for the primary Lead NAAQS, as follows:

- **Approach A** relates air lead levels to blood lead levels using the approach established in previous lead NAAQS calculations;
- **Approach B** uses an epidemiologic approach to derive an adjusted slope factor relating air lead levels to blood levels taking into account all pathways between air lead and blood lead; and
- **Approach C** relates air lead levels to blood lead levels and then to IQ loss in children.

These approaches consider existing information and the following assumptions:

- the population to be protected (99.5% of the population of children);
- the *maximal acceptable blood concentration* (up to 5.0 µg/dl);
- an *appropriate geometric standard deviation (GSD)* for the blood lead levels in children exposed to a given level of air lead (range 1.3–2.0);
- the *non-air background* (1.0–1.4 µg/dl or lower range should be considered);
- the *slope factor* for the relation between air lead and blood lead for levels of blood lead below 10 µg/dl, with the candidate values considered being 2.0 µg/dl per µg/m^3 (m^3/dl) used in 1978, 5.0 m^3/dl used by the World Health Organization (WHO) in 2000, 10.0 m^3/dl noted in recent studies (see the discussion in Approach B below), and assuming 20.0 m^3/dl as a maximum; and
- the most sensitive toxicity endpoint (*i.e.*, IQ loss in children).

In addition, the CASAC Lead Review Panel considers that a population loss of 1-2 IQ points is highly significant from a public health perspective (22). Therefore, the primary lead standard should be set so as to protect 99.5% of the population from exceeding that IQ loss.

The three approaches provide comparable results. Given the Panel's assumptions and preliminary analysis conducted for the three approaches, *the resulting analyses indicate to the CASAC that there is a need for a substantial reduction in the primary Lead NAAQS, to a level of about 0.2 µg/m^3 or less*. CASAC recognizes that these preliminary calculations are dependent upon the results of the Agency's forthcoming uncertainty analyses and: the values chosen for the percent of the population left at risk; acceptable blood levels and IQ loss; and slope factor — the appropriateness of which all depend on certain scientific assumptions and the risk management criteria that are chosen. Imposing more stringent criteria would result in a lower (that is, more

stringent) range of primary Lead NAAQS levels, whereas less stringent criteria would result in the calculation of a higher (*i.e.*, less stringent) range of primary lead standards.

Approach A

The first approach (A) relates air lead levels to blood lead (PbB) levels using a simplified and modified empirical-deterministic approach that is essentially the same approach used in previous EPA NAAQS (1978) and World Health Organization (WHO, 2000) guidance documents. This approach begins with selection of a "not-to-be-exceeded" PbB value or values based on scientific evidence. These "not-to-be-exceeded" PbB values for beginning the 1978 NAAQS and the 2000 WHO uses of the approach were 30 and 10 µg/dl respectively. The current scientific evidence reviewed by the Panel, per the Agency's Final Lead AQCD indicates that the concentration of lead in blood shown to be harmful has declined substantially below those levels, to around 5 µg/dl or less.

For example, based on current evidence, one might consider two "not-to-be-exceeded" PbB values, such as 5.0 and 2.5 µg/dl. These are not to be exceeded at the 99.5 percentile and, for an illustrative GSD of 1.3, produce geometric mean values of 2.5 and 1.3 µg/dl, respectively. The non-air portion of these two means must be subtracted to give the air Pb-based contributions to PbB. Panel member Dr. Paul Mushak (Appendix F) calculated the non-air portion using the Integrated Exposure Uptake Biokinetic Model for Lead in Children (IEUBK).

The use of a "not-to-be-exceeded" PbB value of 5 µg/dl in Approach A and slope factors of 5, 10, or 20 produce corresponding suggested NAAQS values of 0.22, 0.11, or 0.06 µg/m^3. Use of a "not-to-be-exceeded" value of 2.5 µg/dl and the same slope factors produce air lead values half as high, *i.e.*, 0.11, 06 and 0.03 µg/m^3, respectively. (Note that, for the 1.25 mean PbB scenario, the non-air PbB contribution is the dominant source and modeling does not provide an exact value; see Dr. Paul Mushak's detailed derivation comments in Appendix F, pp. F-55 through F-57.) Based on these values alone, the current primary Lead NAAQS set in 1978 should be lowered by at least a factor of seven and by as much as 50, depending on the slope factor used (see, in particular, the individual comments of Lead Panel members Dr. Mushak and Dr. Ian von Lindern found in Appendix F).

Approach B

Approach B is a "top-down" approach. That is, instead of estimating the effect of inhalation alone, the effect of air lead on deposition into dust, food, *etc.* and the uptakes from those pathways, an epidemiologic approach should be used to derive an adjusted slope factor taking into account all pathways between air lead and blood lead. This is based on the changes in blood lead observed when lead began to be phased-out of gasoline. This analysis relies on the results of Schwartz and Pitcher (23).

The Schwartz and Pitcher analysis showed that in 1978, the midpoint of the National Health and Nutrition Examination Survey (NHANES) II, gasoline lead was responsible for 9.1 µg/dl of blood lead in children. Their estimate is based on their coefficient of 2.14 µg/dl per 100 metric tons (MT) per day of gasoline lead use, and usage of 426 MT/day in 1976. Between 1976 and when the phase-out of lead from gasoline was completed, air lead concentrations in U.S.

cities fell a little less than 1 μg/m^3 (24). These two facts imply a ratio of 9–10 μg/dl per μg/m^3 reduction in air lead, taking all pathways into account.

Under this scenario, a decrease of mean air lead concentrations of 0.1 μg/m^3 could be expected to produce a further decrease in average blood lead concentrations of 0.9–1.0 μg/dl. Assuming a slope of three IQ points per μg/dl reduction in blood lead, which is indicated by the pooled analysis of low concentration lead exposure (13), this further decrease would be expected to raise the average IQ of children in U.S. cities by approximately three IQ points — a significant positive health impact. Put another way, the derivation above empirically justifies the use of the slope factor of 10 in Approach C, and the resulting estimates that an air quality standard of 0.11 μg/m^3 (that is, a 13-fold reduction) would be required to keep 99.5% of the children below a blood lead of 5 μg/dl.

Approach C

Approach C is more sophisticated, starting with an air lead level and a blood lead level produced only by airborne lead, and relates that air level to IQ point loss (see Table 2). A linear model between the ranges of 1-7.5 μg/dL PbB for both concurrent and lifetime exposures suggests a three-point decrement in IQ for each unit change in PbB (13). Approach A and C are in agreement on the relationship between air lead levels associated with PbB, dependent on the slope factor used. (Approach A does not consider IQ loss or any other health effect.)

These considerations are summarized in Tables 1 and 2, and are also contained in the individual review comments from various Lead Panel members attached as Appendix F. Depending on the slope factor selected between 5 and 20 m^3/dl, the estimate of blood lead concentrations from various air lead concentrations varies by a factor of four (Table 1). For example, using the linear estimate of IQ loss associated with PbB below 7.5 μg/dl (13), the Lead Panel estimated that, over the range of PbB from 0.5–4.0 μg/dl (*i.e.*, an eight-fold range), the loss of IQ would similarly increase from 1.5 to 12 IQ points (Table 2).

Since the Lead Panel considers a population loss of 1-2 IQ points to be highly significant from a public health perspective, the Lead Panel therefore considers this extent of loss in IQ as a "change in IQ not to be exceeded." Depending upon the slope factor selected, this results in a range of 0.025–0.200 μg/m^3 (*i.e.*, about a 7.5- to 60-fold decrease from the current primary Lead NAAQS) as the estimated air lead concentration to consider under Approach A.

References

23. Schwartz J, Pitcher H. The relationship between gasoline lead and blood lead in the United States. 1989 *J. Official Stat.* 5: 421-431.

24. U.S. Environmental Protection Agency. (1986) *Air Quality Criteria for Lead*. Research Triangle Park, NC: Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office; EPA report no. EPA/600/8-83/028aF-dF. 4v. Available from: NTIS, Springfield, VA; PB87-142378, p. 1-21.

TABLE 1. Relationship of Blood Lead (PbB) to Air Lead (Pb-Air) by Differing Slope Factors

Pb-Air (µg/$m^{3)}$	PbB (µg/dl)		
	S.F.* = 5	S.F.* = 10	S.F.* = 20
0.010	0.05	0.10	0.20
0.025	0.13	0.25	0.50
0.050	0.25	0.50	1.00
0.100	0.50	1.00	2.00
0.200	1.00	2.00	4.00

*S.F. = slope factor (m^3/dl) = PbB/Pb-Air; S.F. value varies with increasing impact of indirect Pb-Air pathway (Dust Pb + Soil Pb)

TABLE 2. Relationship of IQ Point Losses to Increases in Pb-Air and Pb-Air-Based Blood Lead (PbB) Values Above Zero [a,b,c]

Pb-Air ($\mu g/m^3$)	S.F. = 5		S.F. = 10		S.F. = 20	
	PbB [d]	IQ Loss [e,f]	PbB	IQ Loss	PbB	IQ Loss
0	0	0	0	0	0	0
0.010	0.05	< 1	0.10	< 1	0.20	< 1
0.025	0.13	< 1	0.25	< 1	0.50	1.5
0.050	0.25	< 1	0.50	1.5	1.00	3.0
0.100	0.50	1.5	1.00	3.0	2.00	6.0
	1.00	3.0	2.00	6.0		12.0
0.200					4.00	

a Pb-Air-related increases affecting IQ point loss through calculated PbB values using 3 slope factors per Table 1

b IQ vs. PbB dose-response relationships based on Lanphear *et al.*, 2005 (13): sub-7.5 µg/dl linear segment, average slope = 3.0, combining slopes of 2.9 and 3.1 for concurrent and lifetime average dose metrics, respectively

c Slope factors as defined in Table 1 and text

d PbB as derived in Table 1

e Rounding for values < 1 IQ point

f Population, not individual, IQ loss/gain projections; U.S. CDC 2007 estimates 23,380,860 U.S. children 0-71 months of age. Source: U.S. Centers for Disease Control, 2007. CDC Surveillance Data. (Last updated 2/16/2007). URL: http://www.cdc.gov/nceh/surv/stats.htm [accessed 3/8/2007]

Appendix E – Issues Related to the Setting of the Secondary Lead NAAQS

Chapters 6 of the Agency's 1st Draft Lead Staff Paper and Chapter 7 of EPA's Draft (Pilot-Phase) Lead Exposure and Risk Assessments document summarize a very large body of scientific knowledge about environmental and ecological effects of atmospherically deposited lead on the biota, soils, sediments, and surface and ground waters of terrestrial and aquatic ecosystems in various parts of the U.S. and nearby parts of the world. This significant body of scientific knowledge includes environmental effects of both historically-deposited lead and continuing air dispersal or reentrainment of lead compounds from primary and secondary lead smelters, along roadsides, and in ecologically-sensitive areas such the Hubbard Brook Experimental Forest as described in Chapter 7 of the pilot-phase technical support document.

Although lead is recognized in these two chapters as one among a longer list of heavy metals in the environment (including cadmium, zinc, and mercury), these two chapters do not contain adequate discussion of the special characteristics of lead or its ecological effects in the context of these other metals — or other air-dispersed criteria pollutants. Also, little of the information about specific environmental effects of lead is presented in a way that is directly relevant to the issue of whether the EPA Administrator should retain, increase, or decrease the present primary and secondary National Ambient Air Quality Standard (NAAQS) for lead. These identical standards were established in 1978 and have been maintained ever since at a level of 1.5μg/m^3 as a quarterly average (maximum arithmetic mean averaged over a calendar quarter).

The Lead Panel believes that especially Chapter 6 of the draft Lead Staff Paper (and perhaps also at least parts of Chapter 7 of the pilot-phase risk-exposure assessment document) would be much improved in their intended purposes if they were to contain a concise summary of:

1. The knowledge available and (as best they can discern) the rationale used by the Administrator in promulgating the original NAAQS for Lead in 1978;
2. The knowledge available and rationale used in the decisions made in 1989 and 1990 to retain unchanged the identical primary and secondary Lead NAAQS established in 1978; and
3. The knowledge available and rationale used by the Administrator in establishing and maintaining identical primary and secondary standards for criteria air pollutant — not only for lead — but also for most of the other criteria pollutants for NAAQS since 1970.

Despite the limitations mentioned in the last two preceding paragraphs, the Lead Panel believes that the body of scientific knowledge summarized in the Agency's Final Air Quality Criteria Document (AQCD) for lead, and further presented in the aforementioned chapters in the 1st Draft Lead Staff Paper and the Draft (Pilot-Phase) Lead Exposure and Risk Assessments documents, provide compelling scientific justification for both:

1. The original (1978) decision by EPA to regulate lead as a Criteria Air Pollutant and to establish what was then considered to be an appropriately-designed primary (public-health based) NAAQS for lead, with a secondary (public-welfare based) standard set at the same level and form, and

2. Maintaining for the foreseeable future similarly well designed (but contemporarily scientifically well-informed) primary and secondary NAAQS for lead — standards with levels and forms that may be different from, rather than identical to each other.

There are several features of the environmental and ecological effects of lead, and both the chemical and physical properties of lead in the environment, that make lead distinct from the other four criteria pollutants for which NAAQS have been developed by the EPA. These distinctive properties include:

1. The widespread use of lead as an ingredient in decorative paints, lead-acid batteries, as an additive for gasoline used in motor vehicles, and even in some pesticides used earlier to protect some horticultural crops from plant pathogens;
2. The persistence of lead in soils, surface and ground waters, sediments, and in both the structural- and some biologically-active tissues of plants, animals, insects, and microorganisms;
3. The well-known toxicity and interference in development of cognitive functional capacity in humans (especially children) and the much less well-known toxicological and other effects of lead on all the other different types of animals, plants, insects, and microorganisms in managed and natural terrestrial and aquatic ecosystems of the Earth — some of which are undoubtedly even more sensitive to lead than human infants;
4. The very substantial decreases in current air concentrations and atmospheric deposition of lead into the environment that were achieved in recent decades through:

   (a) The Phase-out of lead additives in gasoline during the 1970s, ‘80s, and ‘90s;

   (b) Severe limits on air emissions from lead smelters during earlier decades; and

   (c) Decreases in air emissions from lead battery processing facilities in more recent years.

   Thus, most current exposures of living organisms in natural and managed terrestrial and aquatic ecosystems are caused primarily by redistribution of environmentally persistent airborne lead compounds deposited in soils, sediments, and surface waters during the latter earlier decades of the 20^{th} century.

5. The continuing airborne resuspension and dispersal of lead that persists in soils, fugitive dusts, sediments, and surface waters and are transported and deposited once again from air in both fine and coarse particulate matter and aerosols — especially along roadways.

These distinctive properties of lead suggest to some policy makers that ecological and environmental effects of lead might be managed by other means than maintaining both primary and secondary Lead NAAQS. In the Lead Panel’s considered judgment, the limitations of the other methods of management now available to the EPA are such that none of these alternative

methods would be anywhere near as effective in protecting public health or welfare as maintaining *for the foreseeable future* as appropriately well-designed (and contemporarily scientifically well-grounded) primary and secondary NAAQS for lead.

As indicated in the body of the letter to the EPA Administrator, the members of the CASAC Lead Review Panel have provided a consensus scientific judgment that the present level ($1.5 \mu g/m^3$) of the primary Lead NAAQS should be decreased substantially and that appropriate adjustments probably also should be made in the indicator, averaging time, and statistical form of the primary NAAQS for lead.

The scientific evidence on ecological and environmental effects of lead summarized in the Draft Lead Staff Paper and the Draft Lead Exposure and Risk Assessments documents indicate that any significant decrease in the present level of the primary Lead NAAQS will very likely have similarly significant beneficial effects on the magnitude of lead exposures in the environment and lead toxicity impacts on natural and managed terrestrial and aquatic ecosystems in various regions of the U.S., Canada, Mexico, the Great Lakes, and also in the open-water regions of the Atlantic Ocean.

Since concentrations of historically deposited lead in soils throughout the United States (averaging 0.5 to 4 grams/m^2 of land area) are changing only slowly — with a half-time exceeding a century — these concentrated deposits of lead are expected to remain accessible for exchange with the atmosphere and the rest of the biosphere into the foreseeable future. Fires, changes in land use, or climatic events such as regional dust storms could mobilize significant quantities of lead that would be harmful both to human health and ecosystems downwind. This potential for harm is not adequately recognized in the present Draft Lead Staff Paper and the Draft Lead Exposure and Risk Assessments documents.

Considering the magnitude of important ecological effects of lead in the environment, as described in these document, it is very disappointing to note that the EPA apparently lacks (or chooses not to expend) funds for any additional ecological risk assessment work for this current (2006–2008) review of the NAAQS for lead. This disappointment also is increased by the very welcome attention given in the Final Lead AQCD to the alternative concepts of critical loads, critical limits, target loads, and target times that have been developed in Europe and Canada to guide the processes of decision making regarding both environmental and public health effects of airborne chemicals.

Although these alternative concepts and processes of analysis of multiple pollutant/ multiple effects have not been carefully considered for use in the U. S., the CASAC Lead Review Panel — together with the authors of the National Research Council (NRC)/National Academy of Sciences (NAS) 2004 report on “Air Quality Management in the United States” — believes that these alternatives should be considered very carefully in the future as air quality management tools for use in this country as well as in other countries around the world.

ATTACHMENT B

Clean Air Scientific Advisory Committee Letter

(September 27, 2007)

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON D.C. 20460

OFFICE OF THE ADMINISTRATOR
SCIENCE ADVISORY BOARD

September 27, 2007

EPA-CASAC-07-007

Honorable Stephen L. Johnson
Administrator
U.S. Environmental Protection Agency
1200 Pennsylvania Avenue, NW
Washington, DC 20460

Subject: Clean Air Scientific Advisory Committee's (CASAC) Review of the 2nd Draft Lead Human Exposure and Health Risk Assessments Document

Dear Administrator Johnson:

The Clean Air Scientific Advisory Committee (CASAC or Committee), augmented by subject-matter-expert Panelists — collectively referred to as the CASAC Lead Review Panel (Lead Panel) — met on August 28–29, 2007, in Durham, NC, at the request of EPA's Office of Air Quality Planning and Standards (OAQPS) to conduct a peer review of the Agency's *Lead Human Exposure and Health Risk Assessments for Selected Case Studies, Draft Report* (2nd Draft Lead Human Exposure and Health Risk Assessments, July 2007). This letter provides the Lead Panel's advice and recommendations to you concerning the Agency's exposure/risk assessment that supports the setting of a primary National Ambient Air Quality Standard (NAAQS) for Lead, the secondary Lead NAAQS, and implementation issues associated with EPA's revised NAAQS review process. The CASAC roster is found in Appendix A of this report, and the Lead Panel roster is attached as Appendix B. The charge questions provided to the Lead Panel by EPA staff are contained in Appendix C to this report, and Panelists' individual review comments are provided in Appendix D.

Review of EPA's 2nd Draft Human Lead Exposure and Health Risk Assessments

1. Overall Evaluation

Overall, the CASAC Lead Review Panel judges that *the Agency's 2nd Draft Lead Human Exposure and Health Risk Assessments is not yet a complete, well-documented exposure and risk assessment that presents the full range of pertinent data and analyses.* In particular, in order to support the establishment of National Ambient Air Quality Standards, it is especially important that the EPA develop exposure estimates that will have *national* implications for, and relevance to, urban areas. Agency staff needs to undertake additional case studies of several urban locales

with varying lead exposure levels. More generally, in the view of the Committee, the second draft exposure/risk assessments document was missing certain critical information and is deemed incomplete, which is described in greater detail below. *Therefore, this EPA document, while representing a worthy effort by Agency staff, is not yet deemed to be adequate for regulatory decision-making.* The CASAC looks forward to reviewing OAQPS' Final Lead Exposure and Risk Assessments document — and, especially, the Final Staff Paper for Lead — when these are released by EPA in early November 2007.

2. Reiteration of CASAC Support for Continuing to List Lead as a Criteria Air Pollutant and the Lead Panel's Preliminary Analyses Concerning the Level of a Primary (Health-Based) NAAQS for Lead

Before discussing the Lead Panel's review of the EPA's 2nd Draft Lead Exposure and Risk Assessments document, *the Committee wishes to strongly reiterate its opposition to any considered de-listing of Lead as a criteria air pollutant and its concomitant and unanimous support for maintaining fully-protective NAAQS.* The details of the CASAC's rationale for this recommendation are contained in the Committee's March 27, 2007 letter to you concerning the Lead Panel's review of the Agency's 1st Draft Lead Staff Paper and the Draft Lead Exposure and Risk Assessments documents (EPA-CASAC-07-003).

Furthermore, as described in detail in Appendix D of the Committee's March 2007 letter/report, the Lead Panel previously considered three separate, though related, population-based analytical approaches aimed at deriving an estimated range of alternative levels for the primary Lead NAAQS. On the basis of the CASAC's preliminary scientific analyses and risk management assumptions, *EPA needs to substantially lower the level of the primary NAAQS for Lead, to 0.2 µg/m^3 or less. In the unanimous opinion of the Lead Panel, EPA has not presented any rigorous analyses or other information in its 2nd Draft Lead Exposure and Risk Assessments document that leads the CASAC to reconsider its previous recommendation to you that the upper limit the Agency should consider in revising the Pb NAAQS should be 0.2 µg/m^3 on a monthly average.*

3. Need Population-Based Risk Assessments of Urban Areas of National Significance

In the CASAC's previous letter to you on this topic (March 2007), the Lead Review Panel recommended using a "population-based" risk assessment to supplement the case-study approach used in the "pilot-phase" risk assessment. In addition, the Panel noted that a risk assessment of this type would typically include two key components:

(1) A quantitative description of the relationship between concentrations of lead in ambient air in various parts of the U.S. and resulting distributions of blood lead concentrations; and

(2) A quantitative description of the relationship between blood lead concentrations levels and impacts on IQ.

The Lead Panel was pleased to see that EPA introduced an *urban* model in the second draft of the exposure/risk assessments document, and further recommends that Agency staff focus on the *hybrid* urban model, using available information for several urban areas where there

are multiple monitors. The Committee recognizes that the nature of the national airborne lead database is limited by both its size (*i.e.*, a total of 189 PM-TSP [total suspended particulates] sites measuring ambient lead), as well as the selective location of monitors to mostly source-oriented sites. Some additional PM_{10} measurements are available (see the section below on the use of PM_{10} samplers to monitor lead).

In spite of these limitations, the CASAC strongly believes that it is important that EPA staff make estimates of exposure that will have *national* implications for, and relevance to, *urban* areas; and that, significantly, *the case studies of both primary lead (Pb) smelter sites as well as secondary smelter sites, while relevant to a few atypical locations, do not meet the needs of supporting a Lead NAA*QS. *The Agency should also undertake case studies of several urban areas with varying lead exposure concentrations*, based on the prototypic urban risk assessment that OAQPS produced in the 2nd Draft Lead Human Exposure and Health Risk Assessments. In order to estimate the magnitude of risk, the Agency should estimate exposures and convert these exposures to estimates of blood levels and IQ loss for children living in specific urban areas. In addition, the Agency should make IQ change estimates across the range of exposures to provide estimates for the change in median, as well as the 5th- to the 95th percentile of the population for different standards using the hybrid urban model with a geometric standard deviation (GSD) of 2.0 or 2.1.

The Lead Panel recognizes that there are few urban areas with multiple TSP monitors to estimate distributions of lead exposure. The Panel urges that PM_{10} monitors, with appropriate adjustments, be used to supplement the data. If necessary, other data with lead concentrations from special monitoring studies (*e.g.*, speciation studies of particulate matter [PM]) may provide estimates of the GSD of air lead over urban areas, which could also be used to supplement the limited TSP data. Discussion of the risk estimates obtained should carry appropriate caveats that document where estimates fall outside the range of data used to generate the estimates. EPA should also provide a qualitative discussion of how typical the chosen cities are of the range of what is seen in a broad spectrum of U.S. urban areas.

4. Completeness of 2nd Draft Lead Exposure and Risk Assessments

In a more general sense, the CASAC considers that EPA's 2nd Draft Lead Exposure and Risk Assessments document was missing certain key information and analytical components and was therefore incomplete. Specifically, the Committee believes that a properly comprehensive exposure/risk assessment in support of reviewing either primary or secondary NAAQS should be accompanied by documentation that includes a discussion of the four, policy-relevant elements of selecting a specific NAAQS — that is, indicator variable, averaging time, statistical form, and ranges of alternative levels of the standard — along with analyses that model the impact of current ("as is") standards and any proposed alternatives (*i.e.*, a truly *quantitative* risk assessment). In addition, the Agency needs to provide details on both the scope of the exposure assessment and the results from the modeling, to include components that would describe: selection of urban areas to be modeled; the time periods and the populations that were modeled; and the results from modeling the current lead standard and proposed alternative NAAQS.

Furthermore, with respect to the primary (public-health based) Lead NAAQS, a complete public-health risk assessment should include documentation that provides appropriate details on

both the scope of the health risk model and the associated modeling results vis-à-vis: selection of health endpoint categories; selection of study areas; and air quality considerations for both the current NAAQS and any proposed alternative levels of the standard. Finally, the exposure/risk assessment should include a thorough discussion of the key uncertainties.

Accordingly, the CASAC requests that EPA tailor its future exposure/risk assessments to provide the above documentation and analyses for the CASAC's and the public's review during the comparable phase of the NAAQS review process — that is, *prior* to the Agency's issuance of the Policy Assessment (PA) in the form of an Advance Notice of Proposed Rulemaking (ANPR) — for any given criteria air pollutant.

5. Choice of Biokinetic Model and Steady-State Dose Metric for Both Dose-Response Functions and Use in the Full Risk Assessment

Agency staff selected the Integrated Exposure Uptake Biokinetic (IEUBK) model for lead in children as the preferred method for estimating blood lead as the dose metric in the risk assessment, based on its overall assessment of blood lead estimating methods. The Lead Panel concurs in both OAQPS' selection of the IEUBK model as the biokinetic modeling method for blood lead estimates and its detailed rationale for doing so. The rationale provided by EPA staff for its choice is, overall, scientifically-sound.

In addition, OAQPS selected concurrent blood lead estimates as the best expression for the dose parameter used in both the dose-response functions and the risk assessment. The Panel also concurs in both this selection by Agency staff and the scientific rationales for doing so, particularly its reliance on the findings of the international pooled analysis of many longitudinal studies of cognitive deficits at lower exposures (doses) reported in Lanphear *et al.* (2005).

6. Predicting IQ Changes Based on Concurrent Blood Lead Concentrations

The Panel recommends using the two-piece linear function for relating IQ alterations to current blood lead levels with a slope change or "hinge" point closer to 7.5 µg/dL than 10.82 µg/dL as used by EPA staff in the second draft exposure/risk assessments document. The higher value used by staff underestimates risk at lower blood Pb levels, where most of the population will be located. Epidemiologic data indicate that the slope of the line below 7.5 µg/dL is approximately minus three (-3) IQ decrements per 1 µg/dL blood lead and the vast majority of children in the U.S. have maximal baseline Pb blood levels below 7.5 µg/dL (Lanphear *et al.*, EHP 2005; MMWR 2005). On a population level, the mean increase in blood lead concentration from airborne lead would generally be up to, but not exceeding, a blood lead concentration of 7.5 µg/dL. This approach should also account for sensitive subpopulations of children.

7. Level and Averaging Time for Primary Lead Standard with a Margin of Safety

The most recent epidemiologic studies demonstrate a statistically significant relationship between blood Pb and IQ loss well below 5 µg/dL. The CASAC recognizes that lead is a multimedia pollutant and that most of the country is in compliance with the current Lead NAAQS of 1.5 $\mu g/m^3$. However, the risk analysis scenarios presented by EPA for current conditions using the Agency's hybrid dust model — which the Lead Panel judges to be the most scientifically-defensible and robust dust model currently available — show that the "recent" air exposure path-

way contributes anywhere from 28 to 57% of the total amount of ingested lead. Additionally, recent air exposures still contribute 27% under an alternative primary Pb NAAQS of 0.2 $\mu g/m^3$ maximum monthly average, and only fall to 13% under an alternative primary Lead standard of 0.05 $\mu g/m^3$ maximum monthly average. *Since there is no known threshold in the relationship between blood Pb and IQ loss, the level of the current primary Lead standard clearly provides no margin of safety from ambient air lead exposures.* However, nor would any lower primary Lead NAAQS level provide a margin of safety, and hence, the question becomes:

> What percentage of the population of children in various parts of the U.S. will suffer what amount of IQ loss and other harmful effects due to the contribution of air exposures to the overall toxicity of Pb?

EPA staff should identify what levels of the primary Lead NAAQS would be deemed as being adequately-protective of human health. As a preliminary target, staff should identify the level of the standard that would ensure that 95% or more of the children in the U.S. do not experience decreased IQ from exposure to ambient concentrations of recent airborne lead. As noted in the Committee's previous letter to you on this subject, target levels of IQ decrements that would be of great concern would be one to two (1–2) IQ points or more. After identifying such a level of the standard, Agency staff should investigate alternative levels around this level, including much lower levels, to provide guidance as to how alternative standards would lead to changes in health. For example, if the analyses conducted by EPA staff suggest that a 0.1 $\mu g/m^3$ standard would lead to a decrease in IQ of one point or less for 95% of the children in the U.S., staff should assess other levels of the standard near 0.1 $\mu g/m^3$, both above and below, as well as much lower levels, *e.g.*, on the order of 0.05 and 0.01 $\mu g/m^3$. Further, the Agency should provide additional analyses to adequately inform both the Administrator and CASAC as to how uncertainties impact the level of protectiveness of the proposed alternative standards.

8. Use of PM_{10} Samplers to Monitor for Airborne Lead

Another recommendation that the CASAC provided in its March 2007 letter was to consider use of PM_{10} samplers to monitor lead. A substantial reduction in the level of the Pb NAAQS, combined with a shortening of the averaging time from quarterly to monthly, will require increases in both the number of lead monitoring sites, as well as the frequency of sample collection. Improved sampling precision will also be needed as more locations fall closer to standards and to support future health assessments as ambient lead concentrations are further reduced. For these and other reasons outlined in our previous advisory letter, *the Lead Panel strongly encourages the Agency to consider revising the Pb reference method to allow sample collection by PM_{10}, rather than TSP samplers,* accompanied by analysis with low-cost multi-elemental techniques like X-Ray Fluorescence (XRF) or Inductively Coupled Plasma–Mass Spectroscopy (ICP-MS). (See EPA-CASAC-07-003 for additional details.)

The Lead Panel also recognizes the importance of coarse dust contributions to total Pb ingestion and acknowledge that TSP sampling is likely to capture additional very coarse particles which are excluded by PM_{10} samplers. However, the precision of TSP samplers is poor, the upper particle cut size varies widely as a function of wind speed and direction, and the spatial non-homogeneity of very coarse particles cannot be efficiently captured by a national monitoring network. Generally, it can be expected that PM_{10} Pb will represent a large fraction of, and be

highly correlated with TSP Pb. Ambient Lead data from the (few) collocated TSP and PM_{10} sites presented in the 1st Draft Pb Staff Paper exhibited a high correlation (r = 0.96), with slopes (PM_{10} Pb/TSP Pb) ranging from 0.85 to 1. A single quantitative adjustment factor could be developed from a short period of collocated sampling at multiple sites; or a PM_{10} Pb/TSP Pb "equivalency ratio" could be determined on a regional or site-specific basis.

9. Other, Non-IQ-Related Effects of Lead in Ambient Air

While the CASAC agrees with the Agency's choice of IQ alterations in young children as the priority health effect and population for the risk assessment, the Lead Panel cautions against focusing only on IQ loss (or gain). There are ramifications of lead exposure on other endpoints that have societal and individual implications of great importance. Neurological developmental and functional effects in children exposed to Pb can lead to negative and disruptive behaviors well into teenage years. Moreover, while the adult nervous system has long been recognized as a target of Pb toxicity, epidemiologic and experimental toxicology data are emerging that support the relationship between Pb exposure and increased adverse cardiovascular outcomes, including increased blood pressure, increased incidence of hypertension, and cardiovascular morbidity and mortality at lower and lower levels of exposure.

Secondary Lead NAAQS

The "pilot phase" lead exposure and risk assessment document addressed both human health and environmental effects, but the July 2007 "full-scale" exposure and risk assessment document is focused entirely on human exposures and health risks. Agency staff made it quite clear in the 1st Draft Lead Staff Paper that OAQPS did not anticipate having either sufficient funding or time available to perform additional quantitative ecological risk assessment work during this current review cycle for the NAAQS for Lead. Thus, EPA staff did not conduct a full-scale *ecological* risk assessment for this second draft exposure/risk assessments document. Nevertheless, *the CASAC requests that EPA revise the ecological portion of the "pilot-phase" lead risk assessment on the basis of Lead Panel members' individual review comments found in Appendix E of the Committee's March 2007 letter; and that this be reflected in the welfare-effects sections of both the Final Lead Exposure and Risk Assessments document and the Final Lead Staff Paper.*

With respect to secondary Pb standards, the Lead Panel notes that the secondary Lead NAAQS was initially set equal to the primary Lead standard in 1978 "due to a lack of relevant data at that time." Nearly 30 years later, it now appears that *the Agency still lacks the relevant data to provide a clear, quantitative basis for setting a secondary Pb NAAQS that differs from the primary in indicator, averaging time, level or form.* To collect such data for the next Lead NAAQS review cycle, *the EPA needs to initiate new measurement activities in rural areas* — including those that are remote, close to urban and other sources, and located at high elevations — which quantify and track changes in lead concentrations in the ambient air, soils, deposition, surface waters, sediments and biota, along with other information as may be needed to calculate and apply a critical loads approach for assessing environmental lead exposures and risks in the next review cycle. Depending on the results of these Pb monitoring activities, the Agency may need to set the level of the secondary Lead NAAQS as a to-be-determined fraction of the level of

the primary standard, given the likelihood that many of the millions of animal and plant species are more sensitive to environmental lead pollution than are humans.

Importantly, EPA needs to move away from the traditional practice of simply setting a secondary Lead NAAQS that is *equal* to the primary standard — a practice that may technically meet the Clean Air Act requirements but has no scientific or technical basis. Nevertheless, in the *absence* of essential monitoring or other research data, the Lead Panel continues to recommend that, "at a minimum, the level of *the secondary Lead NAAQS should be at least as low as the lowest-recommended primary lead standard*," as the CASAC wrote in its previous letter to you on this subject. Furthermore, the Panel also continues to recommend that the Agency "*identify the necessary funds to support needed continuing research on the ecological effects of airborne lead pollution and to consider developing alternative secondary standards such as critical loads for lead, which may be different from primary standards in indicator, averaging time, level or form*." (For additional details, see the individual written comments of Drs. Ellis Cowling and Samuel Luoma found in Appendix D; and also see EPA-CASAC-07-003 and Lead Panelists' individual written comments found in Appendix E.)

The large environmental burden of historically-deposited lead is currently decreasing. Accordingly, *the goal should be to set the secondary Lead NAAQS such that there is no reversal of the current downward trend in lead concentrations in the environment.* The limited funds available for monitoring environmental lead should be focused on this critical task.

Comments on Implementation of the Agency's Revised NAAQS Review Process

The review of the NAAQS for ambient Lead has been a *hybrid* process, which began under the EPA's long-standing NAAQS review process and has since evolved into the Agency's new, revised process. Specifically, the Lead Panel in 2006 conducted a peer-review of the 1st and 2nd Drafts of EPA's Lead Air Quality Criteria Document (AQCD) and, in February 2007, reviewed OAQPS' 1st Draft Lead Staff Paper and the Draft Lead Exposure and Risk Assessments documents. The understanding of the CASAC at the time of the February review was that, since the Agency was transitioning to the revised review process midway through the current Lead NAAQS review, this was to be the last version of either of these documents that OAQPS would develop and that the Lead Panel would have an opportunity to review. Indeed, in the CASAC's last letter to you on this subject (March 27, 2007), the Panel expressed its concern that, in the absence of being given an opportunity to review even a 2nd Draft Lead Staff Paper, or to review a second draft or a final lead exposure/risk assessment document, prior to EPA's issuance of the Lead Policy Assessment in the form of an ANPR, the Committee would not have the information — that is, both the data and the analyses — needed to properly advise you concerning the setting of NAAQS for Lead that would be adequately-protective of both human health and welfare. In response to CASAC's concerns, the Agency agreed to produce a second draft of the exposure/ risk assessments document, the peer-review of which is the topic of this letter/report.

However, immediately prior to our August 28–29 public advisory meeting on the 2nd Draft Lead Human Exposure and Health Risk Assessments, OAQPS staff informed me as the CASAC Chair that a recent (August 24, 2007) Federal court order was requiring the Agency to produce a Final Lead Staff Paper by the previously-agreed-upon date of November 1, 2007 —

and, thus, "the pendulum has swung back" to the former NAAQS review process, at least until the issuance of the Lead PA by means of an ANPR no later than November 30, 2007. Although this seesaw process has admittedly been both confusing and vexing, the Committee is looking forward to reviewing the Final Lead Exposure and Risk Assessments document, the Final Staff Paper for Lead, and the Lead PA in a meeting to be held in mid-December 2007.

Both these process-related perturbations and, as detailed above, the absence of certain critical information in the Agency's 2nd Draft Lead Exposure and Risk Assessments document — which as previously noted was intended to be the CASAC's final source of information from EPA with respect to the review of the NAAQS for Lead prior to the issuance of a Policy Assessment in an ANPR — underscore the Committee's concerns about the Agency's revised NAAQS review process as it is presently being implemented and may be implemented in the future. The CASAC has a statutory mandate to provide the EPA with expert advice and recommendations on scientifically-appropriate standards for criteria air pollutants. In order to be able to fulfill this, it is axiomatic that the CASAC must receive, in a timely manner, and be afforded an opportunity to review and comment on the complete suite of relevant risk- and exposure-related data and analyses that will presumably underpin the Agency's regulatory decisions — not only for the Lead standards but also for forthcoming risk/exposure assessments associated with the NAAQS reviews for other criteria pollutants.

In closing, the CASAC is pleased to advise you and OAQPS staff on the 2nd Draft Lead Human Exposure and Health Risk Assessments document. As both EPA and the Committee continue to work through the details associated with implementation of the revised NAAQS review process, we would ask that the Agency ensure that the CASAC receive the full breadth of information and supporting analyses necessary to provide timely, expert advice and recommendations to the EPA. As always, we wish Agency staff well in this important task.

Sincerely,

/Signed/

Dr. Rogene Henderson, Chair
Clean Air Scientific Advisory Committee

Appendices (A–D)

NOTICE

This report has been written as part of the activities of the U.S. Environmental Protection Agency's (EPA) Clean Air Scientific Advisory Committee (CASAC), a Federal advisory committee administratively-located under the EPA Science Advisory Board (SAB) Staff Office that is chartered to provide extramural scientific information and advice to the Administrator and other officials of the EPA. The CASAC is structured to provide balanced, expert assessment of scientific matters related to issue and problems facing the Agency. This report has not been reviewed for approval by the Agency and, hence, the contents of this report do not necessarily represent the views and policies of the EPA, nor of other agencies in the Executive Branch of the Federal government, nor does mention of trade names or commercial products constitute a recommendation for use. CASAC reports are posted on the SAB Web site at: http://www.epa.gov/sab.

Appendix A – Roster of the Clean Air Scientific Advisory Committee

U.S. Environmental Protection Agency
Science Advisory Board (SAB) Staff Office
Clean Air Scientific Advisory Committee (CASAC)

CHAIR

Dr. Rogene Henderson, Scientist Emeritus, Lovelace Respiratory Research Institute, Albuquerque, NM

MEMBERS

Dr. Ellis Cowling, University Distinguished Professor At-Large, North Carolina State University, Colleges of Natural Resources and Agriculture and Life Sciences, North Carolina State University, Raleigh, NC

Dr. James D. Crapo, Professor, Department of Medicine, National Jewish Medical and Research Center, Denver, CO

Dr. Douglas Crawford-Brown, Director, Carolina Environmental Program; Professor, Environmental Sciences and Engineering; and Professor, Public Policy, Department of Environmental Sciences and Engineering, University of North Carolina at Chapel Hill, Chapel Hill, NC

Mr. Richard L. Poirot, Environmental Analyst, Air Pollution Control Division, Department of Environmental Conservation, Vermont Agency of Natural Resources, Waterbury, VT

Dr. Armistead (Ted) Russell, Georgia Power Distinguished Professor of Environmental Engineering, Environmental Engineering Group, School of Civil and Environmental Engineering, Georgia Institute of Technology, Atlanta, GA

Dr. Frank Speizer, Edward Kass Professor of Medicine, Channing Laboratory, Harvard Medical School, Boston, MA

SCIENCE ADVISORY BOARD STAFF

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Appendix B – Roster of the CASAC Lead Review Panel

U.S. Environmental Protection Agency
Science Advisory Board (SAB) Staff Office
Clean Air Scientific Advisory Committee (CASAC)
CASAC Lead Review Panel

CHAIR

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MEMBERS

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Dr. Bruce Fowler, Assistant Director for Science, Division of Toxicology and Environmental Medicine, Office of the Director, Agency for Toxic Substances and Disease Registry, U.S. Centers for Disease Control and Prevention (ATSDR/CDC), Chamblee, GA

Dr. Andrew Friedland, Professor and Chair, Environmental Studies Program, Dartmouth College, Hanover, NH

Dr. Robert Goyer [M.D.], Emeritus Professor of Pathology, Faculty of Medicine, University of Western Ontario (Canada), Chapel Hill, NC

Mr. Sean Hays, President, Summit Toxicology, Allenspark, CO

Dr. Bruce Lanphear [M.D.], Sloan Professor of Children's Environmental Health, and the Director of the Cincinnati Children's Environmental Health Center at Cincinnati Children's Hospital Medical Center and the University of Cincinnati, Cincinnati, OH

Dr. Samuel Luoma, Senior Research Hydrologist, U.S. Geological Survey (USGS), Menlo Park, CA

Dr. Frederick J. Miller, Consultant, Cary, NC

Dr. Paul Mushak, Principal, PB Associates, and Visiting Professor, Albert Einstein College of Medicine (New York, NY), Durham, NC

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APPENDIX 2A:

LARGEST STATIONARY SOURCE CATEGORIES FOR Pb IN THE 2002 NEI

Appendix 2A. Largest Stationary Source Categories for Pb in the 2002 NEI.

Boilers and Process Heaters

Materials including coal, oil, natural gas (or, at times, other substances such as wood and petroleum coke) are burned in boilers and process heaters to produce steam. With regard to boilers, the steam is used to produce electricity or provide heat, while process heaters are used in industrial processes. Lead is present naturally in the fuel and is emitted to air following combustion. The extent of emissions depends on the concentration of Pb in the fuel, the quantity of fuel burned, and PM control devices applied.

Industrial, commercial and institutional boilers and process heaters are used at a wide variety of facilities (e.g., refineries, chemical and manufacturing plants, etc), as well as in a "stand alone" mode to provide heat for large building complexes. Consequently, there are thousands of these sources throughout the country, generally located in urban areas, and they range widely in size. Most coal-fired industrial boilers emit about 0.06 tpy, with the larger ones emitting about 0.07 tpy due to the use of high efficiency particulate matter (PM) control devices (ERG, 2002a). [

Among utility boilers, coal-fired boilers have the highest Pb emissions, oil-fired utility plants emit somewhat lower amounts, and gas-fired plants emit very low levels of Pb (USEPA, 1998). There are approximately 1,300 coal-fired electric utility boilers in the U.S. ranging in size from 25 to approximately 1,400 MWe. Based on emission factor calculations, a 325 MWe coal-fired boiler would be expected to emit approximately 0.021 tpy Pb, based on the use of an electrostatic precipitator for PM control (USEPA, 1998). Although there are exceptions, coal-fired utility boilers tend to be located in non-urban areas.

Iron and Steel Foundries

Iron and steel foundries melt scrap, ingot, and other forms of iron and steel and pour the molten metal into molds for particular products. While located in 44 of the lower 48 states (in both cities and rural areas), the 650 existing foundries in the U.S., are most heavily concentrated in the Midwest (IN, IL, OH, MI, WI, and MN) - roughly 40% of foundries with almost 60% of U.S. production (USEPA, 2002a). Most are iron foundries operated by manufacturers of automobiles and large industrial equipment and their suppliers. The largest Pb emission sources at iron foundries are large furnaces, emissions from which generally range from about 0.3 to 3 tpy (generally released at heights of 25-30 feet), depending on the throughput of the furnace, the type and operating characteristics of the emission control system, and the Pb content in the metal charged to the furnace. Regulations promulgated in 2004 are projected to yield emissions reductions of approximately 25 tpy for this category (USEPA, 2004b).

Hazardous Waste Incineration/ Combustion Facilities

Hazardous waste combustors include hazardous waste incinerators, as well as boilers and industrial furnaces that burn hazardous waste for energy or material recovery (e.g., production of halogen acid from the combustion of chlorine-bearing materials). Industrial furnaces burning hazardous waste include cement kilns, lightweight aggregate kilns, and hydrochloric acid production furnaces. Lead is a trace contaminant in the hazardous waste, fossil fuels, and raw materials used in the combustors. In 2005, there were nearly 270 hazardous waste combustor sources in operation in the United States (70 FR at 59530), with approximately 40 percent of them in the states of Texas and Louisiana. As a result of emissions standards promulgated in 2005, EPA estimates that cumulative Pb emissions from hazardous waste combustors will be reduced to approximately 4.0 tons per year by the compliance date in 2008 (USEPA, 2005), a 95% reduction from 1990 levels.

Primary Lead Smelting

At primary Pb smelters, Pb-bearing ore concentrates are smelted to produce Pb metal. Lead is emitted from primary Pb smelters as process emissions, process fugitive emissions, and fugitive dust emissions (CD, p. 2-21). U.S. EPA promulgated a national emissions standard in 1999 for this category which includes an emissions limit for Pb (U.S. EPA 1999a). In the 1990s, there were three operating primary Pb smelters in the U.S: one in Montana and two in Missouri, emitting an estimated total of about 260 tpy Pb. In 2002, there were two in operation (estimated emissions shown in Table A-1); one of the two had less than 1 tpy Pb emissions. As of 2004, there was only one operating primary Pb smelter in the U.S.,

located in Missouri with estimated total emissions of about 28 tpy in year 2005 (CD, p. 2-20). Thus, total Pb emissions from this category have decreased about 90% since 1990.
Secondary Lead Smelting Secondary Pb smelters are recycling facilities that use blast, rotary, reverberatory, and/or electric furnaces to recover Pb metal from Pb-bearing scrap materials, primarily Pb-acid batteries. This category does not include remelters and refiners or primary Pb smelters. At secondary Pb smelters, Pb may be emitted from process emissions, process fugitive emissions and fugitive dust emissions from wind or mechanically induced entrainment of dust from stockpile and plant yards and roadways. In 1995, U.S. EPA promulgated a national emissions standard for this category which includes an emissions limit for Pb (USEPA, 1995). In 2002, there were 15 secondary smelters operating in 11 states, most of which are in the eastern half of the U.S. Estimates of total emissions (process and fugitive) for individual facilities as of 2002 range between 1 and 4 tpy, with one facility having total lead emissions of about 12 tpy (USEPA, 2007a; EC/R, 2006). Total Pb emissions (tpy) for this category decreased about 60% from 1990 to 2002.
Military Installations This source category includes sources that are military facilities. The types of sources contributing to Pb emissions from this category include, among others, rocket and engine test facilities, ammunition manufacturing, weapons testing, waste combustion and boilers. While there are over 300 military facilities in the NEI, only 10% emit over 0.1 tpy of Pb and only 3% emit over 1 tpy. The two largest facilities (listed in Table A-4) are a missile ammunition production plant and a weapons testing facility and these two facilities account for over 75% of the category emissions.
Mining This category includes various mining facilities that extract ore from the earth containing Pb, zinc, copper and/or other non-ferrous metals (such as gold and silver), and/or non-metallic minerals such as talc and coal. This category does not include the smelting or refining of the metals and minerals. These facilities produce ore concentrates (such as Pb, zinc, and copper concentrates) that are transported to other facilities where further processes, such as smelting and refining take place. The 2002 NEI indicates that there are 3 mining facilities in the U.S. emitting greater than 0.5 tpy Pb, one of which emits more than 5 tpy. This facility is in Missouri and produces Pb, zinc, and copper concentrates that are shipped to customers for further processing.
Integrated Iron & Steel Manufacturing Integrated iron and steel manufacturing includes facilities engaged in the production of steel from iron ore. The processes include sinter plants, blast furnaces that produce iron, and basic oxygen process furnaces that produce steel, as well as several ancillary processes including hot metal transfer, desulfurization, slag skimming, and ladle metallurgy. There are currently 17 facilities in this source category each of whom emit from 2 to 8 tpy of Pb. Stack heights range from 30 - 50 feet. The facilities are located in 9 states, mostly in the Midwest (USEPA, 2003a). EPA promulgated a national emissions standard in 2003 for this category which includes an emissions limit for PM (as a surrogate for metal HAP, including Pb) (USEPA, 2003b).
Municipal Waste Combustors: Small & Large Municipal waste combustors (MWCs) incinerate municipal or municipal-type solid waste. The amount of municipal waste incinerated (about 14% of U.S. municipal waste) has remained stable over the past decade. The amount of Pb emitted from municipal waste combustors depends on the amount of Pb in the refuse, with typical sources including paper, inks, cans and other metal scrap and plastics (CD, pp. 2-35 to 2-36). As of 2005, MACT standards were completed for all existing and new municipal waste incineration units, resulting in nationwide Pb emissions of less than 10 tons per year, roughly a 97% reduction since 1990. There are currently 66 large MWC plants and 26 small MWC plants operating nationally, with individual large MWC plants projected to emit less than 0.1 tpy Pb, and small MWC plants less than 0.02 tpy Pb (ERG, 2002b,c; Stevenson, 2002). However, there are a few MWC facilities that emit about 2 tons per year.
Pressed and Blown Glass and Glassware Manufacturing This category includes manufacturers of flat glass, glass containers, and other pressed and blown glass and glassware, with Pb emitted primarily from the pressed and blown glass industry sector. Some container plants also make a leaded-glass product, but this is not typical of container glass plants. Lead

may also be added to flat glass for use in microwaves and flat-screen TVs. Emissions from individual facilities may range from a few pounds per year up to several tons per year depending on Pb content of their glass and the level of control. Furnace stacks for these facilities are typically of the order of 35-60 feet high. As of 2005, about 22 tons of Pb is emitted from glass manufacturing annually in the U.S. Glass plants are located in 35 States (RTI, 2006). U.S. EPA is currently developing an emissions regulation for this category, scheduled for promulgation in December 2007.

Electric Arc Furnace Steelmaking

In the steelmaking process that uses an electric arc furnace (EAF), the primary raw material is scrap metal, which is melted and refined using electric energy. Since scrap metal is used instead of molten iron, there are no cokemaking or ironmaking operations associated with steel production that use an EAF. There are currently 141 EAFs at 93 facilities, with estimated total nationwide Pb and Pb compound emissions of approximately 80 tons, and the average per facility is approximately 0.75 tpy. Stack heights range from heights of 30 - 50 feet. The facilities are located in 32 states; mostly in the northeast and Midwest, with ninety percent of the facilities located in urban areas. This information is drawn from multiple sources (Lehigh, 1982; Calspan, 1977; RTI, 2005). U.S. EPA is developing a hazardous air pollutant (HAP) emissions regulation for this category, scheduled for promulgation in December 2007.

Lead Acid Battery Manufacturing

The Pb acid battery manufacturing category includes establishments primarily engaged in manufacturing storage batteries from Pb alloy ingots and Pb oxide. The Pb oxide may be prepared by the battery manufacturer or may be purchased from a supplier. There has been a general decline in number of facilities, with 58 facilities currently in operation (data obtained from the Battery Council International (BCI)). The estimated range of facility-specific Pb and Pb compound emissions is from 1×10^{-5} to just below 10 tpy, with an average of about 0.5 tpy. The facilities are located in urban and rural areas of 23 states and Puerto Rico (2002 NEI).

Primary Copper Smelting

This source category includes all industries which refine copper concentrate from mined ore to anode grade copper, using pyrometallic processes. Seven primary copper smelters are currently operating in the U.S. Six of these seven smelters use conventional smelter technology which includes batch converter furnaces for the conversion of matte grade copper to blister copper, while the seventh uses a continuous flash furnace. Two of the three largest smelters are located in AZ, and the third is in Utah. The largest facility emitted an estimated 12.8 tons Pb in 2002, while emissions for the other two large facilities are estimated to be between 0.1 to 5 tpy. No other source in this category emits more than 0.1 tpy. In 2002, U.S. EPA promulgated a national emissions standard, including limits for PM (as a surrogate for metal HAP, including Pb), for this category (USEPA, 2002c).

Portland Cement Manufacturing

Portland cement manufacturing is an energy intensive process in which cement is made by grinding and heating a mixture of raw materials such as limestone, clay, sand, and iron ore in a rotary kiln (a large furnace fueled by coal, oil, gas, coke and/or various waste materials such as tires). Lead, a trace contaminant both of the raw materials and some fuel materials (e.g., coal, tires), is emitted with particulate material from the kiln stacks, which range in height from approximately 10 meters to more than 100 meters. Relatively smaller Pb emissions occur from grinding, cooling, and materials handling steps in the manufacturing process. These facilities are generally located in areas with limestone deposits and in rural areas or near small towns. The largest numbers of facilities are in Pennsylvania and California, although a significant percentage of facilities are in the Midwest. As of 2004, there were 107 Portland cement plants in the U.S. (O'Hare, 2006), with all but three reporting less than 1 tpy of Pb emissions. The highest estimated Pb emissions for a facility in the 2002 NEI is 5.4 tpy. In 1999, U.S. EPA promulgated a national emissions standard, including a limit for PM (as a surrogate for metal HAP, including Pb), for this category (USEPA, 1999b).

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APPENDIX 2B:

ADDITIONAL DETAILS OF AIR QUALITY ANALYSES

Appendix 2B

Table 2B-1. Pb-TSP monitoring site information and 3-year statistics

site	poc	lat	long	state	county_name	cbsa_name	cbsa_pop00	population near site (mile radius)	under age 5 pop. (mile radius)	urban	sum point / nonpt Pb EI TPY w/in 1 mile	source oriented?	prev. source oriented? (see end notes)	3-year data capture (complete periods)			3-year metrics					
														comp. years	comp. qtrs	comp. months	annual mean	max quarterly mean	max monthly mean	2nd max monthly mean	average of 3 overall highest monthly means	average of 3 annual max monthly means
011090003	2	31.79056	-85.97917	AL	Pike	Troy, AL	29,605	461	31		4.5	1		2	10	31	0.6875	1.9233	2.6600	2.4200	2.2867	1.6852
011090006	1	31.79278	-85.98056	AL	Pike	Troy, AL	29,605	461	31		4.5	1		2	10	31	0.3808	0.9100	1.6900	1.3400	1.3233	1.0901
060250005	1	32.67611	-115.48333	CA	Imperial	El Centro, CA	142,361	16,385	1,290	1	0.0			2	11	34	0.0175	0.0248	0.0404	0.0380	0.0380	0.0330
060371103	2	34.06659	-118.22688	CA	Los Angeles	Los Angeles-Long Beach-Santa Ana	12,365,627	29,329	1,633	1	0.3			3	12	36	0.0225	0.0627	0.1460	0.0280	0.0673	0.0663
060371301	1	33.92899	-118.21071	CA	Los Angeles	Los Angeles-Long Beach-Santa Ana	12,365,627	47,423	5,066	1	0.0			3	12	34	0.0188	0.0313	0.0440	0.0360	0.0380	0.0353
060371601	1	34.01407	-118.06056	CA	Los Angeles	Los Angeles-Long Beach-Santa Ana	12,365,627	13,333	1,066	1	0.0			2	9	27	0.0186	0.0300	0.0480	0.0340	0.0373	0.0343
060374002	2	33.82376	-118.18921	CA	Los Angeles	Los Angeles-Long Beach-Santa Ana	12,365,627	20,131	1,232	1	0.0			3	12	36	0.0149	0.0400	0.0960	0.0440	0.0552	0.0427
060374004	2	33.79236	-118.17533	CA	Los Angeles	Los Angeles-Long Beach-Santa Ana	12,365,627	61,497	6,697	1	0.0			2	10	28	0.0112	0.0938	0.1020	0.0840	0.0673	0.0447
060375001	1	33.92288	-118.37026	CA	Los Angeles	Los Angeles-Long Beach-Santa Ana	12,365,627	19,148	1,680	1	0.0			1	5	14	0.0222	0.0667	0.1700	0.0220	0.0693	0.0910
060375005	1	33.95080	-118.43043	CA	Los Angeles	Los Angeles-Long Beach-Santa Ana	12,365,627	33,968	1,358	1	0.0			1	7	17	0.0057	0.0118	0.0150	0.0120	0.0123	0.0135
060651003	2	33.94603	-117.40063	CA	Riverside	Riverside-San Bernardino-Ontario, C	3,254,821	16,320	1,278	1	0.0			3	12	36	0.0097	0.0114	0.0160	0.0140	0.0147	0.0147
060658001	3	33.99958	-117.41601	CA	Riverside	Riverside-San Bernardino-Ontario, C	3,254,821	16,247	1,678	1	0.0			3	12	35	0.0121	0.0179	0.0220	0.0220	0.0213	0.0213
060711004	1	34.10374	-117.62914	CA	San Bernardino	Riverside-San Bernardino-Ontario, C	3,254,821	18,777	1,578	1	0.0			3	12	35	0.0142	0.0343	0.0800	0.0200	0.0394	0.0387
060719004	1	34.10688	-117.27411	CA	San Bernardino	Riverside-San Bernardino-Ontario, C	3,254,821	14,861	1,755	1	0.0			3	12	36	0.0186	0.0773	0.1420	0.0680	0.0873	0.0580
080010005	1	39.79601	-104.97754	CO	Adams	Denver-Aurora, CO	2,157,756	2,025	183		1.9	1		3	12	36	0.1697	0.5558	1.1037	0.4397	0.6195	0.5148
080010006	1	39.82574	-104.93699	CO	Adams	Denver-Aurora, CO	2,157,756	3,313	256	1	0.0			3	12	31	0.0304	0.0957	0.2086	0.0726	0.1085	0.1085
080310002	4	39.75119	-104.98762	CO	Denver	Denver-Aurora, CO	2,157,756	22,019	974	1	0.0			3	12	34	0.0315	0.1780	0.2955	0.2297	0.1906	0.1254
080310015	1	39.70012	-104.98714	CO	Denver	Denver-Aurora, CO	2,157,756	14,438	809	1	0.0			1	7	20	0.0153	0.0212	0.0305	0.0196	0.0228	0.0244
080410011	1	38.83139	-104.82778	CO	El Paso	Colorado Springs, CO	537,484	10,581	552	1	0.0			3	12	35	0.0156	0.0891	0.1387	0.1314	0.0955	0.0551
080650001	1	39.24778	-106.29139	CO	Lake	Edwards, CO	49,471	5,903	361	1	0.0			2	11	28	0.0165	0.0224	0.0310	0.0310	0.0305	0.0294
100010002	1	38.98472	-75.55556	DE	Kent	Dover, DE	126,697	352	22		0.0			1	4	12	0.0033	0.0040	0.0051	0.0041	0.0044	0.0051
100031007	1	39.55111	-75.73083	DE	New Castle	Philadelphia-Camden-Wilmington, I	5,687,147	2,041	209		0.0			1	4	10	0.0039	0.0046	0.0058	0.0051	0.0054	0.0058
100031008	1	39.57778	-75.61111	DE	New Castle	Philadelphia-Camden-Wilmington, I	5,687,147	3,170	160		0.0			1	4	9	0.0052	0.0063	0.0081	0.0058	0.0065	0.0081
100032004	1	39.73944	-75.55806	DE	New Castle	Philadelphia-Camden-Wilmington, I	5,687,147	34,053	2,649	1	0.0			1	4	11	0.0097	0.0115	0.0163	0.0161	0.0142	0.0163
100051002	1	38.64444	-75.61306	DE	Sussex	Seaford, DE	156,638	5,450	390	1	0.0			1	4	12	0.0033	0.0042	0.0048	0.0042	0.0043	0.0048
120571065	5	27.89222	-82.53861	FL	Hillsborough	Tampa-St. Petersburg-Clearwater, F	2,395,997	14,463	612	1	0.0			1	4	12	0.0049	0.0062	0.0094	0.0080	0.0082	0.0094
120571066	1	27.96028	-82.38250	FL	Hillsborough	Tampa-St. Petersburg-Clearwater, F	2,395,997	5,793	465	1	1.3	1		3	12	35	0.5835	1.2600	1.7400	1.3800	1.4733	1.4733
120571073	1	27.96583	-82.37944	FL	Hillsborough	Tampa-St. Petersburg-Clearwater, F	2,395,997	4,541	340	1	1.3	1		3	12	35	0.1934	0.2933	0.4800	0.4400	0.4467	0.4133
120571075	5	28.05000	-82.37806	FL	Hillsborough	Tampa-St. Petersburg-Clearwater, F	2,395,997	10,691	490	1	0.0			1	4	12	0.0041	0.0054	0.0105	0.0072	0.0075	0.0105
121030004	5	27.94639	-82.73194	FL	Pinellas	Tampa-St. Petersburg-Clearwater, F	2,395,997	13,048	557	1	0.0			1	4	12	0.0028	0.0041	0.0067	0.0039	0.0048	0.0067
121030018	5	27.78556	-82.74000	FL	Pinellas	Tampa-St. Petersburg-Clearwater, F	2,395,997	11,289	571	1	0.0			2	8	24	0.0042	0.0071	0.0112	0.0103	0.0103	0.0107
121033005	1	27.87583	-82.69639	FL	Pinellas	Tampa-St. Petersburg-Clearwater, F	2,395,997	2,151	58	1	0.0			3	12	36	0.0006	0.0067	0.0200	0.0000	0.0067	0.0067
130890003	2	33.69833	-84.27333	GA	DeKalb	Atlanta-Sandy Springs-Marietta, GA	4,247,981	7,888	663	1	0.0			3	12	36	0.1000	0.1000	0.1000	0.1000	0.1000	0.1000
132150011	1	32.43083	-84.93167	GA	Muscogee	Columbus, GA-AL	281,768	10,871	1,037	1	0.3	1	1	1	10	34	0.1000	0.1000	0.1000	0.1000	0.1000	0.1000
150032004	1	21.39667	-157.97167	HI	Honolulu	Honolulu, HI	876,156	23,622	1,207	1	0.1			3	12	35	0.0014	0.0029	0.0072	0.0025	0.0040	0.0038
170310001	1	41.67275	-87.73246	IL	Cook	Chicago-Naperville-Joliet, IL-IN-W	9,098,316	13,648	971	1	0.0			3	12	35	0.0143	0.0229	0.0360	0.0250	0.0270	0.0270
170310022	2	41.68920	-87.53932	IL	Cook	Chicago-Naperville-Joliet, IL-IN-W	9,098,316	22,040	1,708	1	0.2			3	12	36	0.0270	0.0353	0.0440	0.0420	0.0427	0.0407
170310026	1	41.87333	-87.64507	IL	Cook	Chicago-Naperville-Joliet, IL-IN-W	9,098,316	28,739	1,203	1	0.0			3	12	34	0.0405	0.0613	0.0900	0.0860	0.0820	0.0753
170310052	1	41.96743	-87.74982	IL	Cook	Chicago-Naperville-Joliet, IL-IN-W	9,098,316	42,187	2,877	1	0.0			3	12	32	0.0214	0.0260	0.0400	0.0380	0.0360	0.0353
170313103	1	41.96528	-87.87639	IL	Cook	Chicago-Naperville-Joliet, IL-IN-W	9,098,316	10,302	670	1	0.0			3	12	34	0.0149	0.0271	0.0440	0.0240	0.0307	0.0280
170313301	1	41.78278	-87.80528	IL	Cook	Chicago-Naperville-Joliet, IL-IN-W	9,098,316	23,749	1,678	1	0.0			3	12	35	0.0308	0.0750	0.1950	0.1140	0.1263	0.1155
170314201	1	42.14000	-87.79917	IL	Cook	Chicago-Naperville-Joliet, IL-IN-W	9,098,316	6,070	303	1	0.0			2	8	24	0.0113	0.0133	0.0175	0.0160	0.0165	0.0168
170316003	1	41.87194	-87.82611	IL	Cook	Chicago-Naperville-Joliet, IL-IN-W	9,098,316	14,862	1,071	1	0.0			3	12	32	0.0303	0.0387	0.0500	0.0480	0.0480	0.0480
171170002	2	39.39804	-89.80975	IL	Macoupin	St. Louis, MO-IL	2,721,491	40	2		0.0			3	12	36	0.0103	0.0113	0.0140	0.0140	0.0133	0.0133
171190010	1	38.69417	-90.15361	IL	Madison	St. Louis, MO-IL	2,721,491	8,014	529	1	1.3	1		3	12	34	0.0768	0.3280	0.9100	0.2880	0.4620	0.4620
171193007	2	38.86056	-90.10583	IL	Madison	St. Louis, MO-IL	2,721,491	5,397	360	1	0.1			3	12	36	0.0150	0.0193	0.0320	0.0240	0.0267	0.0262
171430037	1	40.69889	-89.58474	IL	Peoria	Peoria, IL	366,899	12,643	1,109	1	0.0			3	12	35	0.0137	0.0279	0.0320	0.0300	0.0300	0.0240
171630010	2	38.61222	-90.16028	IL	St. Clair	St. Louis, MO-IL	2,721,491	3,512	430	1	0.3			3	12	36	0.0433	0.0707	0.1050	0.0980	0.0990	0.0913
180350008	1	40.15806	-85.42111	IN	Delaware	Muncie, IN	118,769	2,108	104	1	0.0	1		3	12	34	0.2944	0.4657	0.7371	0.5991	0.6011	0.5585
180350009	2	40.15944	-85.41556	IN	Delaware	Muncie, IN	118,769	980	82		0.0	1		1	6	13	2.6732	4.0931	5.775	5.0220	4.2890	2.8611
180890023	1	41.65278	-87.43944	IN	Lake	Chicago-Naperville-Joliet, IL-IN-W	9,098,316	5,959	603	1	6.5	1		3	12	34	0.0389	0.0691	0.0910	0.0783	0.0786	0.0714
180892008	1	41.63944	-87.49361	IN	Lake	Chicago-Naperville-Joliet, IL-IN-W	9,098,316	7,144	612	1	0.0			3	12	33	0.0219	0.0296	0.0590	0.0484	0.0496	0.0496
180892011	2	41.59250	-87.47194	IN	Lake	Chicago-Naperville-Joliet, IL-IN-W	9,098,316	9,815	729	1	0.0			3	12	34	0.0368	0.1352	0.3050	0.0778	0.1522	0.1397
180930004	1	38.88944	-86.55194	IN	Lawrence	Bedford, IN	45,922	393	32		0.0			2	10	26	0.0270	0.0270	0.0270	0.0270	0.0270	0.0270

Appendix 2B

Table 2B-1. Pb-TSP monitoring site information and 3-year statistics

site	poc	lat	long	state	county_name	cbsa_name	cbsa_pop00	population near site (mile radius)	under age 5 pop. (mile radius)	urban	sum point / nonpt Pb EI TPY w/in 1 mile	source oriented?	prev. source oriented? (see end notes)	3-year data capture (complete periods)			3-year metrics					
														comp. years	comp. qtrs	comp. months	annual mean	max quarterly mean	max monthly mean	2nd max monthly mean	average of 3 overall highest monthly means	average of 3 annual max monthly means
180970063	1	39.76083	-86.29722	IN	Marion	Indianapolis-Carmel, IN	1,525,104	12,176	875	1	1.7	1		3	12	36	0.0320	0.0770	0.1123	0.0802	0.0854	0.0843
180970076	1	39.75889	-86.28972	IN	Marion	Indianapolis-Carmel, IN	1,525,104	9,171	602	1	1.7	1		3	12	35	0.0142	0.0254	0.0360	0.0346	0.0317	0.0251
180970078	1	39.81110	-86.11447	IN	Marion	Indianapolis-Carmel, IN	1,525,104	14,196	1,175	1	0.0			2	11	33	0.0108	0.0251	0.0288	0.0240	0.0251	0.0184
181010001	1	38.89028	-86.76083	IN	Martin			84	5		0.0			3	12	34	0.0272	0.0299	0.0358	0.0270	0.0299	0.0299
181630006	2	37.97167	-87.56722	IN	Vanderburgh	Evansville, IN-KY	342,815	13,666	817	1	0.0			3	12	33	0.0065	0.0126	0.0286	0.0170	0.0181	0.0150
260490021	4	43.04722	-83.67028	MI	Genesee	Flint, MI	436,141	9,889	994	1	0.0			3	12	36	0.0100	0.0153	0.0209	0.0189	0.0188	0.0185
261130001	1	44.31056	-84.89194	MI	Missaukee	Cadillac, MI	44,962	58	3		0.0			3	12	33	0.0032	0.0056	0.0080	0.0046	0.0057	0.0054
261630001	2	42.22861	-83.20833	MI	Wayne	Detroit-Warren-Livonia, MI	4,452,557	14,329	798	1	0.0			3	12	35	0.0087	0.0107	0.0124	0.0115	0.0116	0.0112
261630005	1	42.26722	-83.13222	MI	Wayne	Detroit-Warren-Livonia, MI	4,452,557	11,314	923	1	0.2			2	11	34	0.0166	0.0259	0.0340	0.0315	0.0322	0.0308
261630015	4	42.30278	-83.10667	MI	Wayne	Detroit-Warren-Livonia, MI	4,452,557	17,729	1,771	1	0.0			3	12	36	0.0178	0.0252	0.0299	0.0278	0.0278	0.0275
261630019	1	42.43083	-83.00028	MI	Wayne	Detroit-Warren-Livonia, MI	4,452,557	28,362	2,628	1	0.0			3	12	34	0.0103	0.0138	0.0149	0.0141	0.0144	0.0143
261630027	1	42.29222	-83.10694	MI	Wayne	Detroit-Warren-Livonia, MI	4,452,557	6,024	516	1	1.1	1		1	5	14	0.0256	0.0267	0.0353	0.0340	0.0341	0.0296
261630033	2	42.30667	-83.14889	MI	Wayne	Detroit-Warren-Livonia, MI	4,452,557	17,402	1,843	1	0.5			3	12	34	0.0236	0.0410	0.0601	0.0406	0.0464	0.0451
270370001	1	44.83333	-93.11500	MN	Dakota	Minneapolis-St. Paul-Bloomington,	2,968,806	5,074	404	1	3.2	1		2	8	24	0.0781	0.1153	0.2300	0.2100	0.2107	0.2042
270370020	1	44.76535	-93.03248	MN	Dakota	Minneapolis-St. Paul-Bloomington,	2,968,806	162	7		0.0			3	12	32	0.0051	0.0100	0.0200	0.0120	0.0140	0.0133
270370421	1	44.77720	-93.04097	MN	Dakota	Minneapolis-St. Paul-Bloomington,	2,968,806	478	24		0.0			1	9	27	0.0037	0.0069	0.0120	0.0100	0.0100	0.0100
270370423	1	44.77500	-93.06278	MN	Dakota	Minneapolis-St. Paul-Bloomington,	2,968,806	886	83		0.0			3	12	34	0.0018	0.0050	0.0100	0.0060	0.0073	0.0067
270370442	1	44.74036	-93.00556	MN	Dakota	Minneapolis-St. Paul-Bloomington,	2,968,806	168	11		0.3			2	10	28	0.0027	0.0062	0.0080	0.0060	0.0067	0.0067
270530050	1	45.00123	-93.26712	MN	Hennepin	Minneapolis-St. Paul-Bloomington,	2,968,806	16,318	923	1	0.0			3	12	35	0.0051	0.0093	0.0120	0.0117	0.0112	0.0112
270530963	1	44.95540	-93.25827	MN	Hennepin	Minneapolis-St. Paul-Bloomington,	2,968,806	46,218	3,929	1	0.2			3	12	36	0.0039	0.0071	0.0100	0.0080	0.0085	0.0085
270530964	1	44.88855	-93.19538	MN	Hennepin	Minneapolis-St. Paul-Bloomington,	2,968,806	209	0	1	0.0			1	4	14	0.0045	0.0114	0.0180	0.0080	0.0112	0.0110
270530965	1	45.00448	-93.24005	MN	Hennepin	Minneapolis-St. Paul-Bloomington,	2,968,806	19,106	1,095	1	0.4			3	12	35	0.0039	0.0080	0.0140	0.0100	0.0107	0.0107
270530966	1	44.98133	-93.26615	MN	Hennepin	Minneapolis-St. Paul-Bloomington,	2,968,806	17,156	439	1	0.0			3	12	35	0.0047	0.0080	0.0120	0.0100	0.0107	0.0101
270530967	1	44.99646	-93.23488	MN	Hennepin	Minneapolis-St. Paul-Bloomington,	2,968,806	14,621	580	1	0.4	1		1	7	20	0.0075	0.0142	0.0225	0.0157	0.0161	0.0163
270530968	1	44.89301	-93.23323	MN	Hennepin	Minneapolis-St. Paul-Bloomington,	2,968,806	11,243	789	1	0.0			1	6	18	0.0019	0.0033	0.0080	0.0050	0.0060	0.0065
270531007	1	45.04182	-93.29873	MN	Hennepin	Minneapolis-St. Paul-Bloomington,	2,968,806	14,889	1,118	1	0.0			3	12	35	0.0026	0.0067	0.0080	0.0067	0.0069	0.0069
271231003	1	44.96322	-93.19023	MN	Ramsey	Minneapolis-St. Paul-Bloomington,	2,968,806	9,247	474	1	0.1			3	12	33	0.0065	0.0129	0.0350	0.0200	0.0243	0.0210
271377001	1	47.52336	-92.53631	MN	St. Louis	Duluth, MN-WI	275,486	8,942	428	1	0.1			3	12	33	0.0047	0.0362	0.0900	0.0100	0.0360	0.0347
271377555	1	46.73264	-92.16337	MN	St. Louis	Duluth, MN-WI	275,486	4,527	287	1	0.0			3	12	34	0.0014	0.0031	0.0050	0.0040	0.0043	0.0043
290930016	1	37.62528	-91.12917	MO	Iron			58	4		0.0	1		3	12	34	0.6918	1.3070	4.1933	1.4540	2.2878	2.2878
290930021	1	37.65417	-91.13056	MO	Iron			58	4		0.0	1		3	12	36	0.5460	0.7187	0.9960	0.9840	0.9773	0.9773
290930023	1	37.50333	-90.69556	MO	Iron			138	7		0.0	1	1 *#	1	6	18	0.2291	0.3433	0.6320	0.4275	0.4865	0.3281
290930024	1	37.47972	-90.69028	MO	Iron			32	2		0.0	1	1 *#	1	6	18	0.5898	0.6677	1.6026	0.9927	1.0864	0.8292
290930025	1	37.51056	-90.69750	MO	Iron			138	7		0.0	1	1 *#	1	5	14	0.2477	0.3263	0.6320	0.4189	0.4723	0.3480
290930026	1	37.45917	-90.68639	MO	Iron			32	2		0.0	1	1 *#	1	5	15	0.2266	0.2523	0.3555	0.3370	0.3418	0.2127
290930027	1	37.48611	-90.69000	MO	Iron			32	2		0.0	1	1 *	3	12	33	0.2678	0.8761	1.4414	0.9300	1.0305	0.6387
290930029	1	37.47167	-90.68944	MO	Iron			32	2		0.0	1	1 *	3	12	32	0.2824	0.7148	1.4740	1.1410	1.1597	0.5722
290930030	1	37.46639	-90.69000	MO	Iron			32	2		0.0	1	1 *#	1	6	18	0.1665	0.2017	0.3330	0.2797	0.2734	0.1742
290990004	1	38.26330	-90.37850	MO	Jefferson	St. Louis, MO-IL	2,721,491	2,418	197	1	58.8	1		2	8	24	1.1300	1.4750	2.0731	1.8962	1.8591	1.7524
290990005	3	38.26722	-90.37944	MO	Jefferson	St. Louis, MO-IL	2,721,491	2,418	197	1	58.8	1		3	12	36	0.3711	0.6779	1.0655	0.9278	0.9277	0.8018
290990008	1	38.26194	-90.39417	MO	Jefferson	St. Louis, MO-IL	2,721,491	2,418	197		58.8	1		1	10	31	0.0910	0.1857	0.3700	0.3100	0.3128	0.2661
290990009	1	38.28444	-90.38194	MO	Jefferson	St. Louis, MO-IL	2,721,491	9,804	820	1	0.0	1		2	11	31	0.0957	0.1664	0.1750	0.1560	0.1595	0.1583
290990010	1	38.24110	-90.37680	MO	Jefferson	St. Louis, MO-IL	2,721,491	2,799	215	1	0.0	1		2	11	34	0.0388	0.0813	0.1680	0.1040	0.1207	0.1153
290990011	1	38.26820	-90.37380	MO	Jefferson	St. Louis, MO-IL	2,721,491	2,418	197	1	58.8	1		3	12	36	0.4778	1.3047	2.2070	1.3510	1.5975	1.3399
290990013	1	38.27361	-90.38000	MO	Jefferson	St. Louis, MO-IL	2,721,491	3,570	318	1	58.8	1		3	12	35	0.2633	0.8683	3.5680	0.6420	1.6167	1.5650
290990015	1	38.26167	-90.37972	MO	Jefferson	St. Louis, MO-IL	2,721,491	1,988	178	1	58.8	1		3	12	36	1.4501	1.9277	3.2884	2.2993	2.6139	2.4954
291892003	1	38.64972	-90.35056	MO	St. Louis	St. Louis, MO-IL	2,721,491	12,303	512	1	0.0			2	11	34	0.0063	0.0500	0.0500	0.0500	0.0500	0.0333
295100085	6	38.65630	-90.19810	MO	St. Louis (City)	St. Louis, MO-IL	2,721,491	9,140	783	1	0.0			1	4	11	0.0134	0.0216	0.0290	0.0255	0.0240	0.0290
340231003	1	40.47222	-74.47139	NJ	Middlesex	New York-Northern New Jersey-Lo	18,323,002	13,850	1,124	1	1.7	1		2	10	27	0.0403	0.1537	0.1878	0.1428	0.1496	0.1312
360470122	1	40.71980	-73.94788	NY	Kings	New York-Northern New Jersey-Lo	18,323,002	92,660	5,785	1	0.1			2	9	22	0.0276	0.0333	0.0360	0.0350	0.0345	0.0345
360632008	1	43.08216	-79.00099	NY	Niagara	Buffalo-Niagra Falls, NY Metropoli	1,170,111	6,795	386	1	0.0			1	4	12	0.0054	0.0060	0.0080	0.0080	0.0080	0.0080
360713001	1	41.46107	-74.36343	NY	Orange	Poughkeepsie-Newburgh-Middletow	621,517	1,481	99		1.8	1		2	9	26	0.0606	0.0820	0.1580	0.1100	0.1207	0.1073
360713002	1	41.45887	-74.35392	NY	Orange	Poughkeepsie-Newburgh-Middletow	621,517	1,257	86		1.8	1		2	9	26	0.1257	0.2417	0.4025	0.2400	0.2835	0.2248
360713004	1	41.47633	-74.36827	NY	Orange	Poughkeepsie-Newburgh-Middletow	621,517	6,816	434	1	0.0			2	9	26	0.0305	0.0386	0.0400	0.0400	0.0383	0.0351

Appendix 2B

Table 2B-1. Pb-TSP monitoring site information and 3-year statistics

site	poc	lat	long	state	county_name	cbsa_name	cbsa_pop00	population near site (mile radius)	under age 5 pop. (mile radius)	urban	sum point / nonpt Pb EI TPY w/in 1 mile	source oriented?	prev. source oriented? (see end notes)	3-year data capture (complete periods)			3-year metrics					
														comp. years	comp. qtrs	comp. months	annual mean	max quarterly mean	max monthly mean	2nd max monthly mean	average of 3 overall highest monthly means	average of 3 annual max monthly means
360850067	1	40.59733	-74.12619	NY	Richmond	New York-Northern New Jersey-Lo	18,323,002	21,834	1,373		0.0			1	4	11	0.0059	0.0082	0.0140	0.0125	0.0122	0.0140
390170015	2	39.48990	-84.36407	OH	Butler	Cincinnati-Middletown, OH-KY-IN	2,009,632	4,668	373	1	0.0			2	8	24	0.0107	0.0248	0.0650	0.0160	0.0320	0.0405
390290019	1	40.63111	-80.54694	OH	Columbiana	East Liverpool-Salem, OH	112,075	5,385	322	1	0.0			3	12	36	0.0144	0.0253	0.0300	0.0300	0.0287	0.0247
390290020	1	40.63972	-80.52389	OH	Columbiana	East Liverpool-Salem, OH	112,075	6,414	354	1	0.0			3	12	36	0.0158	0.0247	0.0310	0.0310	0.0307	0.0307
390290022	1	40.63500	-80.54667	OH	Columbiana	East Liverpool-Salem, OH	112,075	3,318	202	1	0.0			3	12	36	0.0139	0.0367	0.0800	0.0300	0.0433	0.0427
390350038	1	41.47694	-81.68194	OH	Cuyahoga	Cleveland-Elyria-Mentor, OH	2,148,143	7,329	585	1	0.1			3	12	36	0.0205	0.0300	0.0600	0.0360	0.0427	0.0423
390350042	1	41.48222	-81.70889	OH	Cuyahoga	Cleveland-Elyria-Mentor, OH	2,148,143	18,776	1,575	1	0.0			2	11	35	0.0169	0.0280	0.0430	0.0390	0.0373	0.0373
390350049	1	41.44667	-81.65111	OH	Cuyahoga	Cleveland-Elyria-Mentor, OH	2,148,143	9,720	758	1	0.0	1	1	3	12	36	0.1214	0.2367	0.4500	0.2600	0.3233	0.3100
390350050	1	41.44250	-81.64917	OH	Cuyahoga	Cleveland-Elyria-Mentor, OH	2,148,143	8,771	695	1	0.0	1	1	3	12	36	0.0362	0.0550	0.1000	0.0940	0.0920	0.0880
390350061	2	41.47506	-81.67596	OH	Cuyahoga	Cleveland-Elyria-Mentor, OH	2,148,143	6,141	444	1	0.3	1	1	3	12	36	0.0477	0.3600	0.5600	0.4700	0.3600	0.2090
390350069	1	41.51918	-81.63794	OH	Cuyahoga	Cleveland-Elyria-Mentor, OH	2,148,143	23,566	1,961	1	0.1			1	6	35	0.0170	0.0233	0.0470	0.0370	0.0377	0.0343
390490025	1	39.92806	-82.98111	OH	Franklin	Columbus, OH	1,612,694	15,220	1,226	1	0.6	1		3	12	36	0.0114	0.0197	0.0270	0.0210	0.0227	0.0203
390510001	1	41.57528	-83.99639	OH	Fulton	Toledo, OH	659,188	1,503	110	1	0.3	1		2	11	36	0.1332	0.2667	0.6100	0.5300	0.5200	0.5067
390910003	1	40.34306	-83.75500	OH	Logan	Bellefontaine, OH	46,005	1,536	108	1	0.1			3	12	36	0.0922	0.1467	0.2700	0.2000	0.2233	0.2233
390910005	1	40.34278	-83.76028	OH	Logan	Bellefontaine, OH	46,005	1,546	126	1	0.1	1		3	12	36	0.1058	0.1467	0.2200	0.2100	0.2067	0.2067
390910006	1	40.34111	-83.75778	OH	Logan	Bellefontaine, OH	46,005	1,217	87	1	0.1	1		3	12	36	0.1578	0.2667	0.3600	0.3600	0.3467	0.3467
390910007	1	40.34472	-83.75444	OH	Logan	Bellefontaine, OH	46,005	2,156	185	1	0.1			3	12	36	0.1497	0.2200	0.2600	0.2500	0.2500	0.2333
391670008	1	39.43361	-81.50250	OH	Washington	Parkersburg-Marietta, WV-OH	164,624	1,947	114		0.0			3	12	36	0.0054	0.0100	0.0130	0.0100	0.0110	0.0097
391670009	1	39.37696	-81.53730	OH	Washington	Parkersburg-Marietta, WV-OH	164,624	314	21		0.0			1	5	14	0.0073	0.0495	0.0880	0.0140	0.0383	0.0510
401159005	2	36.98580	-94.84920	OK	Ottawa	Miami, OK	33,194	1,573	117		0.0			1	4	11	0.0412	0.0613	0.0927	0.0630	0.0677	0.0927
401159006	1	36.98460	-94.82490	OK	Ottawa	Miami, OK	33,194	1,573	117		0.0			1	4	11	0.0316	0.0378	0.0623	0.0420	0.0485	0.0623
401159007	1	36.97190	-94.85180	OK	Ottawa	Miami, OK	33,194	1,573	117		0.0			1	4	11	0.0505	0.1030	0.1257	0.1140	0.1033	0.1257
401159008	1	36.97160	-94.82500	OK	Ottawa	Miami, OK	33,194	1,573	117		0.0			1	4	11	0.0312	0.0408	0.0708	0.0363	0.0474	0.0708
410510246	7	45.56130	-122.67878	OR	Multnomah	Portland-Vancouver-Beaverton, OR	1,927,881	24,303	1,771	1	0.0			1	4	11	0.0081	0.0101	0.0110	0.0105	0.0106	0.0110
420030002	1	40.50056	-80.07194	PA	Allegheny	Pittsburgh, PA	2,431,087	19,559	1,045	1	0.0			3	12	30	0.0096	0.0378	0.0503	0.0377	0.0387	0.0338
420032001	1	40.39667	-79.86361	PA	Allegheny	Pittsburgh, PA	2,431,087	10,120	769	1	0.2			3	12	35	0.0396	0.0567	0.1140	0.0660	0.0811	0.0801
420070505	1	40.68500	-80.32500	PA	Beaver	Pittsburgh, PA	2,431,087	6,497	218	1	0.0			2	11	31	0.0563	0.1531	0.2300	0.2280	0.2167	0.1848
420110005	1	40.46630	-75.75890	PA	Berks	Reading, PA	373,638	692	44		4.8	1		2	11	33	0.0618	0.0940	0.1580	0.1560	0.1400	0.1380
420110717	1	40.47667	-75.75917	PA	Berks	Reading, PA	373,638	575	39	1	4.8	1		2	11	30	0.1301	0.1800	0.2820	0.2740	0.2737	0.2513
420111717	1	40.37722	-75.91444	PA	Berks	Reading, PA	373,638	7,376	390	1	2.1	1		3	12	33	0.2570	0.3967	0.8020	0.5180	0.6013	0.6013
420210808	1	40.34806	-78.88278	PA	Cambria	Johnstown, PA	152,598	2,606	115	1	0.0			3	12	36	0.0383	0.0569	0.0920	0.0560	0.0647	0.0647
420250105	1	40.80306	-75.60833	PA	Carbon	Allentown-Bethlehem-Easton, PA-N	740,395	8,477	513	1	0.0			2	11	33	0.0779	0.2493	0.3560	0.2980	0.2924	0.2093
420450002	1	39.83556	-75.37250	PA	Delaware	Philadelphia-Camden-Wilmington, I	5,687,147	10,156	859	1	0.0			3	12	35	0.0372	0.0400	0.0400	0.0400	0.0400	0.0393
421010449	1	39.98250	-75.08306	PA	Philadelphia	Philadelphia-Camden-Wilmington, I	5,687,147	8,653	413	1	0.0	1	1	3	12	31	0.0203	0.0350	0.0380	0.0360	0.0365	0.0344
421290007	1	40.16667	-79.87500	PA	Westmoreland	Pittsburgh, PA	2,431,087	7,739	445	1	0.0			3	12	36	0.0352	0.0400	0.0400	0.0400	0.0400	0.0400
450031001	1	33.43253	-81.89233	SC	Aiken	Augusta-Richmond County, GA-SC	499,684	437	24		0.0			1	4	12	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000
450130007	1	32.43654	-80.67785	SC	Beaufort	Hilton Head Island-Beaufort, SC	141,615	4,928	330	1	0.0			3	12	34	0.0006	0.0022	0.0070	0.0034	0.0042	0.0035
450190003	2	32.88394	-79.97754	SC	Charleston	Charleston-North Charleston, SC	549,033	4,401	275	1	0.0			3	12	34	0.0014	0.0041	0.0104	0.0078	0.0077	0.0072
450190046	1	32.94275	-79.65718	SC	Charleston	Charleston-North Charleston, SC	549,033	63	4		0.0			3	12	33	0.0005	0.0032	0.0068	0.0035	0.0043	0.0035
450190047	1	32.84461	-79.94804	SC	Charleston	Charleston-North Charleston, SC	549,033	7,000	294	1	0.0			1	4	12	0.0022	0.0037	0.0058	0.0052	0.0052	0.0058
450410001	1	34.19794	-79.79885	SC	Florence	Florence, SC	193,155	3,426	224	1	0.0			1	4	13	0.0010	0.0026	0.0063	0.0023	0.0035	0.0041
450410002	1	34.16764	-79.85040	SC	Florence	Florence, SC	193,155	1,795	106	1	0.0			2	8	24	0.0011	0.0034	0.0102	0.0054	0.0063	0.0052
450430006	1	33.36378	-79.29426	SC	Georgetown	Georgetown, SC	55,797	5,247	427	1	0.3	1		2	11	32	0.0072	0.0166	0.0420	0.0200	0.0270	0.0252
450430007	1	33.34973	-79.29821	SC	Georgetown	Georgetown, SC	55,797	1,579	119		0.3			3	12	35	0.0002	0.0017	0.0054	0.0016	0.0023	0.0023
450430009	1	33.37399	-79.28570	SC	Georgetown	Georgetown, SC	55,797	2,447	185	1	0.3			3	12	35	0.0038	0.0081	0.0158	0.0148	0.0133	0.0120
450430010	1	33.36960	-79.29840	SC	Georgetown	Georgetown, SC	55,797	6,173	511	1	0.3			3	12	33	0.0049	0.0169	0.0265	0.0132	0.0166	0.0153
450450008	2	34.84045	-82.40291	SC	Greenville	Greenville, SC	559,940	7,967	381	1	0.0			3	12	34	0.0023	0.0071	0.0125	0.0066	0.0086	0.0088
450452002	1	34.94165	-82.22961	SC	Greenville	Greenville, SC	559,940	7,266	494	1	0.0			3	12	32	0.0001	0.0006	0.0018	0.0018	0.0017	0.0006
450470001	1	34.18111	-82.15224	SC	Greenwood	Greenwood, SC	66,271	7,853	667	1	0.0			3	12	32	0.0028	0.0063	0.0112	0.0106	0.0101	0.0082
450470002	1	34.16520	-82.16048	SC	Greenwood	Greenwood, SC	66,271	1,490	116		0.0			3	12	31	0.0071	0.0163	0.0320	0.0272	0.0279	0.0213
450510002	2	33.70460	-78.87745	SC	Horry	Myrtle Beach-Conway-North Myrtle	196,629	4,510	227	1	0.0			3	12	35	0.0009	0.0020	0.0053	0.0040	0.0042	0.0042
450630005	2	33.78560	-81.11978	SC	Lexington	Columbia, SC	647,158	736	66		0.0			1	4	12	0.0018	0.0033	0.0052	0.0050	0.0049	0.0052
450631002	2	33.96900	-81.06533	SC	Lexington	Columbia, SC	647,158	8,086	551	1	0.0			3	12	32	0.0046	0.0179	0.0356	0.0125	0.0192	0.0188
450790006	4	34.00740	-81.02329	SC	Richland	Columbia, SC	647,158	17,143	574	1	0.0			1	4	12	0.0030	0.0069	0.0090	0.0072	0.0071	0.0090

Appendix 2B

Table 2B-1. Pb-TSP monitoring site information and 3-year statistics

site	poc	lat	long	state	county_name	cbsa_name	cbsa_pop00	population near site (mile radius)	under age 5 pop. (mile radius)	urban	sum point / nonpt Pb EI TPY w/in 1 mile	source oriented?	prev. source oriented? (see end notes)	3-year data capture (complete periods)			3-year metrics					
														comp. years	comp. qtrs	comp. months	annual mean	max quarterly mean	max monthly mean	2nd max monthly mean	average of 3 overall highest monthly means	average of 3 annual max monthly means
450790007	2	34.09584	-80.96230	SC	Richland	Columbia, SC	647,158	4,405	233	1	0.0			3	12	36	0.0004	0.0014	0.0042	0.0030	0.0031	0.0027
450790019	1	33.99330	-81.02414	SC	Richland	Columbia, SC	647,158	15,569	287	1	0.0			3	12	35	0.0048	0.0097	0.0144	0.0138	0.0137	0.0137
450790021	1	33.81655	-80.78114	SC	Richland	Columbia, SC	647,158	123	10		0.0			3	12	35	0.0001	0.0012	0.0038	0.0000	0.0013	0.0013
450830001	2	34.94774	-81.93255	SC	Spartanburg	Spartanburg, SC	253,791	7,505	552	1	0.0			3	12	34	0.0018	0.0035	0.0062	0.0060	0.0060	0.0057
450850001	1	33.92423	-80.33774	SC	Sumter	Sumter, SC	104,646	4,990	407	1	0.0			3	12	35	0.0025	0.0064	0.0108	0.0104	0.0101	0.0101
450910005	1	34.96303	-81.00085	SC	York	Charlotte-Gastonia-Concord, NC-SC	1,330,448	3,453	221	1	0.0			2	11	27	0.0021	0.0042	0.0082	0.0058	0.0063	0.0052
470930027	1	35.98306	-83.95222	TN	Knox	Knoxville, TN	616,079	8,586	826	1	5.8	1		1	9	26	0.0182	0.0233	0.0400	0.0400	0.0387	0.0200
470931017	1	35.97500	-83.95444	TN	Knox	Knoxville, TN	616,079	7,817	763	1	5.8	1		1	9	26	0.0143	0.0193	0.0375	0.0240	0.0285	0.0192
471570044	1	35.08750	-90.07250	TN	Shelby	Memphis, TN-MS-AR	1,205,204	6,730	548	1	0.0	1	1	1	6	17	0.0100	0.0100	0.0100	0.0100	0.0100	0.0100
471633001	1	36.52556	-82.27333	TN	Sullivan	Kingsport-Bristol-Bristol, TN-VA	298,484	942	65		0.4	1		3	12	35	0.1249	0.1959	0.2843	0.2360	0.2501	0.2381
471633002	3	36.52472	-82.26806	TN	Sullivan	Kingsport-Bristol-Bristol, TN-VA	298,484	942	65		0.4	1		3	12	36	0.0614	0.1463	0.2920	0.1540	0.1880	0.1772
471633003	1	36.52806	-82.26833	TN	Sullivan	Kingsport-Bristol-Bristol, TN-VA	298,484	942	65		0.4	1		3	12	35	0.0651	0.1259	0.2322	0.1260	0.1476	0.1476
471870100	2	35.80222	-86.66028	TN	Williamson	Nashville-Davidson--Murfreesboro,	1,311,789	165	10		2.6	1		2	8	23	0.2527	0.9867	1.9120	0.8200	1.1579	1.0540
471870102	2	35.80222	-86.66028	TN	Williamson	Nashville-Davidson--Murfreesboro,	1,311,789	165	10		2.6	1		2	8	23	0.2575	0.6953	0.9460	0.6000	0.7093	0.5390
471871101	1	35.79944	-86.66500	TN	Williamson	Nashville-Davidson--Murfreesboro,	1,311,789	165	10		2.6	1		2	8	24	0.0811	0.3027	0.7020	0.1820	0.3333	0.4090
480610006	1	25.89251	-97.49382	TX	Cameron	Brownsville-Harlingen, TX	335,227	14,803	1,422	1	0.0			3	12	35	0.0053	0.0085	0.0090	0.0090	0.0089	0.0071
480850003	1	33.14250	-96.82472	TX	Collin	Dallas-Fort Worth-Arlington, TX	5,161,544	3,837	415		3.2	1		3	12	35	0.2271	0.3453	0.7954	0.4436	0.5595	0.5203
480850007	2	33.14722	-96.82556	TX	Collin	Dallas-Fort Worth-Arlington, TX	5,161,544	3,837	415	1	3.2	1		3	12	34	0.1186	0.2111	0.4760	0.3006	0.3408	0.3040
480850009	1	33.14472	-96.82889	TX	Collin	Dallas-Fort Worth-Arlington, TX	5,161,544	3,837	415		3.2	1		3	12	33	0.4961	0.6982	0.9692	0.8914	0.8710	0.8710
481130018	1	32.74556	-96.78250	TX	Dallas	Dallas-Fort Worth-Arlington, TX	5,161,544	6,451	491	1	0.0			3	12	34	0.0274	0.0804	0.2338	0.0880	0.1299	0.1286
481130057	2	32.77890	-96.87306	TX	Dallas	Dallas-Fort Worth-Arlington, TX	5,161,544	4,591	578	1	0.0			3	12	35	0.0362	0.0611	0.1029	0.1016	0.0986	0.0947
481130066	2	32.73972	-96.78278	TX	Dallas	Dallas-Fort Worth-Arlington, TX	5,161,544	8,270	622	1	0.0	1	1	1	7	20	0.0090	0.0209	0.0420	0.0280	0.0320	0.0340
481410033	1	31.77694	-106.50167	TX	El Paso	El Paso, TX	679,622	13,680	1,005	1	0.0			1	6	17	0.0120	0.0585	0.0600	0.0540	0.0540	0.0420
482011034	4	29.76799	-95.22058	TX	Harris	Houston-Sugar Land-Baytown, TX	4,715,407	14,785	1,770	1	0.0			3	12	36	0.0081	0.0220	0.0478	0.0230	0.0283	0.0260
484790016	1	27.51083	-99.51972	TX	Webb	Laredo, TX	193,117	14,880	1,441	1	0.0			3	12	36	0.0121	0.0163	0.0230	0.0214	0.0217	0.0217
490351001	1	40.70861	-112.09472	UT	Salt Lake	Salt Lake City, UT	968,858	215	23	1	0.0			2	11	32	0.0421	0.0762	0.1188	0.1072	0.1106	0.1106
721270003	1	18.44917	-66.05306	PR	San Juan	San Juan-Caguas-Guaynabo, PR	2,509,007	319	5	1				3	12	36	0.0014	0.0100	0.0125	0.0120	0.0122	0.0082

∗ These sites were classified as "previous" source-oriented but because production (and related lead emissions) at the associated source was not terminated until December, 2003, only data for 2004-2005 were considered for the "previous" source oriented characterization.

Data for 2004-2005 did not meet completeness criteria..

Appendix 2B

Table 2B-2. Pb-TSP monitoring site information and 1-year statistics

site	poc	lat	long	state	county_name	cbsa_name	cbsa_pop00	population near site (mile radius)	under age 5 pop. (mile radius)	urban	sum point / nonpt Pb EI TPY w/in 1 mile	source oriented?	prev. source oriented? (see end notes)	1-year metrics								
														max quarterly mean, 2003	max monthly mean, 2003	2nd max monthly mean, 2003	max quarterly mean, 2004	max monthly mean, 2004	2nd max monthly mean, 2004	max quarterly mean, 2005	max monthly mean, 2005	2nd max monthly mean, 2005
011090003	2	31.79056	-85.97917	AL	Pike	Troy, AL	29,605	461	31		4.5	1		1.9233	2.6600	2.4200	1.2267	1.7800	1.0000	0.3948	0.6156	0.3346
011090006	1	31.79278	-85.98056	AL	Pike	Troy, AL	29,605	461	31		4.5	1		0.9100	1.6900	0.8900	0.8433	1.3400	0.9400	0.1661	0.2402	0.1600
060250005	1	32.67611	-115.48333	CA	Imperial	El Centro, CA	142,361	16,385	1,290	1	0.0			0.0248	0.0404	0.0357	0.0179	0.0205	0.0191	0.0229	0.0380	0.0278
060371103	2	34.06659	-118.22688	CA	Los Angeles	Los Angeles-Long Beach-Santa Ana	12,365,627	29,329	1,633	1	0.3			0.0627	0.1460	0.0260	0.0253	0.0280	0.0280	0.0179	0.0250	0.0200
060371301	1	33.92899	-118.21071	CA	Los Angeles	Los Angeles-Long Beach-Santa Ana	12,365,627	47,423	5,066	1	0.0			0.0300	0.0440	0.0360	0.0313	0.0320	0.0320	0.0233	0.0300	0.0280
060371601	1	34.01407	-118.06056	CA	Los Angeles	Los Angeles-Long Beach-Santa Ana	12,365,627	13,333	1,066	1	0.0			0.0300	0.0480	0.0340	0.0215	0.0300	0.0300	0.0160	0.0250	0.0240
060374002	2	33.82376	-118.18921	CA	Los Angeles	Los Angeles-Long Beach-Santa Ana	12,365,627	20,131	1,232	1	0.0			0.0400	0.0960	0.0440	0.0147	0.0180	0.0160	0.0125	0.0140	0.0140
060374004	2	33.79236	-118.17533	CA	Los Angeles	Los Angeles-Long Beach-Santa Ana	12,365,627	61,497	6,697	1	0.0			0.0938	0.1020	0.0840	0.0146	0.0160	0.0160	0.0120	0.0160	0.0125
060375001	1	33.92288	-118.37026	CA	Los Angeles	Los Angeles-Long Beach-Santa Ana	12,365,627	19,148	1,680	1	0.0			0.0667	0.1700	0.0220	0.0107	0.0120	0.0100			
060375005	1	33.95080	-118.43043	CA	Los Angeles	Los Angeles-Long Beach-Santa Ana	12,365,627	33,968	1,358	1	0.0						0.0093	0.0120	0.0100	0.0118	0.0150	0.0100
060651003	2	33.94603	-117.40063	CA	Riverside	Riverside-San Bernardino-Ontario,	3,254,821	16,320	1,278	1	0.0			0.0113	0.0160	0.0120	0.0114	0.0140	0.0125	0.0113	0.0140	0.0100
060658001	3	33.99958	-117.41601	CA	Riverside	Riverside-San Bernardino-Ontario,	3,254,821	16,247	1,678	1	0.0			0.0179	0.0200	0.0200	0.0144	0.0220	0.0200	0.0169	0.0220	0.0180
060711004	1	34.10374	-117.62914	CA	San Bernardino	Riverside-San Bernardino-Ontario,	3,254,821	18,777	1,578	1	0.0			0.0343	0.0800	0.0200	0.0150	0.0180	0.0160	0.0160	0.0180	0.0180
060719004	1	34.10688	-117.27411	CA	San Bernardino	Riverside-San Bernardino-Ontario,	3,254,821	14,861	1,755	1	0.0			0.0773	0.1420	0.0680	0.0144	0.0160	0.0160	0.0133	0.0160	0.0150
080010005	1	39.79601	-104.97754	CO	Adams	Denver-Aurora, CO	2,157,756	2,025	183		1.9	1		0.1739	0.2509	0.2016	0.1384	0.1898	0.1887	0.5558	1.1037	0.4397
080010006	1	39.82574	-104.93699	CO	Adams	Denver-Aurora, CO	2,157,756	3,313	256	1	0.0			0.0388	0.0443	0.0406	0.0404	0.0726	0.0346	0.0957	0.2086	0.0428
080310002	4	39.75119	-104.98762	CO	Denver	Denver-Aurora, CO	2,157,756	22,019	974	1	0.0			0.0290	0.0467	0.0284	0.0222	0.0339	0.0262	0.1780	0.2955	0.2297
080310015	1	39.70012	-104.98714	CO	Denver	Denver-Aurora, CO	2,157,756	14,438	809	1	0.0			0.0212	0.0305	0.0196	0.0151	0.0184	0.0183			
080410011	1	38.83139	-104.82778	CO	El Paso	Colorado Springs, CO	537,484	10,581	552	1	0.0			0.0117	0.0165	0.0120	0.0100	0.0100	0.0100	0.0891	0.1387	0.1314
080650001	1	39.24778	-106.29139	CO	Lake	Edwards, CO	49,471	5,903	361	1	0.0			0.0192	0.0277	0.0209	0.0224	0.0310	0.0310	0.0187	0.0296	0.0170
100010002	1	38.98472	-75.55556	DE	Kent	Dover, DE	126,697	352	22		0.0			0.0040	0.0051	0.0041						
100031007	1	39.55111	-75.73083	DE	New Castle	Philadelphia-Camden-Wilmington,	5,687,147	2,041	209		0.0			0.0046	0.0058	0.0051						
100031008	1	39.57778	-75.61111	DE	New Castle	Philadelphia-Camden-Wilmington,	5,687,147	3,170	160		0.0			0.0063	0.0081	0.0058						
100032004	1	39.73944	-75.55806	DE	New Castle	Philadelphia-Camden-Wilmington,	5,687,147	34,053	2,649	1	0.0			0.0115	0.0163	0.0161						
100051002	1	38.64444	-75.61306	DE	Sussex	Seaford, DE	156,638	5,450	390	1	0.0			0.0042	0.0048	0.0042						
120571065	5	27.89222	-82.53861	FL	Hillsborough	Tampa-St. Petersburg-Clearwater, F	2,395,997	14,463	612	1	0.0			0.0062	0.0094	0.0080						
120571066	1	27.96028	-82.38250	FL	Hillsborough	Tampa-St. Petersburg-Clearwater, F	2,395,997	5,793	465	1	1.3	1		0.7400	1.3800	0.7800	1.2600	1.7400	1.0400	1.1188	1.3000	1.2000
120571073	1	27.96583	-82.37944	FL	Hillsborough	Tampa-St. Petersburg-Clearwater, F	2,395,997	4,541	340	1	1.3	1		0.2533	0.4800	0.4400	0.2333	0.3400	0.2800	0.2933	0.4200	0.3200
120571075	5	28.05000	-82.37806	FL	Hillsborough	Tampa-St. Petersburg-Clearwater, F	2,395,997	10,691	490	1	0.0			0.0054	0.0105	0.0072						
121030004	5	27.94639	-82.73194	FL	Pinellas	Tampa-St. Petersburg-Clearwater, F	2,395,997	13,048	557	1	0.0			0.0041	0.0067	0.0039						
121030018	5	27.78556	-82.74000	FL	Pinellas	Tampa-St. Petersburg-Clearwater, F	2,395,997	11,289	571	1	0.0			0.0056	0.0103	0.0093	0.0071	0.0112	0.0051			
121033005	1	27.87583	-82.69639	FL	Pinellas	Tampa-St. Petersburg-Clearwater, F	2,395,997	2,151	58	1	0.0			0.0067	0.0200	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000
130890003	2	33.69833	-84.27333	GA	DeKalb	Atlanta-Sandy Springs-Marietta, GA	4,247,981	7,888	663	1	0.0			0.1000	0.1000	0.1000	0.1000	0.1000	0.1000	0.1000	0.1000	0.1000
132150011	1	32.43083	-84.93167	GA	Muscogee	Columbus, GA-AL	281,768	10,871	1,037	1	0.3	1	1	0.1000	0.1000	0.1000	0.1000	0.1000	0.1000	0.1000	0.1000	0.1000
150032004	1	21.39667	-157.97167	HI	Honolulu	Honolulu, HI	876,156	23,622	1,207	1	0.1			0.0029	0.0072	0.0021	0.0015	0.0017	0.0015	0.0017	0.0025	0.0019
170310001	1	41.67275	-87.73246	IL	Cook	Chicago-Naperville-Joliet, IL-IN-W	9,098,316	13,648	971	1	0.0			0.0157	0.0250	0.0180	0.0229	0.0360	0.0200	0.0167	0.0200	0.0180
170310022	2	41.68920	-87.53932	IL	Cook	Chicago-Naperville-Joliet, IL-IN-W	9,098,316	22,040	1,708	1	0.2			0.0286	0.0360	0.0350	0.0314	0.0420	0.0420	0.0353	0.0440	0.0360
170310026	1	41.87333	-87.64507	IL	Cook	Chicago-Naperville-Joliet, IL-IN-W	9,098,316	28,739	1,203	1	0.0			0.0613	0.0860	0.0620	0.0557	0.0900	0.0700	0.0347	0.0500	0.0420
170310052	1	41.96743	-87.74982	IL	Cook	Chicago-Naperville-Joliet, IL-IN-W	9,098,316	42,187	2,877	1	0.0			0.0250	0.0280	0.0260	0.0257	0.0400	0.0300	0.0260	0.0380	0.0280
170313103	1	41.96528	-87.87639	IL	Cook	Chicago-Naperville-Joliet, IL-IN-W	9,098,316	10,302	670	1	0.0			0.0180	0.0240	0.0220	0.0140	0.0160	0.0160	0.0271	0.0440	0.0240
170313301	1	41.78278	-87.80528	IL	Cook	Chicago-Naperville-Joliet, IL-IN-W	9,098,316	23,749	1,678	1	0.0			0.0750	0.1950	0.0360	0.0520	0.1140	0.0700	0.0246	0.0375	0.0225
170314201	1	42.14000	-87.79917	IL	Cook	Chicago-Naperville-Joliet, IL-IN-W	9,098,316	6,070	303	1	0.0			0.0133	0.0175	0.0160	0.0120	0.0160	0.0140			
170316003	1	41.87194	-87.82611	IL	Cook	Chicago-Naperville-Joliet, IL-IN-W	9,098,316	14,862	1,071	1	0.0			0.0373	0.0480	0.0400	0.0333	0.0460	0.0420	0.0387	0.0500	0.0360
171170002	2	39.39804	-89.80975	IL	Macoupin	St. Louis, MO-IL	2,721,491	40	2		0.0			0.0113	0.0140	0.0100	0.0113	0.0140	0.0100	0.0107	0.0120	0.0120
171190010	1	38.69417	-90.15361	IL	Madison	St. Louis, MO-IL	2,721,491	8,014	529	1	1.3	1		0.3280	0.9100	0.0620	0.1515	0.2880	0.0900	0.1033	0.1880	0.0750
171193007	2	38.86056	-90.10583	IL	Madison	St. Louis, MO-IL	2,721,491	5,397	360	1	0.1			0.0173	0.0320	0.0240	0.0175	0.0240	0.0200	0.0193	0.0225	0.0200
171430037	1	40.69889	-89.58474	IL	Peoria	Peoria, IL	366,899	12,643	1,109	1	0.0			0.0167	0.0220	0.0180	0.0129	0.0180	0.0100	0.0279	0.0320	0.0300
171630010	2	38.61222	-90.16028	IL	St. Clair	St. Louis, MO-IL	2,721,491	3,512	430	1	0.3			0.0563	0.0940	0.0720	0.0529	0.0750	0.0520	0.0707	0.1050	0.0980
180350008	1	40.15806	-85.42111	IN	Delaware	Muncie, IN	118,769	2,108	104	1	0.0	1		0.2341	0.3394	0.3138	0.4657	0.7371	0.4653	0.4642	0.5991	0.4671
180350009	2	40.15944	-85.41556	IN	Delaware	Muncie, IN	118,769	980	82		0.0	1		0.8073	1.2183	0.967273	4.0931	5.7750	5.0220	1.3890	1.5900	1.3923
180890023	1	41.65278	-87.43944	IN	Lake	Chicago-Naperville-Joliet, IL-IN-W	9,098,316	5,959	603	1	6.5	1		0.0435	0.0620	0.0510	0.0691	0.0910	0.0783	0.0462	0.0613	0.0578
180892008	1	41.63944	-87.49361	IN	Lake	Chicago-Naperville-Joliet, IL-IN-W	9,098,316	7,144	612	1	0.0			0.0277	0.0413	0.0335	0.0289	0.0590	0.0318	0.0296	0.0484	0.0363
180892011	2	41.59250	-87.47194	IN	Lake	Chicago-Naperville-Joliet, IL-IN-W	9,098,316	9,815	729	1	0.0			0.0453	0.0610	0.0420	0.0358	0.0532	0.0463	0.1352	0.3050	0.0778
180930004	1	38.88944	-86.55194	IN	Lawrence	Bedford, IN	45,922	393	32		0.0			0.0270	0.0270	0.0270	0.0270	0.0270	0.0270	0.0270	0.0270	0.0270
180970063	1	39.76083	-86.29722	IN	Marion	Indianapolis-Carmel, IN	1,525,104	12,176	875	1	1.7	1		0.0508	0.0812	0.0584	0.0770	0.1123	0.0638	0.0329	0.0594	0.0380

Appendix 2B

Table 2B-2. Pb-TSP monitoring site information and 1-year statistics

site	poc	lat	long	state	county_name	cbsa_name	cbsa_pop00	population near site (mile radius)	under age 5 pop. (mile radius)	urban	sum point / nonpt Pb EI TPY w/in 1 mile	source oriented?	prev. source oriented? (see end notes)	1-year metrics								
														max quarterly mean, 2003	max monthly mean, 2003	2nd max monthly mean, 2003	max quarterly mean, 2004	max monthly mean, 2004	2nd max monthly mean, 2004	max quarterly mean, 2005	max monthly mean, 2005	2nd max monthly mean, 2005
180970076	1	39.75889	-86.28972	IN	Marion	Indianapolis-Carmel, IN	1,525,104	9,171	602	1	1.7	1		0.0143	0.0190	0.0178	0.0186	0.0204	0.0190	0.0254	0.0360	0.0346
180970078	1	39.81110	-86.11447	IN	Marion	Indianapolis-Carmel, IN	1,525,104	14,196	1,175	1	0.0			0.0057	0.0110	0.0075	0.0103	0.0154	0.0094	0.0251	0.0288	0.0240
181010001	1	38.89028	-86.76083	IN	Martin			84	5		0.0			0.0299	0.0358	0.0270	0.0270	0.0270	0.0270	0.0270	0.0270	0.0270
181630006	2	37.97167	-87.56722	IN	Vanderburgh	Evansville, IN-KY	342,815	13,666	817	1	0.0			0.0051	0.0075	0.0060	0.0126	0.0286	0.0170	0.0083	0.0088	0.0088
260490021	4	43.04722	-83.67028	MI	Genesee	Flint, MI	436,141	9,889	994	1	0.0			0.0153	0.0189	0.0166	0.0121	0.0156	0.0132	0.0117	0.0209	0.0155
261130001	1	44.31056	-84.89194	MI	Missaukee	Cadillac, MI	44,962	58	3		0.0			0.0040	0.0042	0.0039	0.0032	0.0040	0.0036	0.0056	0.0080	0.0046
261630001	2	42.22861	-83.20833	MI	Wayne	Detroit-Warren-Livonia, MI	4,452,557	14,329	798	1	0.0			0.0096	0.0101	0.0100	0.0101	0.0111	0.0105	0.0107	0.0124	0.0115
261630005	1	42.26722	-83.13222	MI	Wayne	Detroit-Warren-Livonia, MI	4,452,557	11,314	923	1	0.2			0.0247	0.0340	0.0208	0.0259	0.0315	0.0310	0.0191	0.0268	0.0228
261630015	4	42.30278	-83.10667	MI	Wayne	Detroit-Warren-Livonia, MI	4,452,557	17,729	1,771	1	0.0			0.0207	0.0247	0.0214	0.0252	0.0299	0.0256	0.0204	0.0278	0.0252
261630019	1	42.43083	-83.00028	MI	Wayne	Detroit-Warren-Livonia, MI	4,452,557	28,362	2,628	1	0.0			0.0136	0.0141	0.0141	0.0108	0.0149	0.0124	0.0138	0.0140	0.0125
261630027	1	42.29222	-83.10694	MI	Wayne	Detroit-Warren-Livonia, MI	4,452,557	6,024	516	1	1.1	1					0.0173	0.0240	0.0112	0.0267	0.0353	0.0340
261630033	2	42.30667	-83.14889	MI	Wayne	Detroit-Warren-Livonia, MI	4,452,557	17,402	1,843	1	0.5			0.0410	0.0601	0.0406	0.0262	0.0384	0.0381	0.0269	0.0368	0.0301
270370001	1	44.83333	-93.11500	MN	Dakota	Minneapolis-St. Paul-Bloomington,	2,968,806	5,074	404	1	3.2	1			0.2100	0.0380	0.1153	0.2300	0.1920	0.0979	0.1725	0.0900
270370020	1	44.76535	-93.03248	MN	Dakota	Minneapolis-St. Paul-Bloomington,	2,968,806	162	7		0.0			0.0086	0.0200	0.0100	0.0060	0.0080	0.0060	0.0100	0.0120	0.0100
270370421	1	44.77720	-93.04097	MN	Dakota	Minneapolis-St. Paul-Bloomington,	2,968,806	478	24		0.0			0.0057	0.0100	0.0060	0.0069	0.0080	0.0067	0.0069	0.0120	0.0080
270370423	1	44.77500	-93.06278	MN	Dakota	Minneapolis-St. Paul-Bloomington,	2,968,806	886	83		0.0			0.0050	0.0100	0.0060	0.0033	0.0060	0.0040	0.0029	0.0040	0.0040
270370442	1	44.74036	-93.00556	MN	Dakota	Minneapolis-St. Paul-Bloomington,	2,968,806	168	11		0.3			0.0062	0.0080	0.0020	0.0027	0.0060	0.0050	0.0036	0.0060	0.0050
270530050	1	45.00123	-93.26712	MN	Hennepin	Minneapolis-St. Paul-Bloomington,	2,968,806	16,318	923	1	0.0			0.0079	0.0100	0.0080	0.0093	0.0120	0.0100	0.0060	0.0117	0.0080
270530963	1	44.95540	-93.25827	MN	Hennepin	Minneapolis-St. Paul-Bloomington,	2,968,806	46,218	3,929	1	0.2			0.0071	0.0080	0.0075	0.0064	0.0075	0.0060	0.0050	0.0100	0.0067
270530964	1	44.88855	-93.19538	MN	Hennepin	Minneapolis-St. Paul-Bloomington,	2,968,806	209	0	1	0.0			0.0114	0.0180	0.0080		0.0040	0.0020			
270530965	1	45.00448	-93.24005	MN	Hennepin	Minneapolis-St. Paul-Bloomington,	2,968,806	19,106	1,095	1	0.4			0.0080	0.0100	0.0080	0.0047	0.0080	0.0067	0.0073	0.0140	0.0060
270530966	1	44.98133	-93.26615	MN	Hennepin	Minneapolis-St. Paul-Bloomington,	2,968,806	17,156	439	1	0.0			0.0080	0.0120	0.0060	0.0079	0.0100	0.0100	0.0053	0.0083	0.0080
270530967	1	44.99646	-93.23488	MN	Hennepin	Minneapolis-St. Paul-Bloomington,	2,968,806	14,621	580	1	0.4	1					0.0076	0.0100	0.0080	0.0142	0.0225	0.0157
270530968	1	44.89301	-93.23323	MN	Hennepin	Minneapolis-St. Paul-Bloomington,	2,968,806	11,243	789	1	0.0						0.0033	0.0080	0.0050	0.0031	0.0050	0.0050
270531007	1	45.04182	-93.29873	MN	Hennepin	Minneapolis-St. Paul-Bloomington,	2,968,806	14,889	1,118	1	0.0			0.0067	0.0080	0.0060	0.0043	0.0060	0.0050	0.0029	0.0067	0.0040
271231003	1	44.96322	-93.19023	MN	Ramsey	Minneapolis-St. Paul-Bloomington,	2,968,806	9,247	474	1	0.1			0.0100	0.0200	0.0100	0.0129	0.0350	0.0180	0.0067	0.0080	0.0080
271377001	1	47.52336	-92.53631	MN	St. Louis	Duluth, MN-WI	275,486	8,942	428	1	0.1			0.0362	0.0900	0.0080	0.0021	0.0040	0.0040	0.0100	0.0100	0.0040
271377555	1	46.73264	-92.16337	MN	St. Louis	Duluth, MN-WI	275,486	4,527	287	1	0.0			0.0020	0.0040	0.0020	0.0027	0.0040	0.0020	0.0040	0.0050	0.0040
290930016	1	37.62528	-91.12917	MO	Iron			58	4		0.0	1		0.6593	1.2160	1.1720	0.7893	1.4540	1.0340	1.3070	4.1933	1.2120
290930021	1	37.65417	-91.13056	MO	Iron			58	4		0.0	1		0.5850	0.9840	0.7820	0.7187	0.9960	0.9280	0.6627	0.9520	0.8660
290930023	1	37.50333	-90.69556	MO	Iron			138	7		0.0	1	1 *#	0.3433	0.6320	0.4275	0.0131	0.0242	0.0136			
290930024	1	37.47972	-90.69028	MO	Iron			32	2		0.0	1	1 *#	0.6677	1.6026	0.9927	0.0333	0.0558	0.0337			
290930025	1	37.51056	-90.69750	MO	Iron			138	7		0.0	1	1 *#	0.3263	0.6320	0.4189	0.0642	0.0640	0.0600			
290930026	1	37.45917	-90.68639	MO	Iron			32	2		0.0	1	1 *#	0.2523	0.3555	0.3370	0.0621	0.0700	0.0660			
290930027	1	37.48611	-90.69000	MO	Iron			32	2		0.0	1	1 *	0.8761	1.4414	0.9300	0.1654	0.3080	0.2200	0.1257	0.1667	0.1480
290930029	1	37.47167	-90.68944	MO	Iron			32	2		0.0	1	1 *	0.7148	1.4740	1.1410	0.0893	0.1025	0.0900	0.1027	0.1400	0.0980
290930030	1	37.46639	-90.69000	MO	Iron			32	2		0.0	1	1 *#	0.2017	0.3330	0.2797	0.0089	0.0154	0.0125			
290990004	1	38.26330	-90.37850	MO	Jefferson	St. Louis, MO-IL	2,721,491	2,418	197	1	58.8	1					1.4750	2.0731	1.8962	1.1215	1.4317	1.1765
290990005	3	38.26722	-90.37944	MO	Jefferson	St. Louis, MO-IL	2,721,491	2,418	197	1	58.8	1		0.5438	0.7900	0.6157	0.6779	1.0655	0.9278	0.3742	0.5499	0.4180
290990008	1	38.26194	-90.39417	MO	Jefferson	St. Louis, MO-IL	2,721,491	2,418	197		58.8	1		0.1500	0.2583	0.1350	0.1368	0.1700	0.1570	0.1857	0.3700	0.3100
290990009	1	38.28444	-90.38194	MO	Jefferson	St. Louis, MO-IL	2,721,491	9,804	820	1	0.0	1		0.1664	0.1440	0.1100	0.1413	0.1750	0.1475	0.1064	0.1560	0.1125
290990010	1	38.24110	-90.37680	MO	Jefferson	St. Louis, MO-IL	2,721,491	2,799	215	1	0.0	1		0.0680	0.1040	0.0900	0.0700	0.0740	0.0680	0.0813	0.1680	0.0660
290990011	1	38.26820	-90.37380	MO	Jefferson	St. Louis, MO-IL	2,721,491	2,418	197	1	58.8	1		0.5321	1.0490	0.7327	1.3047	2.2070	1.3510	0.4200	0.7638	0.7153
290990013	1	38.27361	-90.38000	MO	Jefferson	St. Louis, MO-IL	2,721,491	3,570	318	1	58.8	1		0.2717	0.4850	0.3355	0.8683	3.5680	0.6400	0.3379	0.6420	0.2725
290990015	1	38.26167	-90.37972	MO	Jefferson	St. Louis, MO-IL	2,721,491	1,988	178	1	58.8	1		1.4906	2.2993	1.9442	1.4760	1.8987	1.8531	1.9277	3.2884	2.2541
291892003	1	38.64972	-90.35056	MO	St. Louis	St. Louis, MO-IL	2,721,491	12,303	512	1	0.0			0.0000	0.0000	0.0000	0.0500	0.0500	0.0500	0.0500	0.0500	0.0500
295100085	6	38.65630	-90.19810	MO	St. Louis (City)	St. Louis, MO-IL	2,721,491	9,140	783	1	0.0						0.0216	0.0290	0.0255			
340231003	1	40.47222	-74.47139	NJ	Middlesex	New York-Northern New Jersey-Lo	18,323,002	13,850	1,124	1	1.7	1		0.0419	0.0875	0.0360	0.1146	0.1878	0.1428	0.1537	0.1182	0.0874
360470122	1	40.71980	-73.94788	NY	Kings	New York-Northern New Jersey-Lo	18,323,002	92,660	5,785	1	0.1			0.0293	0.0350	0.0300	0.0333	0.0360	0.0325	0.0309	0.0325	0.0300
360632008	1	43.08216	-79.00099	NY	Niagara	Buffalo-Niagra Falls, NY Metropoli	1,170,111	6,795	386	1	0.0			0.0060	0.0080	0.0080						
360713001	1	41.46107	-74.36343	NY	Orange	Poughkeepsie-Newburgh-Middletov	621,517	1,481	99		1.8	1		0.0820	0.1580	0.0940	0.0746	0.1100	0.0917	0.0453	0.0540	0.0460
360713002	1	41.45887	-74.35392	NY	Orange	Poughkeepsie-Newburgh-Middletov	621,517	1,257	86		1.8	1		0.2417	0.2080	0.1700	0.2369	0.4025	0.2400	0.0520	0.0640	0.0500
360713004	1	41.47633	-74.36827	NY	Orange	Poughkeepsie-Newburgh-Middletov	621,517	6,816	434	1	0.0			0.0313	0.0333	0.0320	0.0307	0.0320	0.0320	0.0386	0.0400	0.0400
360850067	1	40.59733	-74.12619	NY	Richmond	New York-Northern New Jersey-Lo	18,323,002	21,834	1,373		0.0			0.0082	0.0140	0.0125						
390170015	2	39.48990	-84.36407	OH	Butler	Cincinnati-Middletown, OH-KY-IN	2,009,632	4,668	373	1	0.0						0.0129	0.0160	0.0150	0.0248	0.0650	0.0130

Appendix 2B

Table 2B-2. Pb-TSP monitoring site information and 1-year statistics

site	poc	lat	long	state	county_name	cbsa_name	cbsa_pop00	population near site (mile radius)	under age 5 pop. (mile radius)	urban	sum point / nonpt Pb EI TPY w/in 1 mile	source oriented?	prev. source oriented? (see end notes)	1-year metrics								
														max quarterly mean, 2003	max monthly mean, 2003	2nd max monthly mean, 2003	max quarterly mean, 2004	max monthly mean, 2004	2nd max monthly mean, 2004	max quarterly mean, 2005	max monthly mean, 2005	2nd max monthly mean, 2005
390290019	1	40.63111	-80.54694	OH	Columbiana	East Liverpool-Salem, OH	112,075	5,385	322	1	0.0			0.0253	0.0300	0.0300	0.0142	0.0220	0.0140	0.0150	0.0220	0.0190
390290020	1	40.63972	-80.52389	OH	Columbiana	East Liverpool-Salem, OH	112,075	6,414	354	1	0.0			0.0247	0.0300	0.0240	0.0190	0.0310	0.0290	0.0191	0.0310	0.0190
390290022	1	40.63500	-80.54667	OH	Columbiana	East Liverpool-Salem, OH	112,075	3,318	202	1	0.0			0.0367	0.0800	0.0200	0.0180	0.0300	0.0150	0.0142	0.0180	0.0170
390350038	1	41.47694	-81.68194	OH	Cuyahoga	Cleveland-Elyria-Mentor, OH	2,148,143	7,329	585	1	0.1			0.0300	0.0600	0.0300	0.0253	0.0360	0.0320	0.0223	0.0310	0.0300
390350042	1	41.48222	-81.70889	OH	Cuyahoga	Cleveland-Elyria-Mentor, OH	2,148,143	18,776	1,575	1	0.0			0.0233	0.0300	0.0200	0.0230	0.0430	0.0230	0.0280	0.0390	0.0290
390350049	1	41.44667	-81.65111	OH	Cuyahoga	Cleveland-Elyria-Mentor, OH	2,148,143	9,720	758	1	0.0	1	1	0.2367	0.4500	0.2500	0.1380	0.2200	0.1500	0.1503	0.2600	0.2600
390350050	1	41.44250	-81.64917	OH	Cuyahoga	Cleveland-Elyria-Mentor, OH	2,148,143	8,771	695	1	0.0	1	1	0.0400	0.0700	0.0500	0.0543	0.1000	0.0500	0.0550	0.0940	0.0820
390350061	2	41.47506	-81.67596	OH	Cuyahoga	Cleveland-Elyria-Mentor, OH	2,148,143	6,141	444	1	0.3	1	1	0.3600	0.5600	0.4700	0.0257	0.0440	0.0300	0.0183	0.0230	0.0180
390350069	1	41.51918	-81.63794	OH	Cuyahoga	Cleveland-Elyria-Mentor, OH	2,148,143	23,566	1,961	1	0.1			0.0233	0.0290	0.0280		0.0470	0.0370	0.0210	0.0270	0.0200
390490025	1	39.92806	-82.98111	OH	Franklin	Columbus, OH	1,612,694	15,220	1,226	1	0.6	1		0.0167	0.0200	0.0200	0.0197	0.0270	0.0210	0.0085	0.0140	0.0130
390510001	1	41.57528	-83.99639	OH	Fulton	Toledo, OH	659,188	1,503	110	1	0.3	1		0.2667	0.5300	0.2500	0.2460	0.3800	0.2800	0.1867	0.6100	0.4200
390910003	1	40.34306	-83.75500	OH	Logan	Bellefontaine, OH	46,005	1,536	108	1	0.1			0.1467	0.2700	0.2000	0.1337	0.2000	0.1900	0.1070	0.2000	0.1700
390910005	1	40.34278	-83.76028	OH	Logan	Bellefontaine, OH	46,005	1,546	126	1	0.1	1		0.1300	0.1900	0.1100	0.1467	0.2100	0.1300	0.1467	0.2200	0.1900
390910006	1	40.34111	-83.75778	OH	Logan	Bellefontaine, OH	46,005	1,217	87	1	0.1	1		0.1967	0.3200	0.2100	0.2667	0.3600	0.2700	0.2267	0.3600	0.2800
390910007	1	40.34472	-83.75444	OH	Logan	Bellefontaine, OH	46,005	2,156	185	1	0.1			0.1500	0.2100	0.1500	0.2200	0.2600	0.2500	0.1700	0.2300	0.1900
391670008	1	39.43361	-81.50250	OH	Washington	Parkersburg-Marietta, WV-OH	164,624	1,947	114		0.0			0.0100	0.0100	0.0100	0.0072	0.0130	0.0100	0.0051	0.0062	0.0054
391670009	1	39.37696	-81.53730	OH	Washington	Parkersburg-Marietta, WV-OH	164,624	314	21		0.0						0.0495	0.0880	0.0110	0.0106	0.0140	0.0130
401159005	2	36.98580	-94.84920	OK	Ottawa	Miami, OK	33,194	1,573	117		0.0									0.0613	0.0927	0.0630
401159006	1	36.98460	-94.82490	OK	Ottawa	Miami, OK	33,194	1,573	117		0.0									0.0378	0.0623	0.0420
401159007	1	36.97190	-94.85180	OK	Ottawa	Miami, OK	33,194	1,573	117		0.0									0.1030	0.1257	0.1140
401159008	1	36.97160	-94.82500	OK	Ottawa	Miami, OK	33,194	1,573	117		0.0									0.0408	0.0708	0.0363
410510246	7	45.56130	-122.67878	OR	Multnomah	Portland-Vancouver-Beaverton, OR	1,927,881	24,303	1,771	1	0.0			0.0101	0.0110	0.0105						
420030002	1	40.50056	-80.07194	PA	Allegheny	Pittsburgh, PA	2,431,087	19,559	1,045	1	0.0			0.0255	0.0280	0.0260	0.0115	0.0230	0.0143	0.0378	0.0503	0.0377
420032001	1	40.39667	-79.86361	PA	Allegheny	Pittsburgh, PA	2,431,087	10,120	769	1	0.2			0.0567	0.1140	0.0660	0.0394	0.0630	0.0525	0.0546	0.0632	0.0629
420070505	1	40.68500	-80.32500	PA	Beaver	Pittsburgh, PA	2,431,087	6,497	218	1	0.0			0.0913	0.1920	0.0483	0.0925	0.1325	0.1000	0.1531	0.2300	0.2280
420110005	1	40.46630	-75.75890	PA	Berks	Reading, PA	373,638	692	44		4.8	1		0.0757	0.1000	0.0760	0.0940	0.1560	0.1060	0.0881	0.1580	0.0950
420110717	1	40.47667	-75.75917	PA	Berks	Reading, PA	373,638	575	39	1	4.8	1		0.1238	0.1980	0.1580	0.1800	0.2820	0.2650	0.1736	0.2740	0.2320
420111717	1	40.37722	-75.91444	PA	Berks	Reading, PA	373,638	7,376	390	1	2.1	1		0.3860	0.4840	0.4560	0.3967	0.5180	0.4580	0.3907	0.8020	0.2400
420210808	1	40.34806	-78.88278	PA	Cambria	Johnstown, PA	152,598	2,606	115	1	0.0			0.0364	0.0460	0.0320	0.0453	0.0560	0.0400	0.0569	0.0920	0.0417
420250105	1	40.80306	-75.60833	PA	Carbon	Allentown-Bethlehem-Easton, PA-N	740,395	8,477	513	1	0.0			0.0992	0.1300	0.1240	0.1150	0.1420	0.1225	0.2493	0.3560	0.2980
420450002	1	39.83556	-75.37250	PA	Delaware	Philadelphia-Camden-Wilmington,	5,687,147	10,156	859	1	0.0			0.0364	0.0380	0.0380	0.0400	0.0400	0.0400	0.0400	0.0400	0.0400
421010449	1	39.98250	-75.08306	PA	Philadelphia	Philadelphia-Camden-Wilmington,	5,687,147	8,653	413	1	0.0	1	1	0.0350	0.0380	0.0360	0.0269	0.0355	0.0312	0.0236	0.0298	0.0266
421290007	1	40.16667	-79.87500	PA	Westmoreland	Pittsburgh, PA	2,431,087	7,739	445	1	0.0			0.0369	0.0400	0.0400	0.0393	0.0400	0.0400	0.0400	0.0400	0.0400
450031001	1	33.43253	-81.89233	SC	Aiken	Augusta-Richmond County, GA-SC	499,684	437	24		0.0			0.0000	0.0000	0.0000						
450130007	1	32.43654	-80.67785	SC	Beaufort	Hilton Head Island-Beaufort, SC	141,615	4,928	330	1	0.0			0.0022	0.0070	0.0016	0.0020	0.0034	0.0023	0.0000	0.0000	0.0000
450190003	2	32.88394	-79.97754	SC	Charleston	Charleston-North Charleston, SC	549,033	4,401	275	1	0.0			0.0041	0.0104	0.0048	0.0026	0.0078	0.0030	0.0013	0.0033	0.0000
450190046	1	32.94275	-79.65718	SC	Charleston	Charleston-North Charleston, SC	549,033	63	4		0.0			0.0032	0.0068	0.0035	0.0007	0.0020	0.0020	0.0006	0.0018	0.0000
450190047	1	32.84461	-79.94804	SC	Charleston	Charleston-North Charleston, SC	549,033	7,000	294	1	0.0			0.0037	0.0058	0.0052						
450410001	1	34.19794	-79.79885	SC	Florence	Florence, SC	193,155	3,426	224	1	0.0			0.0026	0.0063	0.0023		0.0020				
450410002	1	34.16764	-79.85040	SC	Florence	Florence, SC	193,155	1,795	106	1	0.0				0.0000		0.0021	0.0054	0.0032	0.0034	0.0102	0.0000
450430006	1	33.36378	-79.29426	SC	Georgetown	Georgetown, SC	55,797	5,247	427	1	0.3	1		0.0166	0.0420	0.0077	0.0123	0.0200	0.0190	0.0072	0.0135	0.0100
450430007	1	33.34973	-79.29821	SC	Georgetown	Georgetown, SC	55,797	1,579	119		0.3			0.0017	0.0054	0.0000	0.0000	0.0000	0.0000	0.0005	0.0016	0.0000
450430009	1	33.37399	-79.28570	SC	Georgetown	Georgetown, SC	55,797	2,447	185	1	0.3			0.0081	0.0158	0.0094	0.0042	0.0055	0.0048	0.0069	0.0148	0.0072
450430010	1	33.36960	-79.29840	SC	Georgetown	Georgetown, SC	55,797	6,173	511	1	0.3			0.0102	0.0132	0.0102	0.0169	0.0265	0.0078	0.0033	0.0063	0.0050
450450008	2	34.84045	-82.40291	SC	Greenville	Greenville, SC	559,940	7,967	381	1	0.0			0.0071	0.0125	0.0066	0.0050	0.0120	0.0060	0.0049	0.0018	0.0018
450452002	1	34.94165	-82.22961	SC	Greenville	Greenville, SC	559,940	7,266	494	1	0.0			0.0000	0.0000	0.0000	0.0006	0.0018	0.0018	0.0000	0.0000	0.0000
450470001	1	34.18111	-82.15224	SC	Greenwood	Greenwood, SC	66,271	7,853	667	1	0.0			0.0053	0.0086	0.0082	0.0063	0.0112	0.0106	0.0021	0.0048	0.0036
450470002	1	34.16520	-82.16048	SC	Greenwood	Greenwood, SC	66,271	1,490	116		0.0			0.0100	0.0202	0.0168	0.0163	0.0320	0.0272	0.0094	0.0116	0.0115
450510002	2	33.70460	-78.87745	SC	Horry	Myrtle Beach-Conway-North Myrtl	196,629	4,510	227	1	0.0			0.0018	0.0032	0.0020	0.0020	0.0040	0.0034	0.0020	0.0053	0.0016
450630005	2	33.78560	-81.11978	SC	Lexington	Columbia, SC	647,158	736	66		0.0			0.0033	0.0052	0.0050						
450631002	2	33.96900	-81.06533	SC	Lexington	Columbia, SC	647,158	8,086	551	1	0.0			0.0179	0.0356	0.0090	0.0067	0.0125	0.0096	0.0036	0.0082	0.0036
450790006	4	34.00740	-81.02329	SC	Richland	Columbia, SC	647,158	17,143	574	1	0.0			0.0069	0.0090	0.0072						
450790007	2	34.09584	-80.96230	SC	Richland	Columbia, SC	647,158	4,405	233	1	0.0			0.0006	0.0018	0.0000	0.0007	0.0020	0.0020	0.0014	0.0042	0.0030
450790019	1	33.99330	-81.02414	SC	Richland	Columbia, SC	647,158	15,569	287	1	0.0			0.0097	0.0138	0.0104	0.0078	0.0144	0.0090	0.0096	0.0128	0.0122
450790021	1	33.81655	-80.78114	SC	Richland	Columbia, SC	647,158	123	10		0.0			0.0012	0.0038	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000

Appendix 2B

Table 2B-2. Pb-TSP monitoring site information and 1-year statistics

site	poc	lat	long	state	county_name	cbsa_name	cbsa_pop00	population near site (mile radius)	under age 5 pop. (mile radius)	urban	sum point / nonpt Pb EI TPY w/in 1 mile	source oriented?	prev. source oriented? (see end notes)	1-year metrics								
														max quarterly mean, 2003	max monthly mean, 2003	2nd max monthly mean, 2003	max quarterly mean, 2004	max monthly mean, 2004	2nd max monthly mean, 2004	max quarterly mean, 2005	max monthly mean, 2005	2nd max monthly mean, 2005
450830001	2	34.94774	-81.93255	SC	Spartanburg	Spartanburg, SC	253,791	7,505	552	1	0.0			0.0035	0.0060	0.0058	0.0026	0.0062	0.0054	0.0021	0.0048	0.0022
450850001	1	33.92423	-80.33774	SC	Sumter	Sumter, SC	104,646	4,990	407	1	0.0			0.0064	0.0108	0.0066	0.0047	0.0104	0.0060	0.0044	0.0090	0.0034
450910005	1	34.96303	-81.00085	SC	York	Charlotte-Gastonia-Concord, NC-S(	1,330,448	3,453	221	1	0.0			0.0042	0.0082	0.0050	0.0021	0.0058	0.0032	0.0008	0.0017	0.0016
470930027	1	35.98306	-83.95222	TN	Knox	Knoxville, TN	616,079	8,586	826	1	5.8	1		0.0100	0.0100	0.0100	0.0233	0.0400	0.0400	0.0100	0.0100	0.0100
470931017	1	35.97500	-83.95444	TN	Knox	Knoxville, TN	616,079	7,817	763	1	5.8	1		0.0100	0.0100	0.0100	0.0193	0.0375	0.0240	0.0100	0.0100	0.0100
471570044	1	35.08750	-90.07250	TN	Shelby	Memphis, TN-MS-AR	1,205,204	6,730	548	1	0.0	1	1	0.0100	0.0100	0.0100	0.0100	0.0100	0.0100			
471633001	1	36.52556	-82.27333	TN	Sullivan	Kingsport-Bristol-Bristol, TN-VA	298,484	942	65		0.4	1		0.1515	0.1940	0.1718	0.1577	0.2843	0.1488	0.1959	0.2360	0.2300
471633002	3	36.52472	-82.26806	TN	Sullivan	Kingsport-Bristol-Bristol, TN-VA	298,484	942	65		0.4	1		0.0719	0.0846	0.0730	0.1024	0.1550	0.0840	0.1463	0.2920	0.0933
471633003	1	36.52806	-82.26833	TN	Sullivan	Kingsport-Bristol-Bristol, TN-VA	298,484	942	65		0.4	1		0.0679	0.0844	0.0810	0.1259	0.2322	0.0750	0.0739	0.1260	0.0830
471870100	2	35.80222	-86.66028	TN	Williamson	Nashville-Davidson--Murfreesboro,	1,311,789	165	10		2.6	1		0.9867	1.9120	0.8200	0.1287	0.1960	0.1800			
471870102	2	35.80222	-86.66028	TN	Williamson	Nashville-Davidson--Murfreesboro,	1,311,789	165	10		2.6	1		0.6953	0.9460	0.6000	0.0887	0.1320	0.1100			
471871101	1	35.79944	-86.66500	TN	Williamson	Nashville-Davidson--Murfreesboro,	1,311,789	165	10		2.6	1		0.0853	0.1160	0.1120	0.3027	0.7020	0.1820			
480610006	1	25.89251	-97.49382	TX	Cameron	Brownsville-Harlingen, TX	335,227	14,803	1,422	1	0.0			0.0085	0.0090	0.0090	0.0076	0.0080	0.0078	0.0040	0.0042	0.0040
480850003	1	33.14250	-96.82472	TX	Collin	Dallas-Fort Worth-Arlington, TX	5,161,544	3,837	415		3.2	1		0.3006	0.4436	0.3518	0.2473	0.3220	0.2854	0.3453	0.7954	0.4396
480850007	2	33.14722	-96.82556	TX	Collin	Dallas-Fort Worth-Arlington, TX	5,161,544	3,837	415	1	3.2	1		0.1337	0.2458	0.2223	0.1241	0.1902	0.1728	0.2111	0.4760	0.3006
480850009	1	33.14472	-96.82889	TX	Collin	Dallas-Fort Worth-Arlington, TX	5,161,544	3,837	415		3.2	1		0.6600	0.8914	0.6658	0.5926	0.7524	0.6670	0.6982	0.9692	0.7368
481130018	1	32.74556	-96.78250	TX	Dallas	Dallas-Fort Worth-Arlington, TX	5,161,544	6,451	491	1	0.0			0.0318	0.0640	0.0250	0.0804	0.2338	0.0500	0.0467	0.0880	0.0680
481130057	2	32.77890	-96.87306	TX	Dallas	Dallas-Fort Worth-Arlington, TX	5,161,544	4,591	578	1	0.0			0.0611	0.1016	0.0708	0.0447	0.1029	0.0913	0.0563	0.0796	0.0700
481130066	2	32.73972	-96.78278	TX	Dallas	Dallas-Fort Worth-Arlington, TX	5,161,544	8,270	622	1	0.0	1	1	0.0178	0.0260	0.0217	0.0209	0.0420	0.0280			
481410033	1	31.77694	-106.50167	TX	El Paso	El Paso, TX	679,622	13,680	1,005	1	0.0			0.0585	0.0600	0.0540				0.0147	0.0240	0.0160
482011034	4	29.76799	-95.22058	TX	Harris	Houston-Sugar Land-Baytown, TX	4,715,407	14,785	1,770	1	0.0			0.0136	0.0230	0.0140	0.0220	0.0478	0.0104	0.0054	0.0073	0.0058
484790016	1	27.51083	-99.51972	TX	Webb	Laredo, TX	193,117	14,880	1,441	1	0.0			0.0142	0.0230	0.0147	0.0156	0.0206	0.0202	0.0163	0.0214	0.0196
490351001	1	40.70861	-112.09472	UT	Salt Lake	Salt Lake City, UT	968,858	215	23	1	0.0			0.0628	0.1188	0.0752	0.0718	0.1057	0.1016	0.0762	0.1072	0.1032
721270003	1	18.44917	-66.05306	PR	San Juan	San Juan-Caguas-Guaynabo, PR	2,509,007	319	5	1				0.0042	0.0125	0.0000	0.0000	0.0000	0.0000	0.0100	0.0120	0.0120

* These sites were classified as "previous" source-oriented but because production (and related lead emissions) at the associated source was not terminated until December, 2003, only data for 2004-2005 were considered for the "previous" source oriented characterization.

Data for 2004-2005 did not meet completeness criteria..

Table 2B-3. Pb-TSP monitoring site distribution statistics

All sites

	n	min	pct5	pct10	pct15	pct20	pct25	pct30	pct35	pct40	pct45	median	mean	pct55	pct60	pct65	pct70	pct75	pct80	pct85	pct90	pct95	max
annual mean	189	0.0000	0.0010	0.0019	0.0032	0.0042	0.0052	0.0071	0.0097	0.0114	0.0143	0.0166	0.0934	0.0203	0.0272	0.0316	0.0396	0.0606	0.0957	0.1332	0.2527	0.4778	2.6732
max quarter mean	189	0.0000	0.0031	0.0041	0.0063	0.0071	0.0100	0.0126	0.0179	0.0224	0.0254	0.0299	0.1738	0.0367	0.0495	0.0627	0.0820	0.1259	0.1857	0.2667	0.4657	0.8761	4.0931
max monthly mean	189	0.0000	0.0054	0.0080	0.0100	0.0112	0.0140	0.0200	0.0288	0.0320	0.0380	0.0430	0.3079	0.0503	0.0880	0.1000	0.1460	0.2200	0.2955	0.4760	0.9100	1.6900	5.7750
2nd max monthly mean	189	0.0000	0.0035	0.0051	0.0072	0.0100	0.0117	0.0140	0.0196	0.0240	0.0280	0.0340	0.2066	0.0380	0.0440	0.0726	0.1000	0.1428	0.2360	0.3100	0.5300	0.9927	5.0220
average of 3 overall highest monthly means	189	0.0000	0.0043	0.0060	0.0075	0.0101	0.0122	0.0147	0.0228	0.0279	0.0320	0.0373	0.2253	0.0400	0.0500	0.0811	0.1033	0.1496	0.2501	0.3418	0.6013	1.1597	4.2890
average of 3 annual max monthly means	189	0.0000	0.0042	0.0058	0.0080	0.0100	0.0112	0.0150	0.0203	0.0252	0.0299	0.0344	0.1942	0.0400	0.0496	0.0753	0.1073	0.1380	0.2093	0.3100	0.5390	1.0540	2.8611

Source-oriented sites

	n	min	pct5	pct10	pct15	pct20	pct25	pct30	pct35	pct40	pct45	median	mean	pct55	pct60	pct65	pct70	pct75	pct80	pct85	pct90	pct95	max
annual mean	60	0.0072	0.0095	0.0142	0.0229	0.0375	0.0440	0.0616	0.0775	0.0933	0.1122	0.1253	0.2596	0.1455	0.1815	0.2281	0.2549	0.2655	0.3327	0.4869	0.5866	0.9109	2.6732
max quarter mean	60	0.0100	0.0180	0.0221	0.0309	0.0731	0.0880	0.1206	0.1502	0.1829	0.2064	0.2470	0.4781	0.2800	0.3272	0.3526	0.5107	0.6866	0.7167	0.8930	1.2823	1.6992	4.0931
max monthly mean	60	0.0100	0.0311	0.0378	0.0420	0.1000	0.1580	0.1814	0.2311	0.2881	0.3577	0.4263	0.8572	0.5200	0.6320	0.7663	0.9280	1.0307	1.4577	1.7150	2.1401	3.4282	5.7750
2nd max monthly mean	60	0.0100	0.0205	0.0310	0.0380	0.0871	0.1070	0.1484	0.1690	0.2230	0.2670	0.2943	0.5738	0.3485	0.4336	0.4568	0.5645	0.7310	0.9289	1.0669	1.3655	2.0977	5.0220

Not source-oriented sites

	n	min	pct5	pct10	pct15	pct20	pct25	pct30	pct35	pct40	pct45	median	mean	pct55	pct60	pct65	pct70	pct75	pct80	pct85	pct90	pct95	max
annual mean	129	0.0000	0.0006	0.0014	0.0021	0.0028	0.0038	0.0045	0.0051	0.0057	0.0081	0.0100	0.0162	0.0113	0.0142	0.0153	0.0175	0.0214	0.0272	0.0308	0.0372	0.0433	0.1497
max quarter mean	129	0.0000	0.0022	0.0033	0.0042	0.0060	0.0067	0.0080	0.0100	0.0114	0.0138	0.0179	0.0322	0.0229	0.0253	0.0280	0.0343	0.0386	0.0495	0.0613	0.0773	0.1030	0.2493
max monthly mean	129	0.0000	0.0051	0.0062	0.0080	0.0090	0.0105	0.0120	0.0140	0.0160	0.0220	0.0290	0.0525	0.0320	0.0360	0.0404	0.0480	0.0600	0.0880	0.1000	0.1387	0.2300	0.3560
2nd max monthly mean	129	0.0000	0.0025	0.0040	0.0052	0.0060	0.0080	0.0100	0.0115	0.0125	0.0148	0.0196	0.0358	0.0230	0.0270	0.0300	0.0360	0.0390	0.0440	0.0630	0.0880	0.1140	0.2980

Previous source-oriented sites

	n	min	pct5	pct10	pct15	pct20	pct25	pct30	pct35	pct40	pct45	median	mean	pct55	pct60	pct65	pct70	pct75	pct80	pct85	pct90	pct95	max
annual mean	9	0.0090	0.0090	0.0090	0.0100	0.0100	0.0203	0.0203	0.0362	0.0362	0.0477	0.0477	0.0595	0.0477	0.0799	0.0799	0.1000	0.1000	0.1105	0.1105	0.1214	0.1214	0.1214
max quarter mean	9	0.0100	0.0100	0.0100	0.0209	0.0209	0.0350	0.0350	0.0550	0.0550	0.1000	0.1000	0.1206	0.1000	0.1027	0.1027	0.1654	0.1654	0.2367	0.2367	0.3600	0.3600	0.3600
max monthly mean	9	0.0100	0.0100	0.0100	0.0380	0.0380	0.0420	0.0420	0.1000	0.1000	0.1000	0.1000	0.1942	0.1000	0.1400	0.1400	0.3080	0.3080	0.4500	0.4500	0.5600	0.5600	0.5600
2nd max monthly mean	9	0.0100	0.0100	0.0100	0.0280	0.0280	0.0360	0.0360	0.0940	0.0940	0.1000	0.1000	0.1467	0.1000	0.1025	0.1025	0.2200	0.2200	0.2600	0.2600	0.4700	0.4700	0.4700

Urban sites

	n	min	pct5	pct10	pct15	pct20	pct25	pct30	pct35	pct40	pct45	median	mean	pct55	pct60	pct65	pct70	pct75	pct80	pct85	pct90	pct95	max
annual mean	140	0.0001	0.0012	0.0021	0.0032	0.0045	0.0052	0.0074	0.0097	0.0112	0.0138	0.0149	0.0594	0.0168	0.0187	0.0230	0.0304	0.0365	0.0404	0.0780	0.1200	0.2601	1.4501
max quarter mean	140	0.0006	0.0032	0.0042	0.0067	0.0080	0.0104	0.0131	0.0174	0.0214	0.0247	0.0260	0.1100	0.0300	0.0364	0.0405	0.0612	0.0766	0.0979	0.1534	0.2430	0.4312	1.9277
max monthly mean	140	0.0018	0.0062	0.0081	0.0103	0.0120	0.0149	0.0204	0.0287	0.0315	0.0360	0.0400	0.1958	0.0440	0.0502	0.0800	0.1000	0.1164	0.1814	0.2469	0.4050	0.8560	3.5680
2nd max monthly mean	140	0.0000	0.0040	0.0059	0.0080	0.0100	0.0118	0.0145	0.0192	0.0220	0.0253	0.0305	0.1295	0.0355	0.0385	0.0430	0.0670	0.0870	0.1056	0.2100	0.2930	0.5645	2.2993

Urban sites, located in MSA's ≥ 1 million population

	n	min	pct5	pct10	pct15	pct20	pct25	pct30	pct35	pct40	pct45	median	mean	pct55	pct60	pct65	pct70	pct75	pct80	pct85	pct90	pct95	max
annual mean	91	0.0006	0.0026	0.0042	0.0051	0.0075	0.0090	0.0103	0.0113	0.0142	0.0150	0.0178	0.0711	0.0205	0.0225	0.0276	0.0315	0.0368	0.0396	0.0563	0.1000	0.3711	1.4501
max quarter mean	91	0.0033	0.0060	0.0071	0.0100	0.0114	0.0133	0.0197	0.0220	0.0252	0.0267	0.0300	0.1343	0.0353	0.0400	0.0567	0.0667	0.0773	0.0957	0.1537	0.2367	0.8683	1.9277
max monthly mean	91	0.0067	0.0082	0.0110	0.0124	0.0160	0.0200	0.0290	0.0340	0.0360	0.0400	0.0440	0.2442	0.0500	0.0601	0.0960	0.1029	0.1460	0.1878	0.2338	0.4760	1.7400	3.5680
2nd max monthly mean	91	0.0000	0.0067	0.0080	0.0103	0.0120	0.0160	0.0200	0.0230	0.0255	0.0315	0.0360	0.1530	0.0380	0.0406	0.0500	0.0778	0.0880	0.1016	0.2100	0.2880	0.9278	2.2993

Urban sites, located in MSA's < 1 million population

	n	min	pct5	pct10	pct15	pct20	pct25	pct30	pct35	pct40	pct45	median	mean	pct55	pct60	pct65	pct70	pct75	pct80	pct85	pct90	pct95	max
annual mean	49	0.0001	0.0006	0.0010	0.0014	0.0018	0.0025	0.0030	0.0046	0.0048	0.0065	0.0100	0.0378	0.0121	0.0143	0.0156	0.0175	0.0305	0.0779	0.1000	0.1332	0.1578	0.2944
max quarter mean	49	0.0006	0.0020	0.0026	0.0034	0.0037	0.0063	0.0069	0.0085	0.0126	0.0166	0.0179	0.0649	0.0224	0.0248	0.0279	0.0386	0.0585	0.1000	0.1467	0.2493	0.2667	0.4657
max monthly mean	49	0.0018	0.0048	0.0053	0.0063	0.0072	0.0102	0.0108	0.0144	0.0209	0.0286	0.0310	0.1060	0.0320	0.0400	0.0404	0.0800	0.0920	0.1387	0.2600	0.3560	0.6100	0.8020
2nd max monthly mean	49	0.0018	0.0025	0.0034	0.0042	0.0054	0.0072	0.0090	0.0106	0.0132	0.1700	0.0200	0.0861	0.0240	0.0300	0.0310	0.0400	0.0560	0.1314	0.2100	0.2980	0.5180	0.5991

Appendix 2B

Table 2B-4. Pb-TSP metric correlations

Statistic (Q = quarterly, M = monthly)		All sites					
		annual mean, 2003-2005	max Q mean, 2003-2005	max M mean, 2003-2005	2nd max M mean, 2003-2005	avg. of 3 overall highest M means, 2003-2005	avg. of 3 annual max M means, 2003-2005
number of sites		189	189	189	189	189	189
mean ($\mu g/m^3$)		0.0934	0.1738	0.3079	0.2066	0.2253	0.1942
Correlation coeficients	annual mean, 2003-2005	1.00	0.96	0.88	0.97	0.94	0.90
	max Q mean, 2003-2005		1.00	0.94	0.99	0.98	0.94
	max M mean, 2003-2005			1.00	0.92	0.98	0.97
	2nd max M mean, 2003-2005				1.00	0.97	0.93
	average of 3 overall highest M means, 2003-2005					1.00	0.98
	average of 3 annual max M means, 2003-2005						1.00

Statistic (Q = quarterly, M = monthly)		Urban sites					
		annual mean, 2003-2005	max Q mean, 2003-2005	max M mean, 2003-2005	2nd max M mean, 2003-2005	avg. of 3 overall highest M means, 2003-2005	avg. of 3 annual max M means, 2003-2005
number of sites		140	140	140	140	140	140
mean ($\mu g/m^3$)		0.0594	0.1100	0.1958	0.1295	0.1455	0.1350
Correlation coeficients	annual mean, 2003-2005	1.00	0.95	0.83	0.97	0.94	0.94
	max Q mean, 2003-2005		1.00	0.93	0.99	0.99	0.98
	max M mean, 2003-2005			1.00	0.88	0.97	0.96
	2nd max M mean, 2003-2005				1.00	0.97	0.97
	average of 3 overall highest M means, 2003-2005					1.00	1.00
	average of 3 annual max M means, 2003-2005					1.00	1.00

Statistic (Q = quarterly, M = monthly)		Source-oriented sites					
		annual mean, 2003-2005	max Q mean, 2003-2005	max M mean, 2003-2005	2nd max M mean, 2003-2005	avg. of 3 overall highest M means, 2003-2005	avg. of 3 annual max M means, 2003-2005
number of sites		60	60	60	60	60	60
mean ($\mu g/m^3$)		0.2596	0.4781	0.8572	0.5738	0.6259	0.5333
Correlation coeficients	annual mean, 2003-2005	1.00	0.95	0.85	0.96	0.93	0.88
	max Q mean, 2003-2005		1.00	0.93	0.99	0.98	0.92
	max M mean, 2003-2005			1.00	0.89	0.97	0.96
	2nd max M mean, 2003-2005				1.00	0.97	0.90
	average of 3 overall highest M means, 2003-2005					1.00	0.97
	average of 3 annual max M means, 2003-2005						1.00

Statistic (Q = quarterly, M = monthly)		Urban sites in CBSA's ≥ 1M population					
		annual mean, 2003-2005	max Q mean, 2003-2005	max M mean, 2003-2005	2nd max M mean, 2003-2005	avg. of 3 overall highest M means, 2003-2005	avg. of 3 annual max M means, 2003-2005
number of sites		91	91	91	91	91	91
mean ($\mu g/m^3$)		0.0711	0.1343	0.2442	0.1530	0.1762	0.1634
Correlation coeficients	annual mean, 2003-2005	1.00	0.95	0.82	0.97	0.93	0.94
	max Q mean, 2003-2005		1.00	0.93	0.99	0.99	0.99
	max M mean, 2003-2005			1.00	0.87	0.96	0.96
	2nd max M mean, 2003-2005				1.00	0.97	0.96
	average of 3 overall highest M means, 2003-2005					1.00	1.00
	average of 3 annual max M means, 2003-2005						1.00

Table 2B-5. Pb-TSP metric ratio distribution statistics

TSP Category	Ratio	Sites	min	pct5	pct10	pct15	pct20	pct25	pct30	pct35	pct40	pct45	median	mean	pct55	pct60	pct65	pct70	pct75	pct80	pct85	pct90	pct95	max
All sites	ratio of max quarterly mean to annual mean	189	1.0000	1.1135	1.2080	1.2852	1.3433	1.3848	1.4837	1.5299	1.6127	1.7079	1.7846	2.3541	1.8893	2.0025	2.1421	2.3164	2.5474	2.7853	3.2023	3.9233	5.9868	12.0000
	ratio of max monthly mean to annual mean		1.0000	1.3553	1.5556	1.8176	1.9537	2.1475	2.2817	2.4036	2.5471	2.6265	2.8310	4.4159	2.9634	3.5018	4.0128	4.4273	4.9871	5.7675	6.5038	8.5462	11.8424	39.0000
	ratio of 2nd max monthly mean to annual mean		0.0000	1.1211	1.3057	1.4296	1.5822	1.6784	1.7597	1.8311	1.9092	2.0346	2.1246	2.5728	2.2033	2.3015	2.4453	2.5908	2.7650	3.0986	3.5200	3.9800	6.6439	12.1935
Source-oriented sites	ratio of max quarterly mean to annual mean	60	1.0000	1.0787	1.2484	1.3167	1.3529	1.3966	1.5077	1.5205	1.5559	1.7079	1.7571	2.0471	1.8085	1.9070	1.9759	2.1285	2.3486	2.4706	2.7642	3.2865	3.8592	7.5516
	ratio of max monthly mean to annual mean		1.0000	1.4735	1.8318	1.9768	2.1641	2.2716	2.3528	2.5202	2.5824	2.7379	2.9086	3.7485	3.0625	3.5073	3.6900	4.0401	4.5092	4.6536	5.3012	6.2826	10.2029	13.5518
	ratio of 2nd max monthly mean to annual mean		1.0000	1.4088	1.6543	1.6815	1.7864	1.8292	1.8838	1.9438	2.0124	2.1000	2.1723	2.4356	2.2784	2.4017	2.5034	2.5293	2.6395	2.7960	3.3263	3.5193	3.8639	9.8590
Non-source-oriented sites	ratio of max quarterly mean to annual mean	129	1.0000	1.1368	1.2034	1.2648	1.3095	1.3826	1.4753	1.5591	1.6332	1.7079	1.8151	2.4980	1.9665	2.0293	2.2498	2.4153	2.6899	2.9390	3.4555	4.1647	7.3577	12.0000
	ratio of max monthly mean to annual mean		1.0000	1.3140	1.4769	1.6578	1.8680	2.0164	2.2496	2.3671	2.5200	2.5851	2.7967	4.7287	2.9508	3.4823	4.0858	4.5723	5.5927	6.3223	7.6473	9.1396	12.1935	39.0000
	ratio of 2nd max monthly mean to annual mean		0.0000	1.0746	1.2445	1.3294	1.4296	1.5582	1.6657	1.7556	1.8372	1.9200	2.1185	2.6371	2.1818	2.2633	2.3912	2.6316	2.8245	3.2174	3.8274	4.5000	7.5267	12.1935
Previous source-oriented sites	ratio of max quarterly mean to annual mean	13	1.0000	1.0000	1.0000	1.0000	1.0000	1.2844	1.2844	1.4961	1.4961	1.5195	1.5195	2.2049	1.5195	1.7266	1.7266	1.9492	1.9492	2.3170	2.3170	7.5516	7.5516	7.5516
	ratio of max monthly mean to annual mean		1.0000	1.0000	1.0000	1.0000	1.0000	1.7515	1.7515	1.8746	1.8746	2.7626	2.7626	3.4751	2.7626	2.7863	2.7863	3.7062	3.7062	4.6478	4.6478	11.7469	11.7469	11.7469
	ratio of 2nd max monthly mean to annual mean		1.0000	1.0000	1.0000	1.0000	1.0000	1.7759	1.7759	2.1414	2.1414	2.5969	2.5969	4.9066	2.5969	3.0986	3.0986	8.4131	8.4131	9.8590	9.8590	14.2747	14.2747	14.2747
Urban sites	ratio of max quarterly mean to annual mean	140	1.0000	1.1605	1.2504	1.3070	1.3536	1.4227	1.5010	1.5712	1.6427	1.7167	1.7773	2.3313	1.8683	2.0072	2.1508	2.3167	2.5890	2.7581	3.1770	3.9279	5.6878	12.0000
	ratio of max monthly mean to annual mean		1.0000	1.3391	1.5925	1.7771	1.9220	2.1133	2.2936	2.4105	2.5423	2.6341	2.8310	4.2395	2.9518	3.4976	4.0307	4.4998	5.1122	5.8364	6.6889	8.4122	11.7723	36.0000
	ratio of 2nd max monthly mean to annual mean		0.0000	1.1889	1.3271	1.4700	1.6053	1.6784	1.7660	1.8606	1.9967	2.1038	2.1650	2.6532	2.2452	2.3637	2.5051	2.6213	2.7859	3.0213	3.7220	4.1463	7.4078	12.1935
Urban sites in CBSAs ≥ 1M population	ratio of max quarterly mean to annual mean	91	1.0000	1.1077	1.2080	1.2745	1.3095	1.3759	1.4753	1.5167	1.6157	1.6829	1.7366	2.3159	1.7900	1.8451	2.0496	2.3164	2.4317	2.7033	3.0063	3.8141	7.3577	12.0000
	ratio of max monthly mean to annual mean		1.0000	1.3046	1.4769	1.6578	1.8346	1.9910	2.2676	2.3690	2.5370	2.5619	2.6726	4.0747	2.8714	2.9508	3.5868	4.0253	4.6478	5.6357	6.4892	8.0000	9.3770	36.0000
	ratio of 2nd max monthly mean to annual mean		0.0000	1.0746	1.3057	1.4089	1.4965	1.5855	1.6553	1.7491	1.7890	1.8960	2.0982	2.4155	2.1414	2.2633	2.3912	2.5004	2.5969	2.7659	3.0986	3.6621	7.2888	9.8590
Urban sites in CBSAs < 1M population	ratio of max quarterly mean to annual mean	49	1.0000	1.2648	1.3522	1.3838	1.4152	1.5299	1.5608	1.6127	1.6901	1.9505	2.0025	2.3597	2.0091	2.1715	2.2498	2.6223	2.7241	3.1395	3.4555	3.9762	4.8718	7.6772
	ratio of max monthly mean to annual mean		1.0000	1.4392	1.7365	1.9615	2.0898	2.2033	2.3056	2.5035	2.6417	2.9528	3.1207	4.5456	3.5286	4.4055	4.5723	5.0000	5.5497	5.8680	7.3340	9.3326	12.1935	19.1113
	ratio of 2nd max monthly mean to annual mean		1.0000	1.3116	1.6698	1.7451	1.8787	1.9843	2.0346	2.1235	2.1673	2.2033	2.3435	3.0946	2.3684	2.6953	2.7642	2.9302	3.8199	3.9800	4.4010	5.5005	8.4269	12.1935

Table 2B-6. Pb-PM10 monitoring site information

site	poc	lat	long	state	county_name	cbsa_name	cbsa_pop00	urban	data completeness (complete periods)			3-year metrics		
									qtrs	years	months	annual mean	max quarterly mean	max monthly mean
080770017	1	39.06363	-108.56102	CO	Mesa	Grand Junction, CO	116,255	1	1	4	13	0.0049	0.0056	0.0085
110010043	1	38.91889	-77.01250	DC	District of Columbia	Washington-Arlington-Alexandria,	4,796,183	1	2	7	20	0.0048	0.0085	0.0097
120571065	5	27.89222	-82.53861	FL	Hillsborough	Tampa-St. Petersburg-Clearwater,	2,395,997	1	2	8	23	0.0062	0.0207	0.0469
120573002	5	27.96565	-82.23040	FL	Hillsborough	Tampa-St. Petersburg-Clearwater,	2,395,997		2	8	24	0.0035	0.0048	0.0075
121030018	5	27.78556	-82.74000	FL	Pinellas	Tampa-St. Petersburg-Clearwater,	2,395,997	1	1	4	12	0.0022	0.0030	0.0047
121030026	5	27.85004	-82.71459	FL	Pinellas	Tampa-St. Petersburg-Clearwater,	2,395,997	1	1	6	17	0.0023	0.0034	0.0045
130890002	1	33.68801	-84.29033	GA	DeKalb	Atlanta-Sandy Springs-Marietta, G	4,247,981	1	3	12	34	0.0026	0.0046	0.0106
170314201	6	42.14000	-87.79917	IL	Cook	Chicago-Naperville-Joliet, IL-IN-W	9,098,316	1	1	4	12	0.0060	0.0076	0.0094
211930003	1	37.28306	-83.22028	KY	Perry				3	12	34	0.0040	0.0066	0.0078
250250042	6	42.32944	-71.08278	MA	Suffolk	Boston-Cambridge-Quincy, MA-N	4,391,344	1	1	6	22	0.0049	0.0085	0.0151
261630033	1	42.30667	-83.14889	MI	Wayne	Detroit-Warren-Livonia, MI	4,452,557	1	3	12	35	0.0212	0.0390	0.0667
295100085	6	38.65630	-90.19810	MO	St. Louis (City)	St. Louis, MO-IL	2,721,491	1	2	10	30	0.0127	0.0170	0.0256
360850106	1	40.57811	-74.18430	NY	Richmond	New York-Northern New Jersey-L	18,323,002		1	4	11	0.0071	0.0117	0.0150
360850111	1	40.57997	-74.19872	NY	Richmond	New York-Northern New Jersey-L	18,323,002		1	4	11	0.0074	0.0123	0.0160
360850131	1	40.58806	-74.16882	NY	Richmond	New York-Northern New Jersey-L	18,323,002		1	4	10	0.0069	0.0115	0.0120
360850132	1	40.58061	-74.15158	NY	Richmond	New York-Northern New Jersey-L	18,323,002		1	4	11	0.0095	0.0223	0.0300
410390060	7	44.02631	-123.08374	OR	Lane	Eugene-Springfield, OR	322,959	1	1	3	9	0.0023	0.0032	0.0040
410510030	7	45.49742	-122.67467	OR	Multnomah	Portland-Vancouver-Beaverton, OR	1,927,881	1	1	4	11	0.0056	0.0104	0.0123
410510080	7	45.49667	-122.60222	OR	Multnomah	Portland-Vancouver-Beaverton, OR	1,927,881	1	2	7	22	0.0055	0.0088	0.0144
410510244	8	45.53500	-122.69889	OR	Multnomah	Portland-Vancouver-Beaverton, OR	1,927,881	1	2	7	21	0.0065	0.0098	0.0190
410510246	7	45.56130	-122.67878	OR	Multnomah	Portland-Vancouver-Beaverton, OR	1,927,881	1	2	8	23	0.0097	0.0273	0.0608
410610119	7	45.33897	-117.90480	OR	Union	La Grande, OR	24,530		2	7	20	0.0016	0.0027	0.0030
410670111	7	45.47020	-122.81585	OR	Washington	Portland-Vancouver-Beaverton, OR	1,927,881	1	1	3	11	0.0025	0.0032	0.0051
440070022	1	41.80795	-71.41500	RI	Providence	Providence-New Bedford-Fall Rive	1,582,997	1	3	12	36	0.0098	0.0547	0.1529
440070029	1	41.81644	-71.43790	RI	Providence	Providence-New Bedford-Fall Rive	1,582,997	1	1	4	13	0.0061	0.0092	0.0142
450250001	2	34.61537	-80.19879	SC	Chesterfield				2	8	20	0.0029	0.0049	0.0071
481390017	1	32.47361	-97.04250	TX	Ellis	Dallas-Fort Worth-Arlington, TX	5,161,544		1	6	17	0.0151	0.0211	0.0370
481410041	1	31.76054	-106.50045	TX	El Paso	El Paso, TX	679,622	1	1	4	12	0.0118	0.0167	0.0253
482011035	1	29.73371	-95.25759	TX	Harris	Houston-Sugar Land-Baytown, TX	4,715,407	1	3	12	36	0.0077	0.0106	0.0116
482011039	1	29.67005	-95.12849	TX	Harris	Houston-Sugar Land-Baytown, TX	4,715,407	1	3	12	31	0.0056	0.0113	0.0136
490110004	1	40.90297	-111.88447	UT	Davis	Ogden-Clearfield, UT	442,656	1	2	10	29	0.0059	0.0081	0.0111
530110030	7	45.64168	-122.68123	WA	Clark	Portland-Vancouver-Beaverton, OR	1,927,881	1	1	3	11	0.0032	0.0051	0.0061
530330080	1	47.56833	-122.30806	WA	King	Seattle-Tacoma-Bellevue, WA	3,043,878	1	3	11	32	0.0046	0.0085	0.0146
530630016	1	47.66083	-117.35722	WA	Spokane	Spokane, WA	417,939	1	1	4	12	0.0059	0.0108	0.0211
530630050	1	47.69545	-117.37030	WA	Spokane	Spokane, WA	417,939		1	4	12	0.0049	0.0090	0.0168
530630052	1	47.66512	-117.42909	WA	Spokane	Spokane, WA	417,939		1	4	12	0.0037	0.0055	0.0088
530630053	1	47.68220	-117.30480	WA	Spokane	Spokane, WA	417,939		1	4	12	0.0051	0.0078	0.0134
550270007	1	43.43500	-88.52778	WI	Dodge	Beaver Dam, WI	85,897		1	4	10	0.0054	0.0082	0.0153

Table 2B-7. Pb-PM10 monitoring site distribution statistics

All sites

	n	min	pct5	pct10	pct15	pct20	pct25	pct30	pct35	pct40	pct45	median	mean	pct55	pct60	pct65	pct70	pct75	pct80	pct85	pct90	pct95	max
annual mean	38	0.0016	0.0022	0.0023	0.0026	0.0032	0.0037	0.0046	0.0049	0.0049	0.0054	0.0056	0.0063	0.0056	0.0059	0.0061	0.0065	0.0071	0.0077	0.0097	0.0118	0.0151	0.0212
max quarter mean	38	0.0027	0.0030	0.0032	0.0046	0.0049	0.0055	0.0066	0.0078	0.0082	0.0085	0.0087	0.0117	0.0090	0.0098	0.0106	0.0113	0.0117	0.0167	0.0207	0.0223	0.0390	0.0547
max monthly mean	38	0.0030	0.0040	0.0047	0.0061	0.0075	0.0085	0.0094	0.0106	0.0116	0.0123	0.0135	0.0205	0.0142	0.0146	0.0151	0.0160	0.0190	0.0253	0.0300	0.0469	0.0667	0.1529

Urban sites

	n	min	pct5	pct10	pct15	pct20	pct25	pct30	pct35	pct40	pct45	median	mean	pct55	pct60	pct65	pct70	pct75	pct80	pct85	pct90	pct95	max
annual mean	25	0.0022	0.0023	0.0023	0.0025	0.0029	0.0046	0.0048	0.0049	0.0052	0.0056	0.0056	0.0064	0.0059	0.0060	0.0061	0.0062	0.0065	0.0087	0.0098	0.0118	0.0127	0.0212
max quarter mean	25	0.0030	0.0032	0.0032	0.0034	0.0049	0.0056	0.0076	0.0081	0.0085	0.0085	0.0088	0.0126	0.0092	0.0101	0.0106	0.0108	0.0113	0.0169	0.0207	0.0273	0.0390	0.0547
max monthly mean	25	0.0040	0.0045	0.0047	0.0051	0.0073	0.0094	0.0097	0.0106	0.0114	0.0123	0.0136	0.0235	0.0142	0.0145	0.0151	0.0190	0.0211	0.0254	0.0469	0.0608	0.0667	0.1529

Urban sites, located in MSA's ≥ 1 million population

	n	min	pct5	pct10	pct15	pct20	pct25	pct30	pct35	pct40	pct45	median	mean	pct55	pct60	pct65	pct70	pct75	pct80	pct85	pct90	pct95	max
annual mean	20	0.0022	0.0022	0.0024	0.0025	0.0029	0.0039	0.0047	0.0048	0.0052	0.0056	0.0056	0.0065	0.0058	0.0061	0.0061	0.0063	0.0071	0.0087	0.0097	0.0113	0.0170	0.0212
max quarter mean	20	0.0030	0.0031	0.0033	0.0040	0.0049	0.0064	0.0080	0.0085	0.0085	0.0087	0.0090	0.0136	0.0095	0.0101	0.0105	0.0109	0.0142	0.0189	0.0240	0.0332	0.0469	0.0547
max monthly mean	20	0.0045	0.0046	0.0049	0.0056	0.0077	0.0095	0.0101	0.0111	0.0120	0.0130	0.0139	0.0259	0.0143	0.0145	0.0148	0.0170	0.0223	0.0363	0.0539	0.0637	0.1098	0.1529

Urban sites, located in MSA's < 1 million population

	n	min	pct5	pct10	pct15	pct20	pct25	pct30	pct35	pct40	pct45	median	mean	pct55	pct60	pct65	pct70	pct75	pct80	pct85	pct90	pct95	max
annual mean	5	0.0023	0.0023	0.0023	0.0023	0.0036	0.0049	0.0049	0.0049	0.0054	0.0059	0.0059	0.0061	0.0059	0.0059	0.0059	0.0059	0.0059	0.0089	0.0118	0.0118	0.0118	0.0118
max quarter mean	5	0.0032	0.0032	0.0032	0.0032	0.0044	0.0056	0.0056	0.0056	0.0069	0.0081	0.0081	0.0089	0.0081	0.0095	0.0108	0.0108	0.0108	0.0137	0.0167	0.0167	0.0167	0.0167
max monthly mean	5	0.0040	0.0040	0.0040	0.0040	0.0062	0.0085	0.0085	0.0085	0.0098	0.0111	0.0111	0.0140	0.0111	0.0161	0.0211	0.0211	0.0211	0.0232	0.0253	0.0253	0.0253	0.0253

Table 2B-8. Pb-PM2.5 monitoring site information

site	poc	lat	long	state	county_name	cbsa_name	cbsa_pop00	urban	data completeness (complete periods)			3-year metrics		
									qtrs	years	months	annual mean	max quarterly mean	max monthly mean
010050002	5	31.66414	-85.60623	AL	Barbour	Eufaula, AL-GA	31,636		2	8	25	0.0026	0.0033	0.0053
010730023	5	33.55306	-86.81500	AL	Jefferson	Birmingham-Hoover, AL	1,052,238	1	3	12	36	0.0180	0.0296	0.0475
010731009	5	33.45972	-87.30556	AL	Jefferson	Birmingham-Hoover, AL	1,052,238		3	12	34	0.0021	0.0032	0.0044
010732003	5	33.49972	-86.92417	AL	Jefferson	Birmingham-Hoover, AL	1,052,238	1	3	12	36	0.0450	0.0967	0.2091
010890014	5	34.69083	-86.58306	AL	Madison	Huntsville, AL	342,376	1	3	12	34	0.0024	0.0040	0.0057
010970003	5	30.76972	-88.08750	AL	Mobile	Mobile, AL	399,843	1	3	12	36	0.0038	0.0060	0.0096
011011002	5	32.40694	-86.25639	AL	Montgomery	Montgomery, AL	346,528	1	3	12	34	0.0045	0.0083	0.0115
011030011	5	34.51861	-86.97694	AL	Morgan	Decatur, AL	145,867	1	3	11	30	0.0029	0.0042	0.0060
011130001	5	32.47639	-84.99917	AL	Russell	Columbus, GA-AL	281,768	1	1	3	9	0.0030	0.0037	0.0063
020200018	5	61.20667	-149.82083	AK	Anchorage Municipa	Anchorage, AK	319,605	1	1	6	17	0.0043	0.0067	0.0101
020900010	6	64.84111	-147.72000	AK	Fairbanks North Star	Fairbanks, AK	82,840	1	1	3	8	0.0034	0.0053	0.0070
040130019	5	33.48385	-112.14257	AZ	Maricopa	Phoenix-Mesa-Scottsdale, AZ	3,251,876	1	2	7	20	0.0030	0.0057	0.0100
040134009	5	33.40642	-112.14434	AZ	Maricopa	Phoenix-Mesa-Scottsdale, AZ	3,251,876		1	4	9	0.0062	0.0123	0.0228
040137003	5	33.28936	-112.15732	AZ	Maricopa	Phoenix-Mesa-Scottsdale, AZ	3,251,876		1	4	10	0.0027	0.0049	0.0067
040137020	5	33.47333	-111.85418	AZ	Maricopa	Phoenix-Mesa-Scottsdale, AZ	3,251,876		1	4	11	0.0026	0.0038	0.0058
040138006	5	33.43671	-112.09141	AZ	Maricopa	Phoenix-Mesa-Scottsdale, AZ	3,251,876	1	1	5	13	0.0042	0.0067	0.0084
040139997	7	33.50364	-112.09500	AZ	Maricopa	Phoenix-Mesa-Scottsdale, AZ	3,251,876	1	3	12	36	0.0027	0.0047	0.0069
040139998	5	33.45513	-111.99610	AZ	Maricopa	Phoenix-Mesa-Scottsdale, AZ	3,251,876	1	1	6	18	0.0033	0.0047	0.0075
040191028	5	32.29515	-110.98230	AZ	Pima	Tucson, AZ	843,746	1	3	12	35	0.0017	0.0022	0.0035
050030005	5	33.13944	-91.95000	AR	Ashley			1	2	9	21	0.0027	0.0055	0.0082
051190007	5	34.75611	-92.27583	AR	Pulaski	Little Rock-North Little Rock, AR	610,518	1	3	12	33	0.0029	0.0042	0.0061
051450001	5	35.24861	-91.71528	AR	White	Searcy, AR	67,165	1	2	9	22	0.0026	0.0046	0.0063
060070002	5	39.75750	-121.84222	CA	Butte	Chico, CA	203,171	1	3	12	36	0.0026	0.0039	0.0054
060190008	5	36.78139	-119.77222	CA	Fresno	Fresno, CA	799,407	1	3	12	36	0.0030	0.0050	0.0066
060250005	5	32.67611	-115.48333	CA	Imperial	El Centro, CA	142,361	1	3	12	36	0.0119	0.0172	0.0342
060290014	5	35.35611	-119.04028	CA	Kern	Bakersfield, CA	661,645	1	3	11	32	0.0026	0.0046	0.0061
060371103	5	34.06659	-118.22688	CA	Los Angeles	Los Angeles-Long Beach-Santa An	12,365,627	1	3	12	36	0.0053	0.0098	0.0228
060631009	5	39.80833	-120.47167	CA	Plumas			1	3	12	36	0.0025	0.0041	0.0054
060658001	5	33.99958	-117.41601	CA	Riverside	Riverside-San Bernardino-Ontario,	3,254,821	1	3	12	36	0.0058	0.0088	0.0151
060670006	5	38.61417	-121.36694	CA	Sacramento	Sacramento--Arden-Arcade--Rosev	1,796,857	1	3	12	36	0.0022	0.0031	0.0047
060670010	5	38.55833	-121.49194	CA	Sacramento	Sacramento--Arden-Arcade--Rosev	1,796,857	1	3	12	36	0.0029	0.0037	0.0052
060730003	5	32.79139	-116.94167	CA	San Diego	San Diego-Carlsbad-San Marcos, C	2,813,833	1	3	12	36	0.0039	0.0059	0.0078
060731002	5	33.12778	-117.07417	CA	San Diego	San Diego-Carlsbad-San Marcos, C	2,813,833	1	3	12	36	0.0035	0.0050	0.0064
060850005	5	37.34850	-121.89500	CA	Santa Clara	San Jose-Sunnyvale-Santa Clara, C	1,735,819	1	3	12	36	0.0026	0.0063	0.0138
060990005	5	37.64167	-120.99361	CA	Stanislaus	Modesto, CA	446,997	1	3	12	36	0.0033	0.0065	0.0090
061072002	5	36.33222	-119.29028	CA	Tulare	Visalia-Porterville, CA	368,021	1	3	12	36	0.0034	0.0046	0.0060
061112002	5	34.27750	-118.68472	CA	Ventura	Oxnard-Thousand Oaks-Ventura, C	753,197	1	3	12	33	0.0020	0.0032	0.0042
080010006	5	39.82574	-104.93699	CO	Adams	Denver-Aurora, CO	2,157,756	1	3	12	36	0.0077	0.0163	0.0185
080410011	5	38.83139	-104.82778	CO	El Paso	Colorado Springs, CO	537,484	1	3	12	34	0.0019	0.0028	0.0048
080670008	5	37.26861	-107.87500	CO	La Plata	Durango, CO	43,941	1	1	3	8	0.0014	0.0016	0.0024
080770003	5	39.09083	-108.56389	CO	Mesa	Grand Junction, CO	116,255	1	1	3	8	0.0015	0.0021	0.0031
080770017	5	39.06363	-108.56102	CO	Mesa	Grand Junction, CO	116,255	1	2	9	25	0.0023	0.0035	0.0056
081230008	5	40.20917	-104.82306	CO	Weld	Greeley, CO	180,936		3	12	36	0.0020	0.0034	0.0054
090090027	5	41.30111	-72.90278	CT	New Haven	New Haven-Milford, CT	824,008	1	2	8	20	0.0029	0.0043	0.0066
100010003	5	39.15500	-75.51806	DE	Kent	Dover, DE	126,697	1	3	12	34	0.0024	0.0038	0.0051
100032004	5	39.73944	-75.55806	DE	New Castle	Philadelphia-Camden-Wilmington,	5,687,147	1	3	12	32	0.0042	0.0084	0.0114
110010042	6	38.88083	-77.03250	DC	District of Columbia	Washington-Arlington-Alexandria,	4,796,183	1	1	6	18	0.0037	0.0058	0.0075
110010043	5	38.91889	-77.01250	DC	District of Columbia	Washington-Arlington-Alexandria,	4,796,183	1	3	12	36	0.0035	0.0063	0.0093
120330004	6	30.52500	-87.20417	FL	Escambia	Pensacola-Ferry Pass-Brent, FL	412,153	1	3	12	36	0.0019	0.0026	0.0042
120571075	5	28.05000	-82.37806	FL	Hillsborough	Tampa-St. Petersburg-Clearwater,	2,395,997	1	1	4	12	0.0023	0.0034	0.0052
120573002	5	27.96565	-82.23040	FL	Hillsborough	Tampa-St. Petersburg-Clearwater,	2,395,997		2	8	24	0.0027	0.0042	0.0069
120730012	5	30.43972	-84.34833	FL	Leon	Tallahassee, FL	320,304	1	3	12	36	0.0020	0.0034	0.0049
120861016	5	25.79417	-80.20611	FL	Miami-Dade	Miami-Fort Lauderdale-Miami Bea	5,007,564	1	3	12	36	0.0020	0.0068	0.0163
121030026	5	27.85004	-82.71459	FL	Pinellas	Tampa-St. Petersburg-Clearwater,	2,395,997	1	1	5	16	0.0025	0.0039	0.0088
130210007	5	32.77944	-83.64694	GA	Bibb	Macon, GA	222,368		3	12	34	0.0029	0.0069	0.0147

Table 2B-8. Pb-PM2.5 monitoring site information

site	poc	lat	long	state	county_name	cbsa_name	cbsa_pop00	urban	data completeness (complete periods)			3-year metrics		
									qtrs	years	months	annual mean	max quarterly mean	max monthly mean
130510017	5	32.09278	-81.14417	GA	Chatham	Savannah, GA	293,000	1	2	8	22	0.0017	0.0029	0.0041
130590001	5	33.94583	-83.37222	GA	Clarke	Athens-Clarke County, GA	166,079	1	3	12	29	0.0021	0.0029	0.0041
130690002	5	31.52430	-82.76510	GA	Coffee	Douglas, GA	45,022		3	12	30	0.0013	0.0022	0.0032
130890002	5	33.68750	-84.29028	GA	DeKalb	Atlanta-Sandy Springs-Marietta, G	4,247,981	1	3	12	36	0.0027	0.0042	0.0077
131150005	5	34.26333	-85.27250	GA	Floyd	Rome, GA	90,565		3	12	33	0.0023	0.0030	0.0040
132150011	5	32.43083	-84.93167	GA	Muscogee	Columbus, GA-AL	281,768	1	3	12	32	0.0036	0.0101	0.0086
132450091	5	33.43333	-82.02194	GA	Richmond	Augusta-Richmond County, GA-S	499,684	1	3	12	32	0.0025	0.0038	0.0067
132950002	5	34.96611	-85.29750	GA	Walker	Chattanooga, TN-GA	476,531	1	1	3	9	0.0033	0.0040	0.0051
150032004	5	21.39667	-157.97167	HI	Honolulu	Honolulu, HI	876,156	1	3	12	34	0.0010	0.0021	0.0031
160270004	5	43.56240	-116.56323	ID	Canyon	Boise City-Nampa, ID	464,840	1	3	12	36	0.0022	0.0046	0.0096
170310057	5	41.91473	-87.72273	IL	Cook	Chicago-Naperville-Joliet, IL-IN-V	9,098,316	1	3	12	35	0.0071	0.0115	0.0172
170310076	5	41.75137	-87.71375	IL	Cook	Chicago-Naperville-Joliet, IL-IN-V	9,098,316	1	3	12	36	0.0054	0.0063	0.0087
170314201	5	42.14000	-87.79917	IL	Cook	Chicago-Naperville-Joliet, IL-IN-V	9,098,316	1	3	12	36	0.0040	0.0054	0.0085
170434002	5	41.77120	-88.15250	IL	DuPage	Chicago-Naperville-Joliet, IL-IN-V	9,098,316	1	2	8	23	0.0047	0.0063	0.0072
171150013	5	39.86694	-88.92556	IL	Macon	Decatur, IL	114,706		3	12	35	0.0067	0.0142	0.0228
171192009	5	38.90278	-90.14306	IL	Madison	St. Louis, MO-IL	2,721,491	1	3	12	32	0.0090	0.0208	0.0413
180030004	5	41.09472	-85.10194	IN	Allen	Fort Wayne, IN	390,156	1	2	7	20	0.0257	0.1674	0.3091
180372001	5	38.39139	-86.92917	IN	Dubois	Jasper, IN	52,511	1	1	4	12	0.0042	0.0051	0.0063
180390003	5	41.66778	-85.96944	IN	Elkhart	Elkhart-Goshen, IN	182,791	1	1	4	12	0.0044	0.0048	0.0056
180650003	5	40.01167	-85.52361	IN	Henry	New Castle, IN	48,508		3	12	36	0.0037	0.0055	0.0074
180890022	5	41.60667	-87.30472	IN	Lake	Chicago-Naperville-Joliet, IL-IN-V	9,098,316	1	3	11	32	0.0097	0.0128	0.0204
180892004	5	41.58528	-87.47444	IN	Lake	Chicago-Naperville-Joliet, IL-IN-V	9,098,316	1	2	8	24	0.0090	0.0120	0.0244
180970078	5	39.81110	-86.11447	IN	Marion	Indianapolis-Carmel, IN	1,525,104	1	3	12	36	0.0048	0.0071	0.0087
181411008	5	41.69361	-86.23667	IN	St. Joseph	South Bend-Mishawaka, IN-MI	316,663	1	1	4	12	0.0042	0.0054	0.0072
181630012	5	38.02167	-87.56944	IN	Vanderburgh	Evansville, IN-KY	342,815	1	3	12	34	0.0031	0.0057	0.0080
191130037	5	42.00833	-91.67861	IA	Linn	Cedar Rapids, IA	237,230	1	3	12	35	0.0033	0.0044	0.0071
191530030	5	41.60306	-93.64306	IA	Polk	Des Moines-West Des Moines, IA	481,394	1	3	12	33	0.0027	0.0037	0.0058
191630015	5	41.53000	-90.58750	IA	Scott	Davenport-Moline-Rock Island, IA	376,019	1	3	12	33	0.0065	0.0084	0.0118
201730010	5	37.70111	-97.31389	KS	Sedgwick	Wichita, KS	571,166	1	2	9	18	0.0021	0.0032	0.0053
202090021	5	39.11750	-94.63556	KS	Wyandotte	Kansas City, MO-KS	1,836,038	1	3	12	36	0.0048	0.0066	0.0100
210190017	5	38.45917	-82.64056	KY	Boyd	Huntington-Ashland, WV-KY-OH	288,649	1	3	12	35	0.0043	0.0060	0.0096
210590005	5	37.78083	-87.07556	KY	Daviess	Owensboro, KY	109,875	1	1	4	12	0.0037	0.0044	0.0061
210590014	5	37.74111	-87.11806	KY	Daviess	Owensboro, KY	109,875	1	2	8	19	0.0023	0.0038	0.0036
210670012	5	38.06500	-84.50000	KY	Fayette	Lexington-Fayette, KY	408,326	1	3	12	36	0.0038	0.0066	0.0101
211110043	5	38.23222	-85.82528	KY	Jefferson	Louisville-Jefferson County, KY-I	1,161,975	1	3	12	36	0.0042	0.0070	0.0100
211110048	5	38.24056	-85.73167	KY	Jefferson	Louisville-Jefferson County, KY-I	1,161,975	1	3	12	36	0.0048	0.0071	0.0133
211170007	5	39.07250	-84.52500	KY	Kenton	Cincinnati-Middletown, OH-KY-I	2,009,632	1	3	12	36	0.0048	0.0106	0.0170
211250004	5	37.08722	-84.06333	KY	Laurel	London, KY	52,715	1	3	12	36	0.0037	0.0048	0.0095
211451004	5	37.06556	-88.63778	KY	McCracken	Paducah, KY-IL	98,765	1	3	12	36	0.0029	0.0043	0.0059
211930003	5	37.28306	-83.22028	KY	Perry				3	12	34	0.0041	0.0059	0.0079
212270007	5	36.99333	-86.41833	KY	Warren	Bowling Green, KY	104,166	1	3	12	35	0.0033	0.0056	0.0098
220150008	5	32.53417	-93.74972	LA	Bossier	Shreveport-Bossier City, LA	375,965		3	12	32	0.0046	0.0089	0.0147
220330009	5	30.46111	-91.17694	LA	East Baton Rouge	Baton Rouge, LA	705,973	1	3	11	31	0.0051	0.0101	0.0198
240030019	5	39.10111	-76.72944	MD	Anne Arundel	Baltimore-Towson, MD	2,552,994	1	2	7	17	0.0033	0.0061	0.0087
240053001	5	39.31083	-76.47444	MD	Baltimore	Baltimore-Towson, MD	2,552,994	1	2	9	26	0.0054	0.0080	0.0101
240330030	5	39.05528	-76.87833	MD	Prince George's	Washington-Arlington-Alexandria,	4,796,183	1	1	4	12	0.0039	0.0069	0.0099
250130008	5	42.19446	-72.55571	MA	Hampden	Springfield, MA	680,014		2	10	25	0.0025	0.0035	0.0045
250250042	6	42.32944	-71.08278	MA	Suffolk	Boston-Cambridge-Quincy, MA-N	4,391,344	1	3	12	35	0.0027	0.0039	0.0056
260050003	5	42.76778	-86.14861	MI	Allegan	Allegan, MI	105,665	1	3	11	35	0.0035	0.0055	0.0079
260330901	5	46.49361	-84.36417	MI	Chippewa	Sault Ste. Marie, MI	38,543	1	3	12	36	0.0023	0.0038	0.0046
260770008	5	42.27806	-85.54194	MI	Kalamazoo	Kalamazoo-Portage, MI	314,866	1	3	11	33	0.0050	0.0068	0.0097
260810020	5	42.98417	-85.67139	MI	Kent	Grand Rapids-Wyoming, MI	740,482	1	3	12	35	0.0048	0.0083	0.0104
261130001	5	44.31056	-84.89194	MI	Missaukee	Cadillac, MI	44,962		3	12	33	0.0022	0.0057	0.0102
261150005	5	41.76389	-83.47194	MI	Monroe	Monroe, MI	145,945		3	12	32	0.0042	0.0050	0.0074
261610008	5	42.24056	-83.59972	MI	Washtenaw	Ann Arbor, MI	322,895	1	2	10	30	0.0038	0.0060	0.0087

Table 2B-8. Pb-PM2.5 monitoring site information

site	poc	lat	long	state	county_name	cbsa_name	cbsa_pop00	urban	data completeness (complete periods)			3-year metrics		
									qtrs	years	months	annual mean	max quarterly mean	max monthly mean
261630001	5	42.22861	-83.20833	MI	Wayne	Detroit-Warren-Livonia, MI	4,452,557	1	3	12	36	0.0042	0.0051	0.0063
261630033	5	42.30667	-83.14889	MI	Wayne	Detroit-Warren-Livonia, MI	4,452,557	1	3	12	33	0.0118	0.0182	0.0329
270530963	5	44.95540	-93.25827	MN	Hennepin	Minneapolis-St. Paul-Bloomington	2,968,806	1	3	12	36	0.0031	0.0041	0.0072
270953051	5	46.20703	-93.75941	MN	Mille Lacs				3	11	31	0.0017	0.0023	0.0036
271095008	5	43.99691	-92.45037	MN	Olmsted	Rochester, MN	163,618	1	3	12	35	0.0027	0.0043	0.0067
271230871	5	44.96145	-93.03589	MN	Ramsey	Minneapolis-St. Paul-Bloomington	2,968,806	1	2	9	27	0.0042	0.0073	0.0084
280350004	5	31.32364	-89.28717	MS	Forrest	Hattiesburg, MS	123,812	1	3	12	35	0.0048	0.0128	0.0302
280430001	5	33.83611	-89.79722	MS	Grenada	Grenada, MS	23,263		3	11	31	0.0017	0.0032	0.0056
280470008	5	30.39014	-89.04972	MS	Harrison	Gulfport-Biloxi, MS	246,190	1	3	11	32	0.0023	0.0034	0.0062
280490018	5	32.29681	-90.18831	MS	Hinds	Jackson, MS	497,197	1	3	12	31	0.0046	0.0071	0.0112
280670002	5	31.68844	-89.13506	MS	Jones	Laurel, MS	83,107	1	3	12	35	0.0030	0.0073	0.0180
290470005	5	39.30306	-94.37639	MO	Clay	Kansas City, MO-KS	1,836,038		3	12	36	0.0026	0.0040	0.0050
290530001	5	38.79500	-92.91806	MO	Cooper				3	12	33	0.0020	0.0028	0.0041
290990012	5	38.43778	-90.36139	MO	Jefferson	St. Louis, MO-IL	2,721,491	1	3	12	36	0.0089	0.0126	0.0191
291860005	5	37.89694	-90.42222	MO	Ste Genevieve				3	12	34	0.0045	0.0084	0.0095
292070001	5	36.97000	-90.14000	MO	Stoddard				1	4	11	0.0034	0.0044	0.0068
295100085	6	38.65630	-90.19810	MO	St. Louis (City)	St. Louis, MO-IL	2,721,491	1	3	12	36	0.0095	0.0140	0.0192
300530018	5	48.38417	-115.54806	MT	Lincoln			1	3	12	35	0.0017	0.0029	0.0039
300630031	5	46.87491	-113.99525	MT	Missoula	Missoula, MT	95,802	1	3	12	36	0.0020	0.0035	0.0051
310550019	5	41.24722	-95.97556	NE	Douglas	Omaha-Council Bluffs, NE-IA	767,041	1	3	12	35	0.0030	0.0042	0.0055
320030560	5	36.15861	-115.11083	NV	Clark	Las Vegas-Paradise, NV	1,375,765	1	1	5	15	0.0025	0.0039	0.0061
320030561	5	36.16399	-115.11393	NV	Clark	Las Vegas-Paradise, NV	1,375,765	1	2	7	20	0.0025	0.0044	0.0086
320310016	5	39.52508	-119.80772	NV	Washoe	Reno-Sparks, NV	342,885	1	3	12	36	0.0024	0.0040	0.0060
330110020	5	43.00056	-71.46806	NH	Hillsborough	Manchester-Nashua, NH	380,841	1	3	12	34	0.0034	0.0053	0.0062
330150014	5	43.07528	-70.74806	NH	Rockingham	Boston-Cambridge-Quincy, MA-N	4,391,344	1	3	12	36	0.0024	0.0028	0.0036
340070003	5	39.92304	-75.09762	NJ	Camden	Philadelphia-Camden-Wilmington,	5,687,147	1	3	11	33	0.0042	0.0052	0.0069
340230006	6	40.47279	-74.42251	NJ	Middlesex	New York-Northern New Jersey-L	18,323,002	1	3	12	24	0.0045	0.0063	0.0114
340273001	5	40.78763	-74.67630	NJ	Morris	New York-Northern New Jersey-L	18,323,002	1	3	12	35	0.0027	0.0038	0.0059
340390004	5	40.64144	-74.20836	NJ	Union	New York-Northern New Jersey-L	18,323,002	1	3	12	36	0.0044	0.0059	0.0067
350010023	5	35.13426	-106.58551	NM	Bernalillo	Albuquerque, NM	729,649	1	2	8	22	0.0013	0.0020	0.0027
360050083	6	40.86586	-73.88075	NY	Bronx	New York-Northern New Jersey-L	18,323,002	1	3	12	36	0.0040	0.0059	0.0067
360050110	5	40.81616	-73.90207	NY	Bronx	New York-Northern New Jersey-L	18,323,002	1	3	12	36	0.0047	0.0064	0.0079
360290005	6	42.87684	-78.80988	NY	Erie	Buffalo-Niagra Falls, NY Metropo	1,170,111	1	3	12	36	0.0106	0.0157	0.0192
360310003	5	44.39309	-73.85892	NY	Essex				3	12	34	0.0015	0.0021	0.0028
360551007	5	43.14620	-77.54813	NY	Monroe	Rochester, NY	1,037,831	1	2	7	20	0.0031	0.0040	0.0048
360556001	5	43.16100	-77.60357	NY	Monroe	Rochester, NY	1,037,831	1	1	5	15	0.0031	0.0037	0.0045
360610062	1	40.72052	-74.00409	NY	New York	New York-Northern New Jersey-L	18,323,002	1	1	4	12	0.0070	0.0092	0.0190
360632008	1	43.08216	-79.00099	NY	Niagara	Buffalo-Niagra Falls, NY Metropo	1,170,111	1	1	4	11	0.0052	0.0063	0.0065
360710002	1	41.49947	-74.00973	NY	Orange	Poughkeepsie-Newburgh-Middleto	621,517	1	1	4	11	0.0034	0.0040	0.0053
360810124	6	40.73620	-73.82317	NY	Queens	New York-Northern New Jersey-L	18,323,002	1	3	12	36	0.0038	0.0055	0.0068
361010003	5	42.09071	-77.21025	NY	Steuben	Corning, NY	98,726		3	12	36	0.0028	0.0034	0.0042
361030001	1	40.74583	-73.42028	NY	Suffolk	New York-Northern New Jersey-L	18,323,002	1	1	4	11	0.0032	0.0039	0.0051
370210034	5	35.60972	-82.35083	NC	Buncombe	Asheville, NC	369,171	1	3	12	36	0.0019	0.0031	0.0052
370350004	5	35.72889	-81.36556	NC	Catawba	Hickory-Lenoir-Morganton, NC	341,851	1	3	12	35	0.0025	0.0036	0.0060
370510009	5	35.04142	-78.95311	NC	Cumberland	Fayetteville, NC	336,609	1	2	8	22	0.0021	0.0037	0.0057
370570002	5	35.81444	-80.26250	NC	Davidson	Thomasville-Lexington, NC	147,246	1	2	8	23	0.0032	0.0047	0.0087
370670022	5	36.11056	-80.22667	NC	Forsyth	Winston-Salem, NC	421,961	1	3	12	36	0.0026	0.0036	0.0063
370810013	5	36.10917	-79.80111	NC	Guilford	Greensboro-High Point, NC	643,430	1	2	9	23	0.0028	0.0043	0.0064
371070004	5	35.23146	-77.56879	NC	Lenoir	Kinston, NC	59,648		3	12	34	0.0026	0.0046	0.0062
371190041	5	35.24028	-80.78556	NC	Mecklenburg	Charlotte-Gastonia-Concord, NC-S	1,330,448	1	3	12	36	0.0029	0.0042	0.0052
371590021	5	35.55187	-80.39504	NC	Rowan	Salisbury, NC	130,340	1	1	4	11	0.0032	0.0040	0.0057
371830014	5	35.85611	-78.57417	NC	Wake	Raleigh-Cary, NC	797,071	1	3	12	34	0.0021	0.0038	0.0041
380150003	5	46.82543	-100.76821	ND	Burleigh	Bismarck, ND	94,719	1	3	12	36	0.0012	0.0023	0.0036
380171004	5	46.93375	-96.85535	ND	Cass	Fargo, ND-MN	174,367		3	12	36	0.0019	0.0027	0.0038
380530002	5	47.58120	-103.29950	ND	Mc Kenzie				3	12	35	0.0012	0.0026	0.0040

Table 2B-8. Pb-PM2.5 monitoring site information

site	poc	lat	long	state	county_name	cbsa_name	cbsa_pop00	urban	data completeness (complete periods)			3-year metrics		
									qtrs	years	months	annual mean	max quarterly mean	max monthly mean
390171004	5	39.53000	-84.39250	OH	Butler	Cincinnati-Middletown, OH-KY-IN	2,009,632	1	3	12	36	0.0092	0.0147	0.0273
390350038	6	41.47694	-81.68194	OH	Cuyahoga	Cleveland-Elyria-Mentor, OH	2,148,143	1	3	12	35	0.0120	0.0163	0.0282
390350060	5	41.49396	-81.67854	OH	Cuyahoga	Cleveland-Elyria-Mentor, OH	2,148,143	1	3	12	36	0.0123	0.0207	0.0270
390490081	6	40.08778	-82.95972	OH	Franklin	Columbus, OH	1,612,694	1	3	12	34	0.0038	0.0052	0.0073
390530003	5	38.94996	-82.10910	OH	Gallia	Point Pleasant, WV-OH	57,026		1	6	16	0.0043	0.0072	0.0085
390610040	5	39.12861	-84.50417	OH	Hamilton	Cincinnati-Middletown, OH-KY-IN	2,009,632	1	2	8	25	0.0056	0.0069	0.0113
390610042	5	39.10500	-84.55111	OH	Hamilton	Cincinnati-Middletown, OH-KY-IN	2,009,632	1	1	4	11	0.0079	0.0114	0.0286
390810017	5	40.36610	-80.61500	OH	Jefferson	Weirton-Steubenville, WV-OH	132,008	1	1	5	11	0.0127	0.0150	0.0193
390870010	5	38.51972	-82.66556	OH	Lawrence	Huntington-Ashland, WV-KY-OH	288,649	1	3	12	33	0.0059	0.0095	0.0137
390930016	5	41.43944	-82.16167	OH	Lorain	Cleveland-Elyria-Mentor, OH	2,148,143	1	1	4	10	0.0157	0.0244	0.0450
390933002	5	41.46306	-82.11444	OH	Lorain	Cleveland-Elyria-Mentor, OH	2,148,143		2	8	20	0.0238	0.0337	0.0465
390950026	5	41.62056	-83.64139	OH	Lucas	Toledo, OH	659,188	1	3	12	36	0.0035	0.0053	0.0069
390990014	5	41.09587	-80.65843	OH	Mahoning	Youngstown-Warren-Boardman, O	602,964	1	3	12	35	0.0131	0.0253	0.0382
391130031	5	39.75944	-84.14444	OH	Montgomery	Dayton, OH	848,153	1	3	11	25	0.0042	0.0079	0.0085
391510017	5	40.78667	-81.39444	OH	Stark	Canton-Massillon, OH	406,934	1	1	4	10	0.0114	0.0148	0.0186
391510020	5	40.80056	-81.37333	OH	Stark	Canton-Massillon, OH	406,934	1	2	8	24	0.0060	0.0082	0.0157
391530023	5	41.08806	-81.54167	OH	Summit	Akron, OH	694,960	1	3	11	29	0.0050	0.0069	0.0098
400450890	5	36.08518	-99.93494	OK	Ellis				3	12	34	0.0012	0.0019	0.0027
401091037	5	35.61278	-97.47222	OK	Oklahoma	Oklahoma City, OK	1,095,421	1	3	12	36	0.0022	0.0033	0.0046
401431127	5	36.20490	-95.97654	OK	Tulsa	Tulsa, OK	859,532	1	3	12	36	0.0031	0.0045	0.0056
410170120	5	44.06390	-121.31258	OR	Deschutes	Bend, OR	115,367	1	1	4	11	0.0014	0.0018	0.0021
410290133	5	42.31408	-122.87924	OR	Jackson	Medford, OR	181,269	1	3	12	35	0.0019	0.0029	0.0035
410390060	5	44.02631	-123.08374	OR	Lane	Eugene-Springfield, OR	322,959	1	3	12	35	0.0015	0.0025	0.0041
410510246	6	45.56130	-122.67878	OR	Multnomah	Portland-Vancouver-Beaverton, OR	1,927,881	1	3	12	36	0.0075	0.0182	0.0398
410610119	5	45.33897	-117.90480	OR	Union	La Grande, OR	24,530		2	8	20	0.0012	0.0020	0.0026
420010001	5	39.92000	-77.31000	PA	Adams	Gettysburg, PA	91,292		3	12	35	0.0037	0.0070	0.0082
420030008	6	40.46556	-79.96111	PA	Allegheny	Pittsburgh, PA	2,431,087	1	3	12	36	0.0112	0.0141	0.0252
420030021	5	40.41361	-79.94139	PA	Allegheny	Pittsburgh, PA	2,431,087	1	1	3	9	0.0073	0.0083	0.0129
420030064	6	40.32361	-79.86833	PA	Allegheny	Pittsburgh, PA	2,431,087	1	2	9	23	0.0143	0.0239	0.0356
420270100	5	40.81139	-77.87703	PA	Centre	State College, PA	135,758	1	3	12	35	0.0032	0.0043	0.0061
420290100	5	39.83444	-75.76861	PA	Chester	Philadelphia-Camden-Wilmington,	5,687,147		3	12	32	0.0046	0.0086	0.0105
420430401	5	40.24500	-76.84472	PA	Dauphin	Harrisburg-Carlisle, PA	509,074	1	3	12	34	0.0063	0.0122	0.0190
420450002	5	39.83556	-75.37250	PA	Delaware	Philadelphia-Camden-Wilmington,	5,687,147	1	3	12	34	0.0042	0.0057	0.0073
420490003	5	42.14175	-80.03861	PA	Erie	Erie, PA	280,843	1	3	12	34	0.0057	0.0153	0.0323
420692006	5	41.44278	-75.62306	PA	Lackawanna	Scranton--Wilkes-Barre, PA	560,625	1	3	12	33	0.0054	0.0087	0.0115
420710007	5	40.04667	-76.28333	PA	Lancaster	Lancaster, PA	470,658	1	3	12	35	0.0073	0.0175	0.0231
420950025	5	40.62806	-75.34111	PA	Northampton	Allentown-Bethlehem-Easton, PA-	740,395	1	3	12	36	0.0065	0.0095	0.0152
420990301	5	40.45694	-77.16556	PA	Perry	Harrisburg-Carlisle, PA	509,074		3	12	36	0.0035	0.0056	0.0084
421010004	7	40.00889	-75.09778	PA	Philadelphia	Philadelphia-Camden-Wilmington,	5,687,147	1	3	12	36	0.0052	0.0071	0.0090
421010136	5	39.92750	-75.22278	PA	Philadelphia	Philadelphia-Camden-Wilmington,	5,687,147	1	3	12	34	0.0038	0.0061	0.0104
421255001	5	40.44528	-80.42083	PA	Washington	Pittsburgh, PA	2,431,087		3	12	36	0.0050	0.0067	0.0084
421290008	5	40.30469	-79.50567	PA	Westmoreland	Pittsburgh, PA	2,431,087	1	3	12	35	0.0051	0.0070	0.0097
421330008	5	39.96528	-76.69944	PA	York	York-Hanover, PA	381,751	1	3	12	34	0.0058	0.0112	0.0169
440070022	5	41.80795	-71.41500	RI	Providence	Providence-New Bedford-Fall Rive	1,582,997	1	3	12	36	0.0065	0.0432	0.1103
440071010	5	41.84092	-71.36094	RI	Providence	Providence-New Bedford-Fall Rive	1,582,997	1	1	5	14	0.0030	0.0037	0.0051
450190046	5	32.94275	-79.65718	SC	Charleston	Charleston-North Charleston, SC	549,033		2	6	17	0.0019	0.0026	0.0039
450190049	5	32.79098	-79.95869	SC	Charleston	Charleston-North Charleston, SC	549,033	1	3	12	36	0.0022	0.0035	0.0048
450250001	5	34.61712	-80.19879	SC	Chesterfield				3	12	36	0.0021	0.0035	0.0044
450450009	5	34.90105	-82.31307	SC	Greenville	Greenville, SC	559,940	1	3	12	36	0.0026	0.0050	0.0060
450790019	5	33.99330	-81.02414	SC	Richland	Columbia, SC	647,158	1	3	12	34	0.0048	0.0092	0.0122
460990006	5	43.54429	-96.72644	SD	Minnehaha	Sioux Falls, SD	187,093	1	3	12	36	0.0022	0.0031	0.0052
470370023	5	36.17633	-86.73890	TN	Davidson	Nashville-Davidson--Murfreesboro	1,311,789	1	3	12	34	0.0038	0.0065	0.0107
470654002	5	35.05093	-85.12631	TN	Hamilton	Chattanooga, TN-GA	476,531	1	3	12	34	0.0038	0.0050	0.0071
470931020	5	36.01944	-83.87361	TN	Knox	Knoxville, TN	616,079	1	3	11	29	0.0040	0.0052	0.0059
470990002	5	35.11611	-87.47000	TN	Lawrence	Lawrenceburg, TN	39,926		3	12	35	0.0021	0.0030	0.0040

Table 2B-8. Pb-PM2.5 monitoring site information

site	poc	lat	long	state	county_name	cbsa_name	cbsa_pop00	urban	data completeness (complete periods)			3-year metrics		
									qtrs	years	months	annual mean	max quarterly mean	max monthly mean
471570047	5	35.16895	-90.02157	TN	Shelby	Memphis, TN-MS-AR	1,205,204	1	3	12	36	0.0033	0.0045	0.0076
471631007	5	36.54065	-82.52167	TN	Sullivan	Kingsport-Bristol-Bristol, TN-VA	298,484	1	3	12	33	0.0031	0.0049	0.0086
471650007	5	36.29778	-86.65278	TN	Sumner	Nashville-Davidson--Murfreesboro	1,311,789	1	3	12	32	0.0027	0.0051	0.0068
480430002	5	30.36580	-103.64910	TX	Brewster				3	11	20	0.0014	0.0025	0.0043
480430101	5	29.30250	-103.16782	TX	Brewster				3	12	25	0.0009	0.0018	0.0028
481130050	5	32.77417	-96.79778	TX	Dallas	Dallas-Fort Worth-Arlington, TX	5,161,544	1	3	12	34	0.0027	0.0041	0.0055
481130069	5	32.81995	-96.86008	TX	Dallas	Dallas-Fort Worth-Arlington, TX	5,161,544	1	3	12	36	0.0036	0.0077	0.0169
481390015	5	32.43694	-97.02500	TX	Ellis	Dallas-Fort Worth-Arlington, TX	5,161,544		3	11	31	0.0029	0.0057	0.0085
481410044	5	31.76567	-106.45523	TX	El Paso	El Paso, TX	679,622	1	3	12	34	0.0036	0.0060	0.0090
481410053	5	31.75852	-106.50105	TX	El Paso	El Paso, TX	679,622	1	3	12	34	0.0078	0.0148	0.0236
481670014	5	29.26332	-94.85657	TX	Galveston	Houston-Sugar Land-Baytown, TX	4,715,407		3	11	32	0.0021	0.0028	0.0041
482010024	5	29.90111	-95.32694	TX	Harris	Houston-Sugar Land-Baytown, TX	4,715,407	1	3	12	35	0.0041	0.0066	0.0087
482010026	5	29.80250	-95.12555	TX	Harris	Houston-Sugar Land-Baytown, TX	4,715,407	1	3	11	31	0.0028	0.0038	0.0056
482010055	5	29.69574	-95.49924	TX	Harris	Houston-Sugar Land-Baytown, TX	4,715,407	1	3	11	32	0.0020	0.0026	0.0037
482011034	5	29.76799	-95.22058	TX	Harris	Houston-Sugar Land-Baytown, TX	4,715,407	1	3	11	31	0.0026	0.0073	0.0160
482011039	7	29.67005	-95.12849	TX	Harris	Houston-Sugar Land-Baytown, TX	4,715,407	1	2	10	30	0.0023	0.0042	0.0072
482030002	5	32.66900	-94.16745	TX	Harrison	Marshall, TX	62,110		3	11	32	0.0019	0.0027	0.0035
482430004	5	30.66938	-104.02463	TX	Jeff Davis				3	11	25	0.0008	0.0014	0.0028
482450022	5	29.86395	-94.31776	TX	Jefferson	Beaumont-Port Arthur, TX	385,090		3	11	32	0.0019	0.0030	0.0049
482570005	5	32.56917	-96.31583	TX	Kaufman	Dallas-Fort Worth-Arlington, TX	5,161,544	1	3	11	30	0.0024	0.0063	0.0128
482730314	5	27.42694	-97.29861	TX	Kleberg	Kingsville, TX	31,963		3	11	29	0.0010	0.0017	0.0024
483030001	5	33.59085	-101.84759	TX	Lubbock	Lubbock, TX	249,700	1	2	10	28	0.0010	0.0024	0.0062
483390078	5	30.35030	-95.42514	TX	Montgomery	Houston-Sugar Land-Baytown, TX	4,715,407		3	11	32	0.0031	0.0042	0.0058
483550034	5	27.81180	-97.46563	TX	Nueces	Corpus Christi, TX	403,280	1	3	12	36	0.0013	0.0021	0.0033
483611100	5	30.19417	-93.86694	TX	Orange	Beaumont-Port Arthur, TX	385,090		3	11	32	0.0020	0.0028	0.0041
490110004	5	40.90297	-111.88447	UT	Davis	Ogden-Clearfield, UT	442,656	1	2	10	29	0.0035	0.0059	0.0071
490353006	5	40.73639	-111.87222	UT	Salt Lake	Salt Lake City, UT	968,858	1	3	12	36	0.0042	0.0077	0.0131
490494001	5	40.34139	-111.71361	UT	Utah	Provo-Orem, UT	376,774	1	3	12	36	0.0034	0.0072	0.0095
500070012	5	44.48028	-73.21444	VT	Chittenden	Burlington-South Burlington, VT	198,889	1	3	12	35	0.0023	0.0029	0.0037
510870014	5	37.55833	-77.40028	VA	Henrico	Richmond, VA	1,096,957	1	2	8	24	0.0030	0.0042	0.0064
511390004	5	38.66333	-78.50472	VA	Page				2	8	24	0.0027	0.0045	0.0081
515200006	5	36.60778	-82.16444	VA	Bristol (City)	Kingsport-Bristol-Bristol, TN-VA	298,484	1	3	12	35	0.0036	0.0057	0.0083
517600020	5	37.51056	-77.49833	VA	Richmond (City)	Richmond, VA	1,096,957	1	1	4	12	0.0027	0.0033	0.0064
517700014	5	37.25611	-79.98500	VA	Roanoke (City)	Roanoke, VA	288,309	1	2	8	24	0.0074	0.0140	0.0283
530330024	6	47.75333	-122.27722	WA	King	Seattle-Tacoma-Bellevue, WA	3,043,878	1	3	12	35	0.0030	0.0046	0.0073
530330032	6	47.54556	-122.32222	WA	King	Seattle-Tacoma-Bellevue, WA	3,043,878	1	2	7	22	0.0078	0.0134	0.0201
530330048	6	47.61846	-122.32972	WA	King	Seattle-Tacoma-Bellevue, WA	3,043,878	1	3	12	33	0.0032	0.0052	0.0089
530330057	6	47.56333	-122.33833	WA	King	Seattle-Tacoma-Bellevue, WA	3,043,878	1	3	12	33	0.0074	0.0150	0.0260
530330080	6	47.57027	-122.30860	WA	King	Seattle-Tacoma-Bellevue, WA	3,043,878	1	3	12	36	0.0034	0.0055	0.0075
530630016	5	47.66083	-117.35722	WA	Spokane	Spokane, WA	417,939	1	1	4	11	0.0038	0.0062	0.0087
540390011	5	38.44861	-81.68389	WV	Kanawha	Charleston, WV	309,635		2	9	25	0.0026	0.0043	0.0048
540391005	5	38.36806	-81.69361	WV	Kanawha	Charleston, WV	309,635	1	2	8	25	0.0043	0.0067	0.0077
540511002	5	39.91597	-80.73406	WV	Marshall	Wheeling, WV-OH	153,172	1	1	6	19	0.0065	0.0081	0.0124
550270007	5	43.43500	-88.52778	WI	Dodge	Beaver Dam, WI	85,897		3	12	36	0.0036	0.0059	0.0083
550590019	5	42.50472	-87.80930	WI	Kenosha	Chicago-Naperville-Joliet, IL-IN-W	9,098,316		3	12	36	0.0038	0.0057	0.0073
550710007	5	44.13861	-87.61611	WI	Manitowoc	Manitowoc, WI	82,887		2	8	25	0.0039	0.0060	0.0113
550790026	5	43.06111	-87.91250	WI	Milwaukee	Milwaukee-Waukesha-West Allis,	1,500,741	1	3	12	36	0.0058	0.0115	0.0245
551198001	5	45.20389	-90.60000	WI	Taylor				3	12	36	0.0020	0.0030	0.0050
551330027	5	43.02028	-88.21500	WI	Waukesha	Milwaukee-Waukesha-West Allis,	1,500,741	1	3	12	36	0.0097	0.0185	0.0217
720610001	5	18.42472	-66.11639	PR	Guaynabo	San Juan-Caguas-Guaynabo, PR	2,509,007	1	3	12	36	0.0018	0.0026	0.0058
780100012	5	17.71444	-64.78528	VI	St Croix			1	1	5	12	0.0003	0.0007	0.0009

Table 2B-9. Pb_PM2.5 monitoring site distribution statistics

All sites

	n	min	pct5	pct10	pct15	pct20	pct25	pct30	pct35	pct40	pct45	median	mean	pct55	pct60	pct65	pct70	pct75	pct80	pct85	pct90	pct95	max
annual mean	271	0.0003	0.0014	0.0019	0.0020	0.0022	0.0024	0.0026	0.0027	0.0029	0.0031	0.0033	0.0043	0.0035	0.0038	0.0040	0.0042	0.0046	0.0051	0.0060	0.0074	0.0112	0.0450
max quarter mean	271	0.0007	0.0022	0.0028	0.0030	0.0034	0.0037	0.0039	0.0042	0.0044	0.0047	0.0052	0.0076	0.0056	0.0059	0.0063	0.0067	0.0072	0.0083	0.0101	0.0140	0.0175	0.1674
max monthly mean	271	0.0009	0.0033	0.0040	0.0044	0.0050	0.0053	0.0057	0.0061	0.0064	0.0068	0.0073	0.0123	0.0079	0.0085	0.0089	0.0098	0.0112	0.0133	0.0180	0.0228	0.0302	0.3091

Source-oriented sites

	n	min	pct5	pct10	pct15	pct20	pct25	pct30	pct35	pct40	pct45	median	mean	pct55	pct60	pct65	pct70	pct75	pct80	pct85	pct90	pct95	max
annual mean	8	0.0036	0.0036	0.0036	0.0043	0.0043	0.0053	0.0063	0.0063	0.0067	0.0067	0.0073	0.0086	0.0079	0.0079	0.0106	0.0106	0.0110	0.0114	0.0114	0.0180	0.0180	0.0180
max quarter mean	8	0.0067	0.0067	0.0067	0.0101	0.0101	0.0107	0.0114	0.0114	0.0122	0.0122	0.0132	0.0143	0.0142	0.0142	0.0148	0.0148	0.0153	0.0157	0.0157	0.0296	0.0296	0.0296
max monthly mean	8	0.0077	0.0077	0.0077	0.0086	0.0086	0.0136	0.0186	0.0186	0.0190	0.0190	0.0191	0.0215	0.0192	0.0192	0.0228	0.0228	0.0257	0.0286	0.0286	0.0475	0.0475	0.0475

Not source-oriented sites

	n	min	pct5	pct10	pct15	pct20	pct25	pct30	pct35	pct40	pct45	median	mean	pct55	pct60	pct65	pct70	pct75	pct80	pct85	pct90	pct95	max
annual mean	263	0.0003	0.0014	0.0019	0.0020	0.0022	0.0024	0.0026	0.0027	0.0029	0.0030	0.0032	0.0042	0.0034	0.0037	0.0038	0.0042	0.0045	0.0048	0.0057	0.0073	0.0097	0.0450
max quarter mean	263	0.0007	0.0022	0.0027	0.0030	0.0034	0.0037	0.0039	0.0042	0.0043	0.0046	0.0050	0.0073	0.0055	0.0057	0.0061	0.0066	0.0070	0.0080	0.0092	0.0128	0.0172	0.1674
max monthly mean	263	0.0009	0.0033	0.0040	0.0043	0.0049	0.0053	0.0056	0.0060	0.0063	0.0067	0.0072	0.0120	0.0076	0.0084	0.0087	0.0096	0.0104	0.0124	0.0163	0.0204	0.0283	0.3091

Urban sites

	n	min	pct5	pct10	pct15	pct20	pct25	pct30	pct35	pct40	pct45	median	mean	pct55	pct60	pct65	pct70	pct75	pct80	pct85	pct90	pct95	max
annual mean	216	0.0003	0.0017	0.0020	0.0023	0.0024	0.0026	0.0027	0.0030	0.0031	0.0033	0.0035	0.0046	0.0037	0.0039	0.0042	0.0045	0.0049	0.0056	0.0065	0.0079	0.0118	0.0450
max quarter mean	216	0.0007	0.0025	0.0031	0.0035	0.0038	0.0040	0.0042	0.0044	0.0047	0.0051	0.0055	0.0082	0.0059	0.0063	0.0066	0.0071	0.0079	0.0092	0.0115	0.0148	0.0182	0.1674
max monthly mean	216	0.0009	0.0036	0.0045	0.0051	0.0055	0.0058	0.0061	0.0063	0.0067	0.0072	0.0077	0.0135	0.0085	0.0088	0.0097	0.0104	0.0123	0.0160	0.0191	0.0244	0.0329	0.3091

Urban sites, located in MSA's > 1 million population

	n	min	pct5	pct10	pct15	pct20	pct25	pct30	pct35	pct40	pct45	median	mean	pct55	pct60	pct65	pct70	pct75	pct80	pct85	pct90	pct95	max
annual mean	99	0.0018	0.0022	0.0025	0.0027	0.0027	0.0030	0.0031	0.0033	0.0036	0.0038	0.0041	0.0055	0.0042	0.0047	0.0048	0.0054	0.0065	0.0075	0.0090	0.0097	0.0123	0.0450
max quarter mean	99	0.0026	0.0033	0.0037	0.0039	0.0042	0.0045	0.0051	0.0054	0.0058	0.0061	0.0063	0.0093	0.0066	0.0069	0.0071	0.0083	0.0106	0.0126	0.0147	0.0182	0.0239	0.0967
max monthly mean	99	0.0036	0.0047	0.0052	0.0058	0.0064	0.0068	0.0072	0.0075	0.0078	0.0086	0.0088	0.0160	0.0099	0.0104	0.0128	0.0160	0.0185	0.0201	0.0245	0.0282	0.0413	0.2091

Urban sites, located in MSA's < 1 million population

	n	min	pct5	pct10	pct15	pct20	pct25	pct30	pct35	pct40	pct45	median	mean	pct55	pct60	pct65	pct70	pct75	pct80	pct85	pct90	pct95	max
annual mean	117	0.0003	0.0013	0.0017	0.0020	0.0021	0.0023	0.0025	0.0026	0.0028	0.0030	0.0032	0.0038	0.0033	0.0035	0.0037	0.0038	0.0042	0.0046	0.0051	0.0063	0.0078	0.0257
max quarter mean	117	0.0007	0.0021	0.0026	0.0029	0.0034	0.0037	0.0038	0.0040	0.0042	0.0044	0.0046	0.0072	0.0050	0.0053	0.0057	0.0060	0.0068	0.0079	0.0087	0.0112	0.0150	0.1674
max monthly mean	117	0.0009	0.0031	0.0037	0.0042	0.0051	0.0053	0.0056	0.0059	0.0060	0.0062	0.0064	0.0114	0.0070	0.0079	0.0086	0.0090	0.0097	0.0112	0.0131	0.0186	0.0283	0.3091

APPENDIX 5A:

PREDICTED PERCENT OF COUNTIES WITH A MONITOR NOT LIKELY TO MEET ALTERNATIVE STANDARDS AND ASSOCIATED PERCENT POPULATION

Appendix 5.A. Predicted percent of counties with a monitor not likely to meet alternative standards & associated percent population.

The values below were derived from the Pb-TSP dataset described in Chapter 2 and in Appendix 2.B.

	Total	Northeast	Southeast	Industrial Midwest	Upper Midwest	Southwest	Northwest	Southern California	Outlying areas
Number of counties with monitors (population in those counties) ►	125 (58,035)	14 (7,285)	43 (13,298)	43 (17,166)	7 (2,184)	3 (1,015)	7 (3,002)	6 (12,774)	2 (1,311)
level / form ▼	**Percent of counties with a monitor not likely to meet stated standard and level** (percent of total or regional population in counties with monitors that reside in non-meeting counties)								
0.02, max quarterly	53% (55%)	64% (83%)	35% (29%)	72% (41%)	29% (25%)	33% (67%)	57% (78%)	67% (88%)	0% (0%)
0.02, max monthly	66% (75%)	64% (83%)	51% (58%)	86% (83%)	43% (49%)	67% (81%)	86% (78%)	67% (88%)	0% (0%)
0.02, 2nd max monthly	55% (62%)	64% (83%)	40% (32%)	74% (74%)	14% (16%)	67% (81%)	71% (78%)	50% (75%)	0% (0%)
0.05, max quarterly	39% (36%)	43% (21%)	35% (29%)	44% (15%)	14% (16%)	33% (67%)	57% (78%)	50% (75%)	0% (0%)
0.05, max monthly	45% (42%)	43% (21%)	35% (29%)	56% (25%)	29% (25%)	33% (67%)	57% (78%)	67% (88%)	0% (0%)
0.05, 2nd max monthly	38% (18%)	43% (21%)	35% (29%)	44% (15%)	14% (16%)	0% (0%)	57% (78%)	33% (0%)	0% (0%)
0.10, max quarterly	23% (6%)	29% (11%)	21% (6%)	30% (5%)	14% (16%)	0% (0%)	29% (31%)	0% (0%)	0% (0%)
0.10, max monthly	34% (32%)	43% (21%)	26% (23%)	40% (11%)	14% (16%)	0% (0%)	57% (78%)	50% (75%)	0% (0%)
0.10, 2nd max monthly	27% (10%)	43% (21%)	23% (6%)	30% (5%)	14% (16%)	0% (0%)	57% (78%)	0% (0%)	0% (0%)
0.20, max quarterly	17% (3%)	21% (1%)	16% (5%)	23% (4%)	0% (0%)	0% (0%)	14% (12%)	0% (0%)	0% (0%)
0.20, max monthly	23% (9%)	21% (1%)	23% (23%)	30% (5%)	14% (16%)	0% (0%)	29% (31%)	0% (0%)	0% (0%)
0.20, 2nd max monthly	20% (5%)	21% (1%)	19% (6%)	26% (5%)	14% (16%)	0% (0%)	29% (31%)	0% (0%)	0% (0%)
0.3, max quarterly	14% (3%)	7% (0%)	16% (5%)	19% (3%)	0% (0%)	0% (0%)	14% (12%)	0% (0%)	0% (0%)
0.3, max monthly	18% (3%)	21% (1%)	16% (5%)	26% (4%)	0% (0%)	0% (0%)	14% (12%)	0% (0%)	0% (0%)
0.3, 2nd max monthly	14% (2%)	7% (0%)	16% (5%)	21% (2%)	0% (0%)	0% (0%)	14% (12%)	0% (0%)	0% (0%)
0.4, max quarterly	10% (1%)	0% (0%)	14% (1%)	14% (2%)	0% (0%)	0% (0%)	14% (12%)	0% (0%)	0% (0%)
0.4, max monthly	14% (3%)	7% (0%)	16% (5%)	21% (4%)	0% (0%)	0% (0%)	14% (12%)	0% (0%)	0% (0%)
0.4, 2nd max monthly	14% (2%)	7% (0%)	16% (5%)	19% (2%)	0% (0%)	0% (0%)	14% (12%)	0% (0%)	0% (0%)
0.50, max quarterly	10% (1%)	0% (0%)	14% (1%)	12% (1%)	0% (0%)	0% (0%)	14% (12%)	0% (0%)	0% (0%)
0.50, max monthly	14% (3%)	7% (0%)	16% (5%)	21% (4%)	0% (0%)	0% (0%)	14% (12%)	0% (0%)	0% (0%)
0.50, 2nd max monthly	11% (1%)	7% (0%)	14% (1%)	16% (2%)	0% (0%)	0% (0%)	0% (0%)	0% (0%)	0% (0%)

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