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40 CFR Part 50
National Ambient Air Quality Standards for Lead; Proposed Rule

ENVIRONMENTAL PROTECTION AGENCY

40 CFR Part 50

[EPA–HQ–OAR–2006–0735; FRL–8503–8]

RIN 2060–AN83

National Ambient Air Quality Standards for Lead

AGENCY: Environmental Protection Agency (EPA).

ACTION: Advance notice of proposed rulemaking (ANPR).

SUMMARY: EPA is issuing this ANPR to invite comment from all interested parties on policy options and other issues related to the Agency's ongoing review of the national ambient air quality standards (NAAQS) for lead (Pb). Consistent with recent modifications the Agency has made to its process for reviewing NAAQS, we are seeking broad public comment at this time to help inform the Agency's future proposed decisions on the adequacy of the current Pb NAAQS and on any revisions of the Pb NAAQS that may be appropriate. EPA is also soliciting comment on retaining Pb on the list of criteria pollutants and on maintaining NAAQS for Pb.

As part of this review, the Agency has released several key documents that will inform the Agency's rulemaking. These documents include the *Air Quality Criteria for Lead,* released in 2006, which critically assesses and integrates relevant scientific information; risk assessment reports including the most recent report, *Lead: Human Exposure and Health Risk Assessment for Selected Case Studies,* which documents quantitative exposure analyses and risk assessments conducted for this review; and a recently released Staff Paper, *Review of the National Ambient Air Quality Standards for Lead: Policy Assessment of Scientific and Technical Information,* which presents an evaluation by staff in EPA's Office of Air Quality Planning and Standards (OAQPS) of the policy implications of the scientific information and quantitative assessments and OAQPS staff conclusions and recommendations on a range of policy options for the Agency's consideration.

Under the terms of a court order, the Administrator will sign by September 1, 2008 a Notice of Final Rulemaking for publication in the **Federal Register**. To meet this schedule, we anticipate the Administrator will sign a Notice of Proposed Rulemaking in March 2008 for publication in the **Federal Register**, at which time further opportunity for public comment will be provided.

DATES: Comments must be received by January 16, 2008.

ADDRESSES: Submit your comments, identified by Docket ID No. EPA–HQ–OAR–2006–0735 by one of the following methods:

- *http://www.regulations.gov:* Follow the on-line instructions for submitting comments.
- *E-mail: a-and-r-Docket@epa.gov.*
- *Fax:* 202–566–9744.
- *Mail:* Docket No. EPA–HQ–OAR–2006–0735, Environmental Protection Agency, Mail code 6102T, 1200 Pennsylvania Ave., NW., Washington, DC 20460. Please include a total of two copies.
- *Hand Delivery:* Docket No. EPA–HQ–OAR–2006–0735, Environmental Protection Agency, EPA West, Room 3334, 1301 Constitution Ave., NW., Washington, DC. Such deliveries are only accepted during the Docket's normal hours of operation, and special arrangements should be made for deliveries of boxed information.

Instructions: Direct your comments to Docket ID No. EPA–HQ–OAR–2006–0735. The EPA's policy is that all comments received will be included in the public docket without change and may be made available online at *http://www.regulations.gov,* including any personal information provided, unless the comment includes information claimed to be Confidential Business Information (CBI) or other information whose disclosure is restricted by statute. Do not submit information that you consider to be CBI or otherwise protected through *http://www.regulations.gov* or e-mail. The *http://www.regulations.gov* Web site is an ''anonymous access'' system, which means EPA will not know your identity or contact information unless you provide it in the body of your comment. If you send an e-mail comment directly to EPA without going through *http://www.regulations.gov,* your e-mail address will be automatically captured and included as part of the comment that is placed in the public docket and made available on the Internet. If you submit an electronic comment, EPA recommends that you include your name and other contact information in the body of your comment and with any disk or CD–ROM you submit. If EPA cannot read your comment due to technical difficulties and cannot contact you for clarification, EPA may not be able to consider your comment. Electronic files should avoid the use of special characters, any form of encryption, and be free of any defects or viruses. For additional information about EPA's public docket, visit the EPA Docket Center homepage at *http://www.epa.gov/epahome/dockets.htm.*

Docket: All documents in the docket are listed in the *http://www.regulations.gov* index. Although listed in the index, some information is not publicly available, e.g., CBI or other information whose disclosure is restricted by statute. Certain other material, such as copyrighted material, will be publicly available only in hard copy. Publicly available docket materials are available either electronically in *http://www.regulations.gov* or in hard copy at the Air and Radiation Docket and Information Center, EPA/DC, EPA West, Room 3334, 1301 Constitution Ave., NW., Washington, DC. The Public Reading Room is open from 8:30 a.m. to 4:30 p.m., Monday through Friday, excluding legal holidays. The telephone number for the Public Reading Room is (202) 566–1744 and the telephone number for the Air and Radiation Docket and Information Center is (202) 566–1742.

FOR FURTHER INFORMATION CONTACT: Dr. Deirdre Murphy, Health and Environmental Impacts Division, Office of Air Quality Planning and Standards, U.S. Environmental Protection Agency, Mail code C504–06, Research Triangle Park, NC 27711; *telephone:* 919–541–0729; *fax:* 919–541–0237; e-mail: *Murphy.deirdre@epa.gov.*

SUPPLEMENTARY INFORMATION:

General Information

What Should I Consider as I Prepare My Comments for EPA?

1. *Submitting CBI.* Do not submit this information to EPA through *http://www.regulations.gov* or e-mail. Clearly mark the part or all of the information that you claim to be CBI. For CBI information in a disk or CD ROM that you mail to EPA, mark the outside of the disk or CD ROM as CBI and then identify electronically within the disk or CD ROM the specific information that is claimed as CBI. In addition to one complete version of the comment that includes information claimed as CBI, a copy of the comment that does not contain the information claimed as CBI must be submitted for inclusion in the public docket. Information so marked will not be disclosed except in accordance with procedures set forth in 40 CFR part 2.

2. *Tips for Preparing Your Comments.* When submitting comments, remember to:

- Identify the rulemaking by docket number and other identifying

information (subject heading, **Federal Register** date and page number).

- Follow directions—the agency may ask you to respond to specific questions or organize comments by referencing a Code of Federal Regulations (CFR) part or section number.
- Explain why you agree or disagree, suggest alternatives, and substitute language for your requested changes.
- Describe any assumptions and provide any technical information and/or data that you used.
- If you estimate potential costs or burdens, explain how you arrived at your estimate in sufficient detail to allow for it to be reproduced.
- Provide specific examples to illustrate your concerns, and suggest alternatives.
- Explain your views as clearly as possible, avoiding the use of profanity or personal threats.
- Make sure to submit your comments by the comment period deadline identified.

Availability of Related Information

A number of documents relevant to this rulemaking, including the *Air Quality Criteria for Lead* (Criteria Document) (USEPA, 2006a), the Staff Paper, related risk assessment reports, and other related technical documents are available on EPA's Office of Air Quality Planning and Standards (OAQPS) Technology Transfer Network (TTN) Web site at *http://www.epa.gov/ttn/naaqs/standards/pb/s_pb_index.html*. These and other related documents are also available for inspection and copying in the EPA docket identified above.

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I. Introduction

In the past year EPA has instituted a number of changes to the process that the Agency uses in reviewing the NAAQS to help to improve the efficiency of the process while ensuring that the Agency's decisions are informed by the best available science and broad participation among experts in the scientific community and the public (described at *http://www.epa.gov/ttn/naaqs/*). These changes apply to the four major components of the NAAQS review process: planning, science assessment, risk/exposure assessment, and policy assessment/rulemaking. The process improvements will help the Agency meet the goal of reviewing each NAAQS on a 5-year cycle as required by the Clean Air Act (CAA) without compromising the scientific integrity of the process. These changes are being incorporated into the various ongoing NAAQS reviews being conducted by the Agency, including the current review of the Pb NAAQS.

The issuance of this ANPR is one of the key features of the new NAAQS review process. Historically, a policy assessment that evaluates the policy implications of the available scientific information and risk/exposure assessments has been presented in the form of a Staff Paper, prepared by staff in EPA's OAQPS, which included OAQPS staff conclusions and recommendations on a range of policy options for the Agency's consideration. The new process will enable broader participation of the scientific community and the public early in the NAAQS review by providing scientific information, risk/exposure assessments, and policy options in an ANPR rather than a Staff Paper. The purpose of the ANPR is to identify conceptual evidence- and risk-based approaches for reaching policy judgments, discuss what the science and risk/exposure assessments say about the adequacy of the current standards, and describe a range of options for standard setting, in terms of indicators, averaging times, forms, and ranges of levels for any alternative standards. Discussion of alternative standards is to include a description of the underlying interpretations of the scientific evidence and risk/exposure information that might support such alternative standards and that could be considered by the Administrator in making NAAQS decisions. The issuance of an ANPR provides the opportunity for the Clean Air Scientific Advisory Committee (CASAC) [1] and the public to evaluate and provide comment on a broad range of policy options being considered by the Administrator.

In the case of this Pb NAAQS review, which was initiated well before changes were instituted to the NAAQS review process, both an OAQPS Staff Paper and an ANPR are being issued. As discussed below in section II, the issuance of both documents reflects the terms of a court order that governs this review and requires that a final OAQPS Staff Paper be issued. As a consequence, in addition to soliciting comment, this ANPR summarizes information from the OAQPS Staff Paper (referred to as Staff Paper throughout this notice) and from

[1] As discussed below in section II, CASAC is the independent scientific review committee that provides advice and recommendations to the EPA Administrator related to periodic reviews of NAAQS, as mandated by the Clean Air Act.

the Agency's risk assessment and Criteria Document. This ANPR is structured such that policy options on adequacy of the current standards and aspects of potential alternative standards are discussed in Sections III.C and IV.C. Preceding those policy discussions are sections focused on health and welfare effects in Sections III.A and IV.A, respectively, and on human exposure and risk and ecological risk in Sections III.B and IV.B, respectively.

II. Background

A. 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 be 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.'' [2] 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.'' [3]

The requirement that primary standards include an adequate margin of safety was intended to address uncertainties associated with inconclusive scientific and technical information available at the time of standard setting. It was also intended to provide a reasonable degree of protection against hazards that research has not yet identified. *Lead Industries Association* v. *EPA*, 647 F.2d 1130, 1154 (DC 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 promulgated under this section and shall make such revisions in such criteria and standards and promulgate such new standards as may be appropriate in accordance with section 108 and subsection (b) of this section. The Administrator may review and revise criteria or promulgate new standards earlier or more frequently than required under this paragraph.'' Section 109(d)(2)(A) requires that ''The Administrator shall appoint an independent scientific review committee composed of seven members including at least one member of the National Academy of Sciences, one physician, and one person representing State air pollution control agencies.'' Section 109(d)(2)(B) requires that, ''Not later than January 1, 1980, and at five-year intervals thereafter, the committee referred to in subparagraph (A) shall complete a review of the criteria published under section 108 and the national primary and secondary ambient air quality standards promulgated under this section and shall recommend to the Administrator any new national ambient air quality standards and revisions of existing criteria and standards as may be appropriate under section 108 and subsection (b) of this section.'' [4] 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.

B. History of Lead NAAQS Reviews

On October 5, 1978 EPA promulgated primary and secondary NAAQS for Pb 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 (Pb-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).

[2] 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)

[3] 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.''

[4] In addition to the provisions of Section 109(d)(2)(B), concerning the role of CASAC in providing advice and recommendations to the Administrator on the criteria and standards, Section 109(d)(2)(C) provides that CASAC shall also, ''(i) advise the Administrator of areas in which additional knowledge is required to appraise the adequacy and basis of existing, new, or revised national ambient air quality standards, (ii) describe the research efforts necessary to provide the required information, (iii) advise the Administrator on the relative contribution to air pollution concentrations of natural as well as anthropogenic activity, and (iv) advise the Administrator of any adverse public health, welfare, social economic, or energy effects which may result from various strategies for attainment and maintenance of such national ambient air quality standards.''

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.

C. Current Related Lead Control Programs

States are primarily responsible for ensuring attainment and maintenance of ambient air quality standards once EPA has established them. Under section 110 of the Act (42 U.S.C. 7410) and related provisions, States are to submit, for EPA approval, State implementation plans (SIP's) that provide for the attainment and maintenance of such standards through control programs directed to sources of the pollutants involved. The States, in conjunction with EPA, also administer the prevention of significant deterioration program (42 U.S.C. 7470–7479) for these pollutants. In addition, Federal programs provide for nationwide reductions in emissions of these and other air pollutants through the Federal Motor Vehicle Control Program under Title II of the Act (42 U.S.C. 7521–7574), which involves controls for automobile, truck, bus, motorcycle, nonroad engine, and aircraft emissions; the new source performance standards under section 111 of the Act (42 U.S.C. 7411); and the national emission standards for hazardous air pollutants under section 112 of the Act (42 U.S.C. 7412).

As Pb is a multimedia pollutant, a broad range of Federal programs beyond those identified above that focus on air pollution control provide for nationwide reductions in environmental releases and human exposures. The Centers for Disease Control and Prevention (CDC) programs provide for the tracking of children's blood Pb levels nationally and provide guidance on levels at which medical and environmental case management activities should be implemented (CDC, 2005a; ACCLPP, 2007).[5] In 1991, the Secretary of the Health and Human Services (HHS) characterized Pb poisoning as the ''number one environmental threat to the health of children in the United States'' (Alliance to End Childhood Lead Poisoning. 1991). And, in 1997, President Clinton created, by Executive Order 13045, the President's Task Force on Environmental Health Risks and Safety Risks to Children in response to increased awareness that children face disproportionate risks from environmental health and safety hazards (62 FR 19885).[6] By Executive Orders issued in October 2001 and April 2003, President Bush extended the work for the Task Force for an additional three and a half years beyond its original charter (66 FR 52013 and 68 FR 19931). The Task Force set a Federal goal of eliminating childhood Pb poisoning by the year 2010 and reducing Pb poisoning in children was the Task Force's top priority.

Federal abatement programs provide for the reduction in human exposures and environmental releases from in-place materials containing Pb (e.g., Pb-based paint, urban soil and dust and contaminated waste sites). Federal regulations on disposal of Pb-based paint waste help facilitate the removal of Pb-based paint from residences (See ''Criteria for Classification of Solid Waste Disposal Facilities and Practices and Criteria for Municipal Solid Waste Landfills: Disposal of Residential Lead-Based Paint Waste; Final Rule'' EPA–HQ–RCRA–2001–0017). Further, in 1991, EPA lowered the maximum levels of Pb permitted in public water systems from 50 parts per billion (ppb) to 15 ppb (56 FR 26460).

Federal programs to reduce exposure to Pb in paint, dust and soil are specified under the comprehensive federal strategy developed under the Residential Lead-Based Paint Hazard Reduction Act (Title X). Under Title X and Title IV of the Toxic Substances Control Act, EPA has established regulations in the following four categories: (1) Training and certification requirements for persons engaged in lead-based paint activities; accreditation of training providers; work practice standards for the safe, reliable, and effective identification and elimination of lead-based paint hazards; (2) Ensuring that, for most housing constructed before 1978, lead-based paint information flows from sellers to purchasers, from landlords to tenants, and from renovators to owners and occupants; (3) Establishing standards for identifying dangerous levels of lead in paint, dust and soil; and (4) Providing information on lead hazards to the public, including steps that people can take to protect themselves and their families from lead-based paint hazards.

Under Title X of TSCA, EPA established dust lead standards for residential housing and soil dust in 2001. This regulation supports the implementation of other regulations which deal with worker training and certification, lead hazard disclosure in real estate transactions, lead hazard evaluation and control in federally-owned housing prior to sale and housing receiving Federal assistance, and U.S. Department of Housing and Urban Development grants to local jurisdictions to perform lead hazard control. In addition, this regulation also establishes, among other things, under authority of TSCA section 402, residential lead dust cleanup levels and amendments to dust and soil sampling requirements (66 FR 1206). The Title X term ''lead-based paint hazard'' implemented through this regulation identifies lead-based paint and all residential lead-containing dusts and soils regardless of the source of lead, which, due to their condition and location, would result in adverse human health effects. One of the underlying principles of Title X is to move the focus of public and private decision makers away from the mere presence of lead-based paint, to the presence of lead-based paint hazards, for which more substantive action should be undertaken to control exposures, especially to young children. In addition the success of the program will rely on the voluntary participation of states and tribes as well as counties and cities to implement the programs and on property owners to follow the standards and EPA's recommendations. If EPA

[5] As described in Section III below the CDC stated in 2005 that no ''safe'' threshold for blood Pb levels in young children has been identified (CDC, 2005a).

[6] Co-chaired by the Secretary of the HHS and the Administrator of the EPA, the Task Force consisted of representatives from 16 Federal departments and agencies.

were to set unreasonable standards (e.g., standards that would recommend removal of all lead from paint, dust and soil), States and Tribes may choose to opt out of the Title X lead program and property owners may choose to ignore EPA's advice believing it lacks credibility and practical value. Consequently, EPA needed to develop standards that would not waste resources by chasing risks of negligible importance and that would be accepted by States, Tribes, local governments and property owners.

On January 10, 2006, EPA issued a Notice of Proposed Rulemaking covering renovations performed for compensation in target housing. The 2006 Proposal contains requirements designed to address lead hazards created by renovation, repair, and painting activities that disturb lead-based paint. The 2006 Proposal includes requirements for training renovators, other renovation workers, and dust sampling technicians; for certifying renovators, dust sampling technicians, and renovation firms; for accrediting providers of renovation and dust sampling technician training; for renovation work practices; and for recordkeeping. The 2006 Proposal proposes to make the rule effective in two stages. Initially, the rule proposes to apply to all renovations for compensation performed in target housing where a child with an increased blood lead level resided and rental target housing built before 1960. The proposed rule also proposes application to owner-occupied target housing built before 1960, unless the person performing the renovation obtained a statement signed by the owner-occupant that the renovation would occur in the owner's residence and that no child under age 6 resided there. As proposed, the rule would take effect one year later in all rental target housing built between 1960 and 1978 and owner-occupied target housing built between 1960 and 1978. EPA also proposes to allow interested States, Territories, and Indian Tribes the opportunity to apply for and receive authorization to administer and enforce all of the elements of the new renovation provisions.

A significant number of commenters observed that the proposal did not cover buildings where children under age 6 spend a great deal of time, such as day care centers and schools. Commenters noted that the risk posed to children from lead-based paint hazards in schools and day-care centers is likely to be equal to, if not greater than, the risk posed from these hazards at home. These commenters suggested that EPA expand its proposal to include such places, and several suggested that EPA use the existing definition of ''child-occupied facility'' in 40 CFR § 745.223 to define the expanded scope of coverage. EPA felt that these comments had merit, and, because adding child-occupied facilities was beyond the scope of the 2006 Proposal, an expansion of the 2006 Proposal was necessary to give this issue full and fair consideration. Accordingly, on June 5, 2007, EPA issued a Supplemental Notice of Proposed Rulemaking to add child-occupied facilities to the universe of buildings covered by the 2006 Proposal. EPA is working expeditiously to finalize this rulemaking and expects to do so in the first calendar quarter of 2008.

Programs associated with the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA or Superfund) and Resource Conservation Recovery Act (RCRA) also implement abatement programs, reducing exposures to Pb and other pollutants. For example, EPA determines and implements protective levels for Pb in soil at Superfund sites and RCRA corrective action facilities. Federal programs, including those implementing RCRA, provide for management of hazardous substances in hazardous and municipal solid waste (e.g., ''Hazardous Waste Management System; Identification and Listing of Hazardous Waste: Inorganic Chemical Manufacturing Wastes; Land Disposal Restrictions for Newly Identified Wastes and CERCLA Hazardous Substance Designation and Reportable Quantities; Final Rule'', *http://www.epa.gov/epaoswer/hazwaste/state/revision/frs/fr195.pdf* and *http://www.epa.gov/epaoswer/hazwaste/ldr/basic.htm*). For example, Federal regulations concerning batteries in municipal solid waste facilitate the collection and recycling or proper disposal of batteries containing Pb (e.g., See ''Implementation of the Mercury-Containing and Rechargeable Battery Management Act'' *http://www.epa.gov/epaoswer/hazwaste/recycle/battery.pdf* and ''Municipal Solid Waste Generation, Recycling, and Disposal in the United States: Facts and Figures for 2005'' *http://www.epa.gov/epaoswer/osw/conserve/resources/msw-2005.pdf*). Similarly, Federal programs provide for the reduction in environmental releases of hazardous substances such as Pb in the management of wastewater (*http://www.epa.gov/owm/*).

A variety of federal nonregulatory programs also provide for reduced environmental release of Pb containing materials through more general encouragement of pollution prevention, promote reuse and recycling, reduce priority and toxic chemicals in products and waste, and conserve energy and materials. These include the Resource Conservation Challenge (*http://www.epa.gov/epaoswer/osw/conserve/index.htm*), the National Waste Minimization Program (*http://www.epa.gov/epaoswer/hazwaste/minimize/leadtire.htm*), ''Plug in to eCycling'' (a partnership between EPA and consumer electronics manufacturers and retailers; *http://www.epa.gov/epaoswer/hazwaste/recycle/electron/crt.htm#crts*), and activities to reduce the practice of backyard trash burning (*http://www.epa.gov/msw/backyard/pubs.htm*).

Efforts such as those programs described above have been successful in that blood Pb levels in all segments of the population have dropped significantly from levels around 1990. In particular, blood Pb levels for the general population of children 1 to 5 years of age have dropped to a median level of 1.6 µg/dL and a level of 3.9 µg/dL for the 90th percentile child in the 2003–2004 NHANES as compared to median and 90th percentile levels in 1988–1991 of 3.5 µg/dL and 9.4 µg/dL, respectively (*http://www.epa.gov/envirohealth/children/body_burdens/b1-table.htm*). These levels (median and 90th percentile) for the general population of young children [7] are at the low end of the historic range of blood Pb levels for general population of children aged 1–5 years and are below a level of 5 µg/dL—a level that has been associated with adverse effects with a higher degree of certainty in the published literature (than levels such as 2 µg/dL) and is a level where cognitive deficits were identified with statistical significance (Lanphear et al., 2000). The decline in blood Pb levels in the United States has resulted from coordinated, intensive efforts at the national, state and local levels. The Agency has continued to grapple with soil and dust Pb levels from the historical use of Pb in paint and gasoline and other sources. In doing so, the agency has faced the difficulty of determining the level at which to set standards for residential dust levels given the uncertainties at what environmental levels and in which specific medium may actually cause particular blood Pb levels that are

[7] It is noted that although the 95th percentile value for the 2003–2004 NHANES is not currently available, that value for 2001–2002 was 5.8 µg/dL. Also, as discussed in Section III.A.1 (including footnote 15), levels have been found to vary among children of different socioeconomic status and other demographic characteristics (CD, p. 4–21).

associated with adverse effects (66 FR 1206).[8]

EPA's research program, with other Federal agencies defines, encourages and conducts research needed to locate and assess serious risks and to develop methods and tools to characterize and help reduce risks. For example, EPA's Integrated Exposure Uptake Biokinetic Model for Lead in Children (IEUBK model) for Pb in children and the Adult Lead Methodology are widely used and accepted as tools that provide guidance in evaluating site specific data. More recently, in recognition of the need for a single model that predicts Pb concentrations in tissues for children and adults, EPA is developing the All Ages Lead Model (AALM) to provide researchers and risk assessors with a pharmacokinetic model capable of estimating blood, tissue, and bone concentrations of Pb based on estimates of exposure over the lifetime of the individual. EPA research activities on substances including Pb focus on better characterizing aspects of health and environmental effects, exposure and control or management of environmental releases (see *http://www.epa.gov/ord/researchaccomplishments/index.html*).

D. 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 Criteria Document 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. The first draft of the Criteria Document (USEPA, 2005b) 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 Criteria Document (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 Criteria Document), released on July 31, 2006, was discussed at an August 15, 2006 CASAC teleconference. The final Criteria Document was released on September 30, 2006 (USEPA, 2006a; cited throughout this preamble as CD). While the Criteria Document focuses on new scientific information available since the last review, it 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 (Henderson, 2006). 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 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 Criteria Document. These documents were reviewed by CASAC and the public at a public meeting on February 6–7, 2007 (Henderson, 2007a).

Subsequent to that meeting, EPA conducted full-scale human exposure and health risk assessments, although no further work was done on the ecological assessment due to resource limitations. A second draft Risk Assessment Report (USEPA, 2007a), containing full-scale human exposure and health risk assessments, 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, we conducted additional human exposure and health risk assessments. A final Risk Assessment Report (USEPA, 2007b) and final Staff Paper (USEPA, 2007c) were released on November 1, 2007.

The final Staff Paper presents OAQPS staff's evaluation of the policy implications of the key studies and scientific information contained in the Criteria Document and presents and interprets results from the quantitative risk/exposure analyses conducted for this review. Further, the Staff Paper presents OAQPS staff recommendations on a range of policy options for the Administrator to consider concerning whether, and if so how, to review the primary and secondary Pb NAAQS. Such an evaluation is intended to help ''bridge the gap'' between the scientific assessment contained in the Criteria Document 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, the Staff Paper considered the available scientific evidence and quantitative risk-based analyses, together with related limitations and uncertainties, and focused on the information that is most pertinent to evaluating the basic elements of air quality standards: Indicator,[9] averaging time, form,[10] 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 OAQPS staff recommendations presented in the Staff Paper were informed by comments and advice received from CASAC in its reviews of the earlier draft Staff Paper and drafts of related risk/exposure assessment reports, as well as comments on these earlier draft documents submitted by public commenters.

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 finalize the Staff Paper no later than November 1, 2007, which we have done. 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. 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.

[8] See 2001 regulation to establish standards for lead-based paint hazards in most pre-1978 housing and child-occupied facilities (66 FR 1206).

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

[10] The ''form'' of a standard defines the air quality statistic that is to be compared to the level of the standard in determining whether an area attains the standard.

The EPA invites general, specific, and/or technical comments on all issues discussed in this ANPR, including issues related to the Agency's review of the primary and secondary Pb NAAQS (sections III and IV below) and associated monitoring considerations (section V below). EPA also invites comments on all information, findings, and recommendations presented in this notice (section VI below).

A public meeting of the CASAC will be held on December 12–13, 2007 for the purpose of providing advice and recommendations to the Administrator based on its review of this ANPR and the recently released final Staff Paper and Risk Assessment Report. Information about this meeting was published in the **Federal Register** on November 20, 2007 (72 FR 65335–65336).

E. Implementation Considerations

Currently only two areas in the United States are designated as non-attainment of the Pb NAAQS. If the Pb NAAQS is significantly lowered as a result of this review, it is likely (based on a review of the current air quality monitoring data) that many more areas would be classified as non-attainment (see section 2.3.2.5 of the Staff Paper for more details). States with Pb non-attainment areas would be required to develop ''State Implementation Plans'' that identify and implement specific air pollution control measures that would reduce the ambient Pb concentrations to below the Pb NAAQS. If the Pb NAAQS is revised to a lower level, States may be able to attain the revised NAAQS by implementing air pollution controls on lead emitting industrial sources. These controls include such measures as fabric filter particulate controls and fugitive dust controls. However, at some of the lower Pb concentration levels that have been identified for consideration in this review, it may become necessary in some areas to implement controls on nonindustrial sources such as dust from roadways, dust from construction, and/or demolition sites.

As described in further detail in the Staff Paper (see Section 2.2), Pb is emitted from a wide variety of source types. The top five categories of sources of Pb emissions included in the EPA's 2002 National Emissions Inventory (NEI) include: Mobile sources;[11] industrial, commercial, institutional and process boilers; utility boilers; iron and steel foundries; and primary Pb smelting (see Staff Paper Section 2.2).

III. The Primary Standard

This section presents information relevant to the review of the primary Pb NAAQS, including information on the health effects associated with Pb exposures, results of the human exposure and health risk assessment, and considerations related to evaluating the adequacy of the current standard and alternative standards that might be appropriate for the Administrator to consider.

A. Health Effects Information

The following summary focuses on health endpoints associated with the range of exposures considered to be most relevant to current exposure levels and makes note of 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 can be recognized as a significant Pb exposure pathway, particularly for young children, for which dust ingested via hand-to-mouth activity can be a more important source of Pb exposure than inhalation, although dust can be resuspended through household activities and pose an inhalation risk as well (CD, p. 3–27 to 3–28).[12] Some studies have found that dietary intake of Pb may be a predominant source of Pb exposure among adults, greater than consumption of water and beverages or inhalation (CD, p. 3–43).[13] 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, neurological effects in young children, are epidemiological data substantiated by toxicological data that provide biological plausibility and insights on mechanisms of action (CD, sections 5.3, 6.2 and 8.4.2).

In recognition of the multi-pathway aspects of Pb, and the use of an internal exposure metric in health risk assessment, the next section describes the internal disposition or distribution of Pb, and the use of blood Pb as an internal exposure or dose metric. This is followed by a discussion of the nature of Pb-induced health effects that emphasizes those with the strongest evidence. Potential impacts of Pb exposures on public health, including recognition of potentially susceptible or vulnerable subpopulations, are then discussed. Finally, key observations about Pb-related health effects are summarized.

1. Internal Disposition—Blood Lead as Dose Metric

The health effects of Pb 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 briefly summarizes the current state of knowledge of Pb disposition pertaining to both inhalation and ingestion routes of exposure as described in the Criteria Document.

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 to the gastrointestinal tract (CD, pp. 4–3). Lead associated with larger particles, which are predominantly deposited in the head and conducting airways (e.g., nasal

[11] The emissions estimates identified as mobile sources in the current NEI are currently limited to combustion of general aviation gas in piston-engine aircraft. Lead emissions estimates for other mobile source emissions of Pb (e.g., brake wear, tire wear, and others) are not included in the current NEI.

[12] For example, the Criteria Document states the following: ''Given the large amount of time people spend indoors, exposure to Pb in dusts and indoor air can be significant. For children, dust ingested via hand-to-mouth activity is often a more important source of Pb exposure than inhalation. Dust can be resuspended through household activities, thereby posing an inhalation risk as well. House dust Pb can derive both from Pb-based paint and from other sources outside the home. The latter include Pb-contaminated airborne particles from currently operating industrial facilities or resuspended soil particles contaminated by deposition of airborne Pb from past emissions.'' (CD, p. E–6)

[13] Some recent exposure studies have evaluated the relative importance of diet to other routes of Pb exposure. In reports from the NHEXAS, Pb concentrations measured in households throughout the Midwest were significantly higher in solid food compared to beverages and tap water (Clayton *et al.*, 1999; Thomas *et al.*, 1999). However, beverages appeared to be the dominant dietary pathway for Pb according to the statistical analysis (Clayton *et al.*, 1999), possibly indicating greater bodily absorption of Pb from liquid sources (Thomas *et al.*, 1999). Dietary intakes of Pb were greater than those calculated for intake from home tap water or inhalation on a µg/day basis (Thomas *et al.*, 1999). The NHEXAS study in Arizona showed that, for adults, ingestion was a more important Pb exposure route than inhalation (O'Rourke *et al.*, 1999). (CD, p. 3–43)

pharyngeal and tracheobronchial regions of respiratory tract), may be transported 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 directly through the ingestion route or indirectly following inhalation.

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). 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 Criteria Document ''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).

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). For example, 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, 2005a). 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 preventative measures (CDC, 2005a, ACCLPP, 2007).[14]

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). The Criteria Document summarizes related information as follows (CD, p. E–6).

> In the United States, decreases in mobile sources of Pb, resulting from the phasedown of Pb additives created a 98% decline in emissions from 1970 to 2003. NHANES data show a consequent parallel decline in blood-Pb levels in children aged 1 to 5 years from a geometric mean of ~15 μg/dL in 1976–1980 to 1–2 μg/dL in the 2000–2004 period.
>
> While levels in the U.S. general population, including geometric mean levels in children aged 1–5, have declined significantly, mean levels have been found to vary among children of different socioeconomic status (SES) and other demographic characteristics (CD, p. 4–21).[15]
>
> 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).

[14] 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, 2005a). CDC's Advisory Committee on Childhood Lead Poisoning Prevention recently provided recommendations regarding interpreting and managing blood Pb levels below 10 μg/dL in children and reducing childhood exposures to Pb (ACCLPP, 2007).

[15] For example, while the 2001–2004 median blood level for children aged 1–5 of all races and ethnic groups is 1.6 μg/dL, the median for the subset living below the poverty level is 2.3 μg/dL and 90th percentile values for these two groups are 4.0 μg/dL and 5.4 μg/dL, respectively. Similarly, the 2001–2004 median blood level for black, non-hispanic children aged 1–5 is 2.5 μg/dL, while the median level for the subset of that group living below the poverty level is 2.9 μg/dL and the median level for the subset living in a household with income more than 200% of the poverty level is 1.9 μg/dL. Associated 90th percentile values for 2001–2004 are 6.4 μg/dL (for black, non-hispanic children aged 1–5), 7.7 μg/dL (for the subset of that group living below the poverty level) and 4.1 μg/dL (for the subset living in a household with income more than 200% of the poverty level). (*http://www.epa.gov/envirohealth/children/body_burdens/b1-table.htm*—then click on ''Download a universal spreadsheet file of the Body Burdens data tables'').

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

2. 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 includes heme biosynthesis and related functions; neurological development and function; reproduction and physical development; kidney function; cardiovascular function; and immune function. The weight of evidence varies across this array of effects and is comprehensively described in the Criteria Document. There is also some evidence of Pb carcinogenicity, primarily from animal studies, together with limited human evidence of suggestive associations (CD, Sections 5.6.2, 6.7, and 8.4.10).[16]

This review is focused on those effects most pertinent to ambient exposures, which given the reductions in ambient Pb levels over the past 30 years, are generally those associated with blood Pb levels in children and adults in the range of 10 μg/dL and lower. Tables 8–5 and 8–6 in the Criteria Document highlight the key such effects observed in children and adults, respectively (CD, pp. 8–60 to 8–62). The effects include neurological, hematological and immune effects for children, and hematological, cardiovascular and renal effects for adults. As evident from the discussions in Chapters 5, 6 and 8 of the Criteria Document, ''neurotoxic effects in children and cardiovascular effects in adults are among those best substantiated as occurring at blood Pb concentrations as low as 5 to 10 μg/dL (or possibly lower); and these categories are currently clearly 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 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). Some health effects associated with blood Pb levels extend below 5 μg/dL, and some studies have observed these effects at the lowest blood levels considered. Threshold levels for these effects cannot be discerned from the currently available studies. For example, the Criteria Document also states the following (CD, p. 6–269).

> Recent studies of Pb neurotoxicity in children consistently indicate that blood Pb levels <10 μg/dL are associated with neurocognitive deficits. The data are also suggestive that these effects may be seen at blood Pb levels ranging down to 5 μg/dL, or perhaps somewhat lower, but the evidence is less definitive.[17]

Since effects on children's developing nervous system are considered to be the sentinel effects in this review, and are the focus of the quantitative risk assessment conducted for this review (discussed below in section III.B), these effects are discussed briefly below. 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). The differing evidence and associated strength of the evidence for these different effects is described in detail in the Criteria Document.

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 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). 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). As discussed in the Criteria Document, ''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 Criteria Document 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

[16] 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 considers Pb a probable carcinogen (*http://www.epa.gov/iris/subst/0277.htm;* CD, p. 6–195).

[17] The Criteria Document further states ''Collectively, the prospective cohort and cross-sectional studies offer evidence that exposure to Pb affects the intellectual attainment of preschool and school age children at blood Pb levels <10 μg/dL (most clearly in the 5 to 10 μg/dL range, but, less definitively, possibly lower).'' (p. 6–269)

(CD, Section 5.3.5), as well as associated mechanistic findings. With regard to persistence of effects the Criteria Document states the following (CD, p. 8–67):

> Persistence or apparent ''irreversibility'' of effects can result from two different scenarios: (1) Organic damage has occurred without adequate repair or compensatory offsets, or (2) exposure somehow persists. As Pb exposure can also derive from endogenous sources (e.g., bone), a performance deficit that remains detectable after external exposure has ended, rather than indicating irreversibility, could reflect ongoing toxicity due to Pb remaining at the critical target organ or Pb deposited at the organ post-exposure as the result of redistribution of Pb among body pools.
>
> The persistence of effect appears to depend on the duration of exposure as well as other factors that may affect an individual's ability to recover from an insult. The likelihood of reversibility also seems to be related, at least for the adverse effects observed in certain organ systems, to both the age-at-exposure and the age-at-assessment.

The evidence with regard to persistence of Pb-induced deficits observed in animal and epidemiological studies is described in discussion of those studies in the Criteria Document (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 in real life,'' as well as increased risk of antisocial and delinquent behavior (CD, Section 6.2.16).

As discussed in the Criteria Document, 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 Criteria Document (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.

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); ''the most compelling evidence for effects at blood Pb levels <10 μg/dL comes from an international pooled analysis of seven prospective cohort studies (n = 1,333) by Lanphear et al. (2005)'' (CD, p. 6–67 and sections 6.2.13 and 6.2.3.1.11). This pooled analysis estimated a decline of 6.2 points in full scale IQ (with a 95% confidence interval bounded by 3.8 and 8.6) occurring between approximately 1 and 10 μg/dL blood Pb level, measured concurrent with the IQ test (CD, p. 6–76). As discussed below in section III.B, this analysis (Lanphear et al., 2005) was relied upon in the quantitative risk assessment.

3. 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).[18] Blood Pb levels have also declined in the U.S. adult population over this time period (CD, Section 4.3.1.3).[19] As noted in the Criteria Document, ''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).

a. At-Risk Subpopulations

Potentially at-risk subpopulations include 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) (USEPA 1986a, p. 1–154). 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, 2005c) 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 Criteria Document (e.g., CD, Section 8.5.3): Age, health status, and genetic composition. 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). Further early life exposures have been associated with increased risk of cardiovascular effects in humans later in life (CD, p. 8–74). Early life exposures have also been associated with increased risk, in animals, of neurodegenerative effects later in life (CD, p. 8–74).[20] Health status is another

[18] 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). Median and 90th percentile values have also declined from 15 μg/dL and 25 μg/dL, respectively, in 1976–1980, to 1.6 μg/dL and 3.9 μg/dL, respectively in 2003–04 (*http://www.epa.gov/envirohealth/children/body_burdens/b1-table.htm*).

[19] For example, NHANES data for older adults (60 years of age and older) indicate a decline in overall population geometric mean blood Pb level from 3.4 μg/dL in 1991–1994 to 2.2 μg/dL in 1999–2002; the trend for adults between 20 and 60 years of age is similar to that for children 1 to 5 years of age (*http://www.cdc.gov/mmwr/preview/mmwrhtml/mm5420a5.htm).*

[20] Specifically, among young adults who lived as children in an area heavily polluted by a smelter and whose current Pb exposure was low, higher bone Pb levels were associated with higher systolic and diastolic blood pressure (CD, p. 8–74). In adult rats, greater early exposures to Pb are associated with increased levels of amyloid protein precursor, a marker of risk for neurodegenerative disease (CD, p. 8–74).

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 maximal (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). 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 helps inform an understanding of specific periods of development with increased vulnerability to specific types of effect (CD, Section 5.3), and indicates the 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 the potential importance of exposures of duration as short as weeks to months. For example, the animal studies suggest that the gestation period is the most sensitive life stage followed by early neonatal stage, and that within these life stages, critical windows of vulnerability are likely to exist (CD, Section 5.9 and p. 5–245).

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 because of its influence on exposure and susceptibility. 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. The Criteria Document recognizes, however, other population subgroups as described above may also be at risk of Pb-related health effects of public health concern.

b. Potential Public Health Impacts

As discussed in the Criteria Document, there are potential public health implications of low-level Pb exposure, indexed by blood Pb levels, associated with several health endpoints identified in the Criteria Document (CD, Section 8.6).[21] These include potential impacts on population IQ, which is the focus of the quantitative risk assessment conducted for this review, as well as heart disease and chronic kidney disease, which are not included in the quantitative risk assessment (CD, Sections 8.6, 8.6.2, 8.6.3 and 8.6.4). It is noted that there is greater uncertainty associated with effects at the lower levels of blood Pb, and that there are differing weights of evidence across the effects observed.[22] With regard to potential implications of Pb effects on IQ, the Criteria Document recognizes the ''critical'' distinction between population and individual risk, noting 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).[23] A downward shift in the mean IQ value is associated with both substantial decreases in percentages achieving very high scores and substantial increases in the percentage of individuals achieving very low scores (CD, p. 8–81).[24] For an individual functioning in the low IQ 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).

The magnitude of a public health impact is dependent upon the size of population affected and type or severity of the effect. As summarized above, there are several population groups that may be susceptible or vulnerable to effects associated with exposure to Pb, including young children, particularly those in families of low SES (CD, p. E–15), as well as individuals with hypertension, diabetes, and chronic renal insufficiency (CD, p. 8–72). 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 (as noted in Table 3–3 of the Staff Paper).[25]

With regard to estimates of the size of potentially vulnerable subpopulations living in areas of increased exposure related to ambient Pb, the information is still more limited. 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). Air quality analyses (presented in Chapter 2 of the Staff Paper) indicate dramatically higher Pb concentrations at monitors near sources as compared with those more remote. As described in Section 2.3.2.1 of the Staff Paper, however, since the 1980s the number of Pb monitors has been significantly reduced by states (with EPA guidance that monitorings well below the current NAAQS could be shut down) and a lack of monitors near some large sources may lead to underestimates of the extent of occurrences of relatively higher Pb concentrations. The significant limitations of our monitoring and emissions information constrain our efforts to characterize the size of at-risk populations in areas influenced by

[21] The differing evidence and associated strength of the evidence for these different effects is described in detail in the Criteria Document.

[22] As is described in Section III.B.2.a, CASAC, in their comments on the analysis plan for the risk assessment described in this notice, placed higher priority on modeling the child IQ metric than the adult endpoints (e.g., cardiovascular effects).

[23] 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).

[24] 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).

[25] For example, approximately 4.8 million children live in poverty, while the estimates of numbers of adults with hypertension, diabetes or chronic kidney disease are on the order of 20 to 50 million (see Table 3–3 of Staff Paper).

policy-relevant sources of ambient Pb. For example, the limited size and spatial coverage of the current Pb monitoring network constrains our ability to characterize current levels of airborne Pb in the U.S. 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 National Emissions Inventory (NEI), with monitors within a mile of only 2 of 26 facilities in the 2002 NEI with emissions greater than 5 tons per year (tpy). Additionally, there are various uncertainties and limitations associated with source information in the NEI.

In recognition of the significant limitations associated with the currently available information on Pb emissions and airborne concentrations in the U.S. and the associated exposure of potentially at-risk populations, Chapter 2 of the Staff Paper summarizes the information in several different ways. For example, analyses of the current monitoring network indicated the numbers of monitoring sites that would exceed alternate standard levels, taking into consideration different statistical forms. These analyses are also summarized with regard to population size in counties home to those monitoring sites (see Appendix 5.A of the Staff Paper). 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 magnitude of Pb emissions from sources that could influence air Pb concentrations. 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 was not feasible within the time and data constraints of this review. Instead, we have 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 or in counties in which the aggregate Pb emissions is greater than or equal to 0.1 tpy per 1000 square miles (see Tables 3–4 and 3–5, respectively, in the Staff Paper).

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 Criteria 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. [26]

4. Key Observations

The following key observations are based on the available health effects evidence and the evaluation and interpretation of that evidence in the Criteria Document.

- Lead exposures occur both by inhalation and by ingestion (CD, Chapter 3). As stated in the Criteria Document, ''given the large amount of time people spend indoors, exposure to Pb in dusts and indoor air can be significant'' (CD, p. 3–27).
- 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 normal childhood hand-to-mouth activity (CD, p. E–15, Chapter 3 and Section 6.2.1).
- 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, although difficulty of sample collection has precluded widespread use in epidemiological studies to date (CD, Chapter 4).
- 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 (CD, Section 4.3).
- Both epidemiological and toxicologic studies have shown that environmentally relevant levels of Pb affect many different organ systems (CD, p. E–8). Many associations of health effects with Pb exposure 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 for neurological effects (see subsequent bullet). Supportive evidence from toxicological studies provides biological plausibility for the observed effects. (CD, Chapters 5, 6 and 8)
- Pb exposure is associated with a variety of neurological effects in children, notably intellectual attainment and school performance. Both qualitative and quantitative evidence, with further support from animal research, indicates a robust and consistent effect of Pb exposure on neurocognitive ability at mean concurrent blood Pb levels in the range of 5 to 10 μg/dL. A recent analysis of a nationally representative U.S. sample suggested Pb effects on intellectual attainment of young children at population mean concurrent blood Pb levels ranging down to as low as 2 μg/dL. (CD, Sections 5.3, 6.2, 8.4.2 and 6.10)
- 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. (CD, Sections 6.1 and 8.4.2)
- For the quantitative risk assessment for neurocognitive ability in young children (described in Chapter 4 of the Staff Paper), the Staff Paper chose 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, particularly below 10 μg/dL (CD, Sections 6.2.13 and 8.6).
- At mean blood Pb levels, in children, on the order of 10 μg/dL, and somewhat lower, associations have been found with effects to the immune system, including altered macrophage activation, increased IgE levels and associated increased risk for autoimmunity and asthma (CD, Sections 5.9, 6.8, and 8.4.6).
- In adults, with regard to cardiovascular outcomes, the Criteria Document included the following summary (CD, p. E–10).

Epidemiological studies have consistently demonstrated associations between Pb exposure and enhanced risk of deleterious cardiovascular outcomes, including increased blood pressure and incidence of hypertension. [27] A meta-analysis of

[26] 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).

[27] The Criteria Document states that ''While several studies have demonstrated a positive correlation between blood pressure and blood Pb concentration, others have failed to show such association when controlling for confounding

Continued

numerous studies estimates that a doubling of blood-Pb level (e.g., from 5 to 10 μg/dL) is associated with ~1.0 mm Hg increase in systolic blood pressure and ~0.6 mm Hg increase in diastolic pressure. Studies have also found that cumulative past Pb exposure (e.g., bone Pb) may be as important, if not more, than present Pb exposure in assessing cardiovascular effects. The evidence for an association of Pb with cardiovascular morbidity and mortality is limited but supportive.

Studies of nationally representative U.S. samples observed associations between blood Pb levels and increased systolic blood pressure at population mean blood lead levels less than 5 μg/dL, particularly among African Americans (CD, Section 6.5.2). With regard to gender differences, the Criteria Document states the following (CD, p. 6–154).

Although females often show lower Pb coefficients than males, and Blacks higher Pb coefficients than Whites, where these differences have been formally tested, they are usually not statistically significant. The tendencies may well arise in the differential Pb exposure in these strata, lower in women than in men, higher in Blacks than in Whites. The same sex and race differential is found with blood pressure.

Animal evidence provides confirmation of Pb effects on cardiovascular functions. (CD, Sections 5.5, 6.5, 8.4.3 and 8.6.3)

- Renal effects, evidenced by reduced renal filtration, have also been associated with Pb exposures indexed by bone Pb levels and also with mean blood Pb levels in the range of 5 to 10 μg/dL in the general adult population, with the potential adverse impact of such effects being enhanced for susceptible subpopulations including those with diabetes, hypertension, and chronic renal insufficiency (CD, Sections 6.4, 8.4.5, and 8.6.4). The full significance of this effect is unclear, given that other evidence of more marked signs of renal dysfunction have not been detected at blood Pb levels below 30–40 μg/dL in large studies of occupationally-exposed Pb workers (CD, pp. 6–270 and 8–50). [28]
- 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. (CD, Sections 5.2, 5.3, 6.3 and 6.9.2)

B. Human Exposure and Health Risk Assessments

This section presents a brief summary of the human exposure and health risk assessments conducted by EPA for this review. The complete full-scale assessment, which includes specific analyses conducted to address CASAC comments and advice on an earlier draft assessment, is presented in the final Risk Assessment Report (USEPA, 2007b).

The focus of this Pb NAAQS risk assessment is on Pb derived from those sources emitting Pb to ambient air. The design and implementation of this assessment needed to address significant limitations and complexity that go far beyond the situation for similar assessments typically performed for other criteria pollutants. Not only was the risk assessment constrained by the timeframe allowed for this review in the context of breadth of information to address, it was also constrained by significant limitations in data and modeling tools for the assessment. Furthermore, the multimedia and persistent nature of Pb, and the role of multiple exposure pathways, add significant 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, the risk assessment could not fully incorporate all of the important complexities associated with Pb. Consequently, in characterizing risk associated with the ambient air-related [29] (policy-relevant) sources and exposures, simplifying assumptions were made in several areas. For example, people are also exposed to Pb that originates from nonair sources, including leaded paint or drinking water distribution systems. For this assessment, the Pb from these nonair sources is collectively referred to as ''policy-relevant background.'' [30] [31] Although deposition of airborne Pb is a major source of Pb in food (CD, p. 3–54) and may also contribute to Pb in drinking water, the contribution from air pathways to these nonair exposure pathways could not be explicitly modeled, and these contributions are treated as though they were part of the policy-relevant background. [32] This means that some benefits associated with emissions reductions are excluded to the extent that reduced air emissions will eventually mean less Pb in water and food.

An overview of the human health risk assessment completed in the last review of the Pb NAAQS in 1990 (USEPA, 1990a) is presented first below, followed

factors such as tobacco smoking, exercise, body weight, alcohol consumption, and socioeconomic status. Thus, the studies that have employed blood Pb level as an index of exposure have shown a relatively weak association with blood pressure. In contrast, the majority of the more recent studies employing bone Pb level have found a strong association between long-term Pb exposure and arterial pressure (Chapter 6). Since the residence time of Pb in the blood is relatively short but very long in the bone, the latter observations have provided rather compelling evidence for a positive relationship between Pb exposure and a subsequent rise in arterial pressure'' (CD, pp. 5–102 to 5–103). Further, in consideration of the meta-analysis also described here, the Criteria Document stated that ''The meta-analysis provides strong evidence for an association between increased blood Pb and increased blood pressure over a wide range of populations'' (CD, p. 6–130) and ''the meta-analyses results suggest that studies not detecting an effect may be due to small sample sizes or other factors affecting precision of estimation of the exposure effect relationship'' (CD, p. 6–133).

[28] In the general population, both cumulative and circulating Pb has been found to be associated with longitudinal decline in renal functions. In the large NHANES III study, alterations in urinary creatinine excretion rate (one indicator of possible renal dysfunction) was observed in hypertensives at a mean blood Pb of only 4.2 μg/dL. These results provide suggestive evidence that the kidney may well be a target organ for effects from Pb in adults at current U.S. environmental exposure levels. The magnitude of the effect of Pb on renal function ranged from 0.2 to −1.8 mL/min change in creatinine clearance per 1.0 μg/dL increase in blood Pb in general population studies. However, the full significance of this effect is unclear, given that other evidence of more marked signs of renal dysfunction have not been detected at blood Pb levels below 30–40 μg/dL among thousands of occupationally-exposed Pb workers that have been studied. (CD, p. 6–270)

[29] Ambient air related sources are those emitting Pb into the ambient air (including resuspension of previously emitted Pb, that may include Pb paint from older buildings which has weathered and impacted outdoor soil with subsequent resuspension), 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).

[30] 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. Rather, it is simply intended to define the focus of this analysis.

[31] In the context of NAAQS for other criteria pollutants which are not multimedia in nature, such as ozone, the term policy-relevant background is used to distinguish anthropogenic air emissions from naturally occurring non-anthropogenic emissions to separate pollution levels that can be controlled by U.S. regulations from levels that are generally uncontrollable by the United States (USEPA, 2007d). In the case of Pb, however, due to the multimedia, multipathway nature of human exposures to Pb, policy-relevant background is defined more broadly to include not only the ''quite low'' levels of naturally occurring Pb emissions into the air from non-anthropogenic sources such as volcanoes, sea salt, and windborne soil particles from areas free of anthropogenic activity, but also Pb from nonair sources, generally including leaded paint or drinking water distribution systems, which are collectively referred to in the risk assessment described here as ''policy-relevant background'' (USEPA, 2007b, p. 2–28, p. 1–3).

[32] Furthermore, although Pb from indoor paint is considered a component of policy-relevant background, for this analysis, it may be reflected somewhat in estimates developed for policy-relevant sources due to modeling constraints (see USEPA, 2007b).

by a summary of key aspects of the approach used in this assessment, including key limitations and uncertainties. The key assessment results are then summarized.

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, 1990b; 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 assessment 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 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.

2. Design Aspects of Exposure and Risk Assessments

This section provides an overview of key elements of the assessment design, inputs, and methods, and includes identification of key uncertainties and limitations.

a. CASAC Advice

The CASAC conducted a consultation 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 plan included: (1) Placing a higher priority on modeling the child IQ metric than the adult endpoints (e.g., cardiovascular effects), (2) 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 (3) concurring with use of the IEUBK biokinetic blood Pb model. Taking these comments into account, a pilot phase assessment was conducted to test the risk assessment methodology being developed for the subsequent full-scale assessment. The pilot phase assessment is described in the first draft Staff Paper and accompanying technical report (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 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). These included a recommendation that the general urban case study be augmented by the inclusion of risk analyses in specific urban areas of the U.S. In this regard, they specifically stated the following (Henderson, 2007b, p. 3).

> * * * 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 NAAQS. 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.

Hence, EPA included additional case studies in the risk assessment. Further, CASAC recommended using a concentration-response function with a change in slope near 7.5 μg/dL. Accordingly, EPA included such an additional concentration-response function in the risk assessment. Results from the initial full-scale analyses, along with comments from CASAC, such as those described here, and the public resulted in a final version of the full-scale assessments which is summarized in this notice and presented in greater detail in the Risk Assessment Report and associated appendices (USEPA, 2007b). While these additional analyses were developed in response to CASAC recommendations, there has not been review of the completed analyses by CASAC.

b. Health Endpoint, Risk Metric and Concentration-Response Functions

The health endpoint on which the quantitative health risk assessment focuses 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 Criteria Document, 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 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 Criteria Document 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).

Given the evidence described in detail in the Criteria Document (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 in the Criteria Document (CD, p. 8–63 to 8–64), 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). As discussed in the Criteria Document, 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.

In the additional risk analyses performed subsequent to the August 2007 CASAC public meeting, the 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 Pb 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 Pb 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 (of 1 μg/dL for the concurrent blood Pb metric), 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 stratification 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 Pb concentrations and higher blood Pb concentrations. For these analyses, the study population was stratified at the specified peak blood Pb level and separate linear models were fitted to the concurrent blood Pb data for the children in the two study population subgroups. 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 Pb levels less than 7.5 μg/dL and 244 children had maximal blood Pb levels less than 10 μg/dL. While these children may better represent current blood Pb 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 study population subgroup with peak blood Pb below 7.5 μg/dL, 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, greater confidence is placed in the log-linear model form compared to the dual-linear stratified models for purposes of the risk assessment described in this notice. 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), greatest confidence is assigned 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.

c. Case Study Approach

For the risk assessment described in this notice, a case study approach was employed as described in Sections 2.2 (and subsections) and 5.1.3 of the Risk Assessment Report (USEPA, 2007b). The four types of case studies included in the assessment are the following:

- *Location-specific urban case studies:* Three urban case studies focus on specific urban areas (Cleveland, Chicago and Los Angeles) 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. As stated above, these case studies were developed in response to CASAC recommendations and there has not been review of the completed analyses for these case studies by CASAC
- *General urban case study:* The general urban case study is a nonlocation-specific analysis that uses several simplifying assumptions regarding ambient air Pb levels and demographics to produce a simplified representation of urban areas.
- *Primary Pb smelter case study:*[33] This case study estimates 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:*[34] 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 III.B.2.g below, however, a variety of significant limitations in the approaches employed for this case and associated large uncertainties in these results are recognized that preclude considering this case study to be illustrative of the larger set of areas influenced by similarly sized Pb sources. Risk estimates for this case study (presented in detail in the Risk Assessment Report (USEPA, 2007b)) are lower than those for the other case studies.

d. Air Quality Scenarios

Air quality scenarios assessed 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 levels of 0.5, 0.2, 0.05, and 0.02 μg/m^3, with a monthly averaging time, as well as a level of 0.2 μg/m^3 scenario using a quarterly averaging time.[35] The current NAAQS scenario for the urban case studies assumes ambient air Pb concentrations higher than actual current conditions. While it is extremely unlikely that Pb concentrations in urban areas would rise to meet the current NAAQS and there are limitations and uncertainties associated with the approach used (as described in Section III.B.2.g below), this scenario was included to provide some perspective on risks associated with just meeting the current NAAQS relative to current conditions. When evaluating these results it is important to keep the limitations and uncertainties in mind.

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 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. 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 μg/m^3 as a maximum quarterly average) and a high-end ambient air Pb level in large urban areas (0.87 μg/m^3 as a maximum quarterly average).

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).

e. Categorization of Policy-Relevant Exposure Pathways

To inform policy aspects of the Pb NAAQS review, the assessment estimates for blood Pb and IQ loss were divided into two components: The fraction associated with policy-relevant pathways, which include inhalation, outdoor soil/dust ingestion and indoor dust ingestion, and the fraction associated with background (e.g., diet and drinking water). The policy-relevant pathways are further divided into two categories, ''recent air'' and ''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.,

[33] See Section III.B.2.a for a summary of CASAC's comment with regard to the primary and secondary Pb smelter case studies.

[34] See Section III.B.2.a for a summary of CASAC's comment with regard to the primary and secondary Pb smelter case studies.

[35] For further discussion of the air quality scenarios and averaging times included in the risk assessment, see section 2.3.1 of the Risk Assessment Report (USEPA, 2007b)

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 indoor Pb paint. In the risk assessment, estimates of contribution to blood Pb and IQ loss were developed for the following pathways or pathway combinations:

- Inhalation of ambient air Pb (i.e., ''recent air'' Pb): This is derived using the blood Pb estimate resulting from Pb exposure limited to the inhalation pathway (and will include exposures to Pb in ambient air from all sources contributing to the ambient air concentration estimate).
- Ingestion of ''recent air'' indoor dust Pb: This is derived using the blood Pb estimate resulting from Pb exposure limited to ingestion of the Pb in indoor dust that is predicted to be associated with ambient air concentrations (i.e., via the air concentration coefficient in the regression-based dust models or via the mechanistic component of the hybrid blood Pb model (see Section 3.1.4 of the Risk Assessment Report). For the primary Pb smelter case study, estimates for this pathway are not separated from estimates for the pathway described in the subsequent bullet due to uncertainty regarding this categorization with the model used for this case study (Section 3.1.4.2 of the Risk Assessment Report).
- Ingestion of ''other'' indoor dust Pb: This is derived using the blood Pb estimate resulting from Pb exposure limited to ingestion of the Pb in indoor dust that is not predicted to be associated with ambient air concentrations (i.e., that predicted by the intercept in the dust models plus that predicted by the outdoor soil concentration coefficient, for models that include an intercept (Section 3.1.4 of the Risk Assessment Report)). This is interpreted to represent indoor paint, outdoor soil/dust, and additional sources of Pb to indoor dust including historical air (see Risk Assessment Report, Section 2.4.3). As the intercept in regression dust models will be inclusive of error associated with the model coefficients, this category also includes some representation of dust Pb associated with current ambient air concentrations (described in previous bullet). For the primary Pb smelter case study, estimates for this pathway are not separated from estimates for the pathway described above due to uncertainty regarding this categorization with the model used for this case study (Risk Assessment Report, Section 3.1.4.2). This pathway is included in the ''past air'' category.
- Ingestion of outdoor soil/dust Pb: This is derived using the blood Pb estimate resulting from Pb exposure limited to ingestion of outdoor soil/dust Pb. This pathway is included in the ''past air'' category (and could include contamination from historic Pb emissions from automobiles and Pb paint).
- Ingestion of drinking water Pb: This is derived using the blood Pb estimate resulting from Pb exposure limited to ingestion of drinking water Pb. This pathway is included in the policy-relevant background category.
- Ingestion of dietary Pb: This is derived using the blood Pb estimate resulting from Pb exposure limited to ingestion of dietary Pb. This pathway is included in the policy-relevant background category.

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), modeling for the assessment has only affected the exposure pathways categorized as recent air (inhalation and ingestion of that portion of indoor dust associated with outdoor ambient air). The assessment has 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 aspect of the analysis will tend to underestimate the reductions in risk associated with alternative NAAQS. However, this does not mean that overall risk has been underestimated. The net effect of all sources of uncertainty or bias in the analysis, which may also tend to under- or overestimate risk, could not be quantified.

Additionally, there is uncertainty related to parsing out exposure and risk between background 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, indoor 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). The inclusion of indoor lead Pb as a component of ''other'' indoor air (and consequently as a component of ''past air'' exposure) represents a source of potential high bias in our prediction of total exposure and risk associated with past air because conceptually, exposure to indoor paint Pb is considered part of background exposure.

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.

f. Analytical Steps

The risk assessment includes four analytical steps, briefly described below and presented in detail in Sections 2.4.4, 3.1, 3.2, 4.1, and 5.1 of the Risk Assessment Report (USEPA, 2007b).

- *Characterization of Pb in ambient air:* The characterization of outdoor ambient air Pb levels uses different approaches depending on the case study (as explained in more detail below): (a) Source-oriented and non-source oriented monitors are assumed to represent different exposure zones in the city-specific case studies, (b) a single exposure level is assumed, based on monitoring data for various cities, for the general urban case study, and (c) ambient levels are estimated using air dispersion modeling based on Pb emissions from a particular facility in the point source case studies.
- *Characterization of outdoor soil/dust and indoor dust Pb concentrations:* Outdoor soil Pb levels are estimated using empirical data and/or fate and transport modeling. Indoor dust Pb levels are 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.[36]
- *Characterization of blood Pb levels:* Blood Pb levels for each exposure zone are derived from central-tendency blood Pb concentrations estimated using the

[36] Additional detail on the methods used in characterizing Pb concentrations in outdoor soil and indoor dust are presented in Sections 3.1.3 and 3.1.4 of the Risk Assessment, respectively. Data, methods and assumptions here used in characterizing Pb concentrations in these exposure media may differ from those in other analyses that serve different purposes.

Integrated Exposure and Uptake Biokinetic (IEUBK) model, and concurrent or lifetime average blood Pb is estimated from these outputs as described in Section 3.2.1.1 of the Risk Assessment Report (USEPA, 2007b). 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 study area, and (c) a geometric standard deviation (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. Additional detail on the methods used to model population blood Pb levels is presented in Sections 3.2.2 and 5.2.2.3 of the Risk Assessment Report (USEPA, 2007b).

• *Risk characterization (estimating IQ loss):* Concurrent or lifetime average blood Pb estimates for each simulated child in each case study population are converted into total Pb-related IQ loss estimates using the concentration-response functions described above.

Key limitations and uncertainties associated with the application of these specific analytical steps are summarized in Section III.B.2.g below.

g. Generating Multiple Sets of Risk Results

In the initial analyses for the full-scale assessment (USEPA, 2007a), EPA 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. These multiple modeling approaches are described in Section 2.4.6.2 of the final Risk Assessment Report (USEPA, 2007b). In consideration of comments provided by CASAC (Henderson, 2007b) on these analyses regarding which modeling approach they felt had greater scientific support, a pared down set of modeling combinations was identified as the core approach for the subsequent analyses. This core modeling approach includes the following key elements:

• Ambient air Pb estimates (based on monitors or modeling and proportional rollbacks, as described below),
• Background exposure from food and water (as described above),
• The hybrid indoor dust model specifically developed for urban residential applications (which predicts Pb in indoor dust as a function of ambient air Pb and nonair contribution),
• The IEUBK blood Pb model (which predicts blood Pb in young children exposed to Pb from multiple exposure pathways),
• The concurrent blood Pb metric,
• A GSD for concurrent blood Pb of 2.1 to characterize interindividual variability in blood Pb levels for a given ambient level, and
• four different functions relating concurrent blood Pb to IQ loss, including two log-linear models (one with a cutpoint and one with low-exposure linearization) and two dual-linear models with stratification, one stratified at 7.5 μg/dL peak blood Pb and the other at 10 μg/dL peak blood Pb.

For each case study, the core modeling approach employs a single set of modeling elements to estimate exposure and the four different concentration-response functions referenced above to derive four sets of risk results from the single set of exposure estimates. The spread of estimates resulting from application of all four functions captures much of the uncertainty associated model choice in this analytical step. Among these four functions, greater confidence is associated with estimates derived using the log-linear with low-exposure linearization concentration-response function as discussed above.

In addition to employing multiple concentration-response functions, the assessment includes various sensitivity analyses to characterize the potential impact of uncertainty in other key analysis steps on exposure and risk estimates. The sensitivity analyses and uncertainty characterization completed for the risk analysis are described in Sections 3.5, 4.3, 5.2.5 and 5.3.3 of the Risk Assessment Report (USEPA, 2007b).

h. Key Limitations and Uncertainties

As recognized above, EPA has made simplifying assumptions in several areas of this assessment due to the limited data, models, and time available. These assumptions and related limitations and uncertainties are described in the Risk Assessment Report (USEPA, 2007b). Key assumptions, limitations and uncertainties are briefly identified below. EPA considers these aspects of the assessment to be important to the interpretation of the exposure and risk estimates. In the presentation below, limitations (and associated uncertainty) are listed, beginning with those regarding design of the assessment or case studies, followed by those regarding estimation of Pb concentrations in ambient air indoor dust, outdoor soil/dust, and blood, and lastly regarding estimation of Pb-related IQ Loss.

• *Temporal aspects:* Exposure for the simulated child population begins at birth (including a prenatal maternal contribution) 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. These aspects are a simplification of population exposures that contributes some uncertainty to our exposure and risk estimates.

• *General urban case study:* This case study differs from the others in several ways. 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. 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. The details of this modeling is presented in the Risk Assessment Report (USEPA, 2007b). 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 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. This limitation is likely to have more significant impact on risk estimates associated with the full study than on those for the subareas (which are the portions of the study area with 1.5 km from the smelter facilities), and to perhaps have a more significant impact on risk estimates associated with the smaller secondary Pb smelter (see below). As noted above, 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), including a recommendation that the general urban case study be augmented by the inclusion of risk analyses in specific urban areas of the U.S. In this regard, they specifically stated the following (Henderson, 2007b, p. 3):

> 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 NAAQS. 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.

• *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 fugitive emissions), (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.[37] 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 results in low confidence in estimates for this case study. It is observed that exposure and risk estimates are lower than those for the other case studies. Although this case study was initially intended to be used as an example of areas near stationary sources of intermediate size (smaller than the primary Pb smelter), 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.

• *Location-specific urban case studies:* 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 U.S. Census block groups (and the children within those demographic units) were distributed among the exposure zones. The details of the approach used are described in Section 5.1.3 of the Risk Assessment Report (USEPA, 2007b). 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 adequately represents all locations that are closer to that monitor than to any of the others in the study area. In reality, across block groups there are 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. As recognized in Section, III.B.2.a, the analyses for these case studies were developed in response to CASAC recommendations on the July 2007 draft Risk Assessment (Henderson, 2007b) and there has not been review of the completed analyses by CASAC.

• *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, USEPA, 2007b, for detailed discussion). EPA 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 a proportional roll-up was simulated, 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 (USEPA, 2007b)).

• *Alternative NAAQS air quality scenarios:* In all case studies, proportional roll-down procedures were used to adjust ambient air Pb concentrations downward to attain

[37] The information supporting the air dispersion modeling for the primary Pb smelter case study provides substantially greater confidence in estimates for that case study.

alternative NAAQS (see Sections 2.3.1 and 5.2.2.1 of the Risk Assessment Report, USEPA, 2007b). 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 the 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.

• *Estimates of outdoor soil/dust Pb concentrations:* Outdoor soil Pb concentration for both the urban case studies and the primary Pb smelter case study are based on empirical data (see Section 3.1.3 of the Risk Assessment). To the extent that the underlying sampling data included areas containing older structures, the impact of Pb paint weathered from older structures on soil Pb levels will be reflected in these empirical estimates. In the case of the urban case studies, a mean value from a sample of houses built between 1940 and 1998 was used to represent soil Pb levels (see Section 3.1.3.1 of the Risk Assessment). Outdoor soil/dust Pb concentrations in all air quality scenarios have been set equal to the values for the current conditions scenarios. An impact of changes in air Pb concentrations on soil concentrations, and the associated impact on dust concentrations, blood Pb and risk estimates were not simulated. 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 (USEPA, 2007b), 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, it is possible 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, USEPA, 2007b) 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, USEPA, 2007b). 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, in evaluating exposure and risk reduction trends related to reducing ambient air Pb levels, focus has been placed on changes in total blood Pb rather than on estimates of ''recent air'' blood 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). However 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 unaccounted-for variables are roadside dust Pb and historical contributions to current levels of indoor dust Pb (e.g., Pb that entered a house in the past and continues to contribute to current dust Pb levels).

• *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). 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 [38] 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 95th 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 assumed 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 differ in 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, greater confidence is placed in estimates of total Pb exposure and risk in evaluating the impact of the current NAAQS and alternative NAAQS relative to current conditions.

• *Relating blood Pb levels to IQ loss:* Specification of the quantitative relationship between blood Pb level and

[38] For example, the GSD for the urban case studies, in the risk assessment described in this notice, was derived using NHANES data for the years 1999–2000.

IQ loss is subject to significant uncertainty at lower blood Pb levels (e.g., below 5 µg/dL concurrent blood Pb). As discussed earlier, 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. 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, USEPA, 2007b). The difference in absolute IQ loss estimates for the four concentration-response functions for a given case study/air quality scenario combination is typically close to a factor of 3. Estimates of differences in IQ loss between air quality scenarios (in terms of percent), however, are more similar across the four functions, although the function producing higher overall risk estimates (the dual linear function, stratified at 7.5 µg/dL, peak blood Pb) also produces larger absolute reductions in IQ loss compared with the other three functions.

3. Summary of Results

This section presents blood Pb and IQ loss estimates generated in the exposure and risk assessments. Blood Pb estimates are presented first, followed by IQ loss estimates.

a. Blood Pb Estimates

This section presents blood Pb estimates for the median (Table 1) and 95th (Table 2) population percentiles.[39] Each table presents estimates of blood Pb levels resulting from total Pb exposure across all pathways (policy relevant and background), as well as estimates of the percent contribution from ''recent air'' and ''recent plus past air'' exposure categories. As noted in Sections 4.2.4 of the Staff Paper 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 above (and in Section 4.2.7 of the Staff Paper). The ''recent air'' component of indoor dust Pb is the projected level associated with outdoor ambient air Pb levels, with outdoor ambient air potentially including resuspended, previously deposited Pb which may reflect the resuspension of historic levels of Pb from gasoline and from exterior house and building Pb paint. In presenting the 95th population percentile estimates, it is recognized that 5 percent of the child study population at each case study are estimated to have blood Pb levels above these estimates. Due to technical limitations, however, we believe that it is not possible at this point to reasonably predict the distribution of blood Pb levels for that top 5 percent. Observations regarding the blood Pb results presented in Tables 1 and 2 are presented in Section 4.3 of the Staff Paper.

[39] Blood Pb level estimates for current conditions for these cases studies differ from the national values associated with NHANES. For example, median blood Pb levels presented in Table 1 for the current conditions scenario for the urban case studies are somewhat larger than the national median from the NHANES data for 2003–2004. Specifically, values for the three location-specific urban case studies range from 1.7 to 1.8 µg/dL with the general urban case study having a value of 1.9 µg/dL (current-conditions mean) (see Table 1), while the median value from NHANES (2003–2004) is 1.6 µg/dL (*http://www.epa.gov/envirohealth/children/body_burdens/b1-table.htm*). NHANES values for the 95th percentile were not available for 2003–2004, precluding a comparison of modeled estimates presented in Table 2 against NHANES data. We note, however, that the 95th percentile value in 2001–2002 was 5.8 µg/dL (see footnote 7). However, NHANES values for the 90th percentile (for 2003–2004) were identified and these values can be compared against 90th percentile estimates generated for the urban case studies (see Risk Assessment Report, Appendix O, Section O.3.2 for the location-specific urban case study and Appendix N, Section N.2.1.2 for the general urban case study). The 90th percentile blood Pb levels for the current conditions scenario, for the three location-specific urban case studies range from 4.5 to 4.6 µg/dL, while the estimate for the general urban case study is 5.0 µg/dL. These 90th percentile values for the case study populations are larger than the 90th percentile value of 3.9 µg/dL reported by NHANES for all children in 2003–2004. It is noted that ambient air levels reflected in the urban case studies are likely to differ from those underlying the NHANES data.

Table 1. Summary of blood Pb estimates for median total blood Pb.[40,41]

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).

[40] As noted in footnote 39, median blood Pb levels generated for the three location-specific urban case studies and the general urban case study for the current conditions scenario are somewhat larger than the median value from NHANES for 2003–2004.

[41] As recognized in section III.B.2.d above, to simulate air concentrations associated with the current NAAQS, a proportional roll-up of concentrations from those for current conditions was performed for the location-specific urban case studies. This was not necessary for the primary Pb smelter case study in which air concentrations currently exceed the current standard.

Table 2. Summary of blood Pb level estimates for 95th percentile total blood Pb.[42] [43]

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).

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[42] As noted in footnote 39, 90th percentile blood Pb levels generated for the three location specific urban case studies and the general urban case study for the current conditions scenario are larger than the 90th percentile value from NHANES for 2003–2004. Note, 95th percentile values were not available for the NHANES 2003–2004 dataset, preventing a direct comparison to modeled estimates presented in Table 2. However, in 2001–2002, the 95th percentile value was 5.8 μg/dL (see footnote 7).

[43] As recognized in section III.B.2.d above, to simulate air concentrations associated with the current NAAQS, a proportional roll-up of concentrations from those for current conditions was performed for the location-specific urban case studies. This was not necessary for the primary Pb smelter case study in which air concentrations currently exceed the current standard.

b. IQ Loss Estimates

This section presents IQ loss estimates in Tables 3 through 6. These IQ loss estimates need to be understood in the context of the broader and more comprehensive and detailed presentation provided Risk Assessment Report (USEPA, 2007b). The tables presented here include three types of risk estimates:

• *Estimates of IQ loss for all air quality scenarios (based on total Pb exposure):* Tables 3 and 4 present IQ loss estimates for total Pb exposure for each of the air quality scenarios simulated for each case study. Table 3 presents estimates for the population median and Table 4 presents results for the 95th population percentile. These results included both median and 95th population percentile estimates. To reflect the variation in estimates derived from the four different concentration-response functions included in the analysis, three categories of estimates are considered including (a) IQ loss estimates generated using the low concentration-response function (the model that generated the lowest IQ loss estimates), (b) estimates generated using the log-linear with low-exposure linearization (LLL) model, and (c) IQ loss estimates generated using the high concentration-response function (the model that generated the highest IQ loss estimates). For reasons described above, estimates generated using the LLL model have been given emphasis in the summary below.

• *Estimates of IQ loss under the current NAAQS air quality scenario (with pathway apportionment):* Tables 5 and 6 present IQ loss estimates 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 5 presents estimates of the total Pb-related IQ loss for the population median and Table 6 presents estimates for the 95th population percentile. Both of these tables present total IQ loss estimates for (a) total Pb exposure (including both policy-relevant pathways and background sources) and (b) 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 for each location-specific urban case study projected to have total Pb-related IQ loss greater than one point are summarized in Table 7, and similar estimates for IQ loss greater than 7 points are summarized in Table 8. Also presented are the changes in incidence of the current NAAQS and alternative NAAQS scenarios compared to current conditions, with emphasis placed on estimates generated using the LLL concentration-response function. 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.

The IQ loss results presented in Tables 3 through 8 need to be understood in the context of the broader and more comprehensive and detailed presentation provided in the Risk Assessment Report. Observations regarding the IQ loss results presented in Tables 3 through 8 are presented in Section 4.4 of the Staff Paper.

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 typically around a factor of three. However, the relative (proportional) change in IQ loss across air quality scenarios (i.e., the pattern of IQ loss reduction across air quality scenarios for the same case study) is fairly consistent across all four models. This suggests uncertainty in estimates of absolute IQ loss for a median or 95th 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 and this is reflected in the observations presented in Section 4.4 of the Staff Paper. As with the blood Pb estimates, 5 percent of the child study population at each case study location is estimated to have IQ loss above the 95th percentile estimates presented here, however, due to technical limitations of our modeling tools, it is not feasible at this point to reasonably predict the distribution of IQ loss levels for that top 5 percent.

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Table 3. Summary of risk estimates for medians of total-exposure risk distributions. [44]

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 - These columns present the estimates of total IQ loss resulting from total Pb exposure (policy-relevant plus background). 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.

[44] As recognized in section III.B.2.d above, to simulate air concentrations associated with the current NAAQS, a proportional roll-up of concentrations from those for current conditions was performed for the location-specific urban case studies. This was not necessary for the primary Pb smelter case study in which air concentrations currently exceed the current standard.

Table 4. Summary of risk estimates for 95th percentile of total-exposure risk distributions.[45]

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 - These columns present the estimates of total IQ loss resulting from total Pb exposure (policy-relevant plus background). 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.

[45] As recognized in section III.B.2.d above, to simulate air concentrations associated with the current NAAQS, a proportional roll-up of concentrations from those for current conditions was performed for the location-specific urban case studies. This was not necessary for the primary Pb smelter case study in which air concentrations currently exceed the current standard.

Table 5. Median IQ loss estimates for the current NAAQS scenario.[46]

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 Risk Assessment Report, 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 6. 95th percentile IQ loss estimates for the current NAAQS scenario[47]

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	c	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 Risk Assessment Report, 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).

[46] As recognized in section III.B.2.d above, to simulate air concentrations associated with the current NAAQS, a proportional roll-up of concentrations from those for current conditions was performed for the location-specific urban case studies. This was not necessary for the primary Pb smelter case study in which air concentrations currently exceed the current standard.

[47] As recognized in section III.B.2.d above, to simulate air concentrations associated with the current NAAQS, a proportional roll-up of concentrations from those for current conditions was performed for the location-specific urban case studies. This was not necessary for the primary Pb smelter case study in which air concentrations currently exceed the current standard.

Table 7. Incidence of children with >1 point Pb-related IQ loss. [48]

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		236,257	
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		9,526		8,515	
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		170,474	
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	-10,929	158,234	-12,240

[48] As recognized in section III.B.2.d above, to simulate air concentrations associated with the current NAAQS, a proportional roll-up of concentrations from those for current conditions was performed for the location-specific urban case studies. This was not necessary for the primary Pb smelter case study in which air concentrations currently exceed the current standard.

Table 8. Incidence of children with >7 points Pb-related IQ loss.[49]

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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[49] As recognized in section III.B.2.d above, to simulate air concentrations associated with the current NAAQS, a proportional roll-up of concentrations from those for current conditions was performed for the location-specific urban case studies. This was not necessary for the primary Pb smelter case study in which air concentrations currently exceed the current standard.

C. Considerations in Review of the Standard

This section presents an integrative synthesis of information in the Criteria Document together with EPA analyses and evaluations. EPA notes that the final decision on retaining or revising the current primary Pb standard is a public health policy judgment to be made by the Administrator. The Administrator's final decision will 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. These judgments will be informed by 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 (section III.C.1), including both the basis for derivation of the current standard and considerations and conclusions from the 1990 Staff Paper (USEPA, 1990b). This is followed by a summary of the general approach for this current review (section III.C.2). Considerations with regard to the adequacy of the current standard are discussed in section III.C.3, with evidence and exposure-risk-based considerations in subsections III.C.3.a and b, respectively, followed by a summary of CASAC advice and recommendations (section III.C.3.c) and, lastly, solicitation of comment on the broad range of policy options (section III.C.3.d). Considerations with regard to elements of alternative standards—indicator, averaging time and form, and level—are discussed in sections III.C.4.a., III.C.4.b, and III.C.4.c, respectively. The discussion with regard to level includes subsections on evidence and exposure-risk-based considerations (sections III.C.4.a and b), followed by a summary of CASAC advice and recommendations (section III.C.4.c) and, lastly, solicitation of comment on the broad range of policy options (section III.C.4.d).

1. Background on the Current Standard

a. Basis for Setting the Current Standard

The current primary standard is set at a level of 1.5 $\mu g/m^3$, measured as Pb-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.

i. Level

EPA's objective in selecting the level of the current standard was ''to estimate the concentration of Pb 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 below.

The assessment of the science that was presented in the 1977 Criteria Document (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).

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 Criteria Document reported a threshold for inhibition of this enzyme in children at 10 µg Pb/dL. The 1977 Criteria Document 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. [50]

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 GSD 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).

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 μg/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.

In determining the air Pb level consistent with an air contribution of 3 μg Pb/dL, EPA reviewed studies assessed in the 1977 Criteria Document 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 μg/m^3 air Pb concentration), and is recognized to fall within the range of values reported in the 1977 Criteria Document. On the basis of this 2-to-1 relationship, EPA concluded that the ambient air standard should be 1.5 μg Pb/m^3 (43 FR 46254).

In consideration of the appropriate margin of safety during the development of the current NAAQS, EPA identified the following factors: (1) The 1977 Criteria Document 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

[50] The CDC subsequently revised their advisory level for children's blood Pb to 25 μg/dL in 1985, and to 10 μg/dL 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 in children had been identified, and emphasizing the importance of preventative measures (CDC, 2005a). Recently, CDC's Advisory Committee on Childhood Lead Poisoning Prevention noted the 2005 CDC statements and reported on a review of the clinical interpretation and management of blood Pb levels below 10 μg/dL (ACCLPP, 2007). More details on this level are provided in Section III.A.1.

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).

ii. 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).

b. 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 Criteria Document 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.[51]

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. 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 1990 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.''

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

(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 Pb 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 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 at primary and secondary Pb smelters, as well as other Pb sources.

2. 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, EPA is considering an approach in this review like that used in the Staff Paper. As discussed below, this approach 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.

This approach is 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 are informed by air quality and related analyses, quantitative exposure and risk assessments when possible, and qualitative assessment of impacts that could not be quantified.

In conducting this assessment, EPA is aware of the dramatic reductions in air Pb emissions in the U.S. in recent decades.[52] 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 the Clean Air Act Amendments of 1990, which amended 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.[53] EPA 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 evaluation will consider the status of Pb as a criteria pollutant and assesses whether revocation of the standard is an appropriate option for the Administrator to consider.

As discussed below, in conducting this evaluation, EPA will take into account both evidence-based and quantitative exposure- and risk-based considerations. To the extent that the available information suggests that revision of the current standard may be appropriate to consider, EPA will also evaluate the currently available information to determine the extent to which it supports consideration of a revised standard. In this evaluation, EPA will consider the specific elements of the standard to identify options (in terms of an indicator, averaging time, level, and form) for consideration 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.

To help inform the Agency's consideration of the quantitative exposure and risk assessments, summarized above in section III.B, EPA solicits comment on the appropriate weight to be placed on the results from these assessments in evaluating the adequacy of the current primary standard and in considering alternative standards. Specifically, we solicit comment on a number of aspects of the design of the assessments and interpretation of the assessment results, including in particular: (1) The appropriateness of rolling up ambient Pb concentrations to simulate just meeting the current standard for areas in which current concentrations are well below the level of the current standard; [54] (2) the use of a proportional

[52] Detailed information on air Pb emissions, and temporal trends in emissions since 1980 is provided in Section 2.2 of the Staff Paper.

[53] The use of Pb paint in new houses has declined substantially over the 20th century. For example ''an estimated 68% of U.S. homes built before 1940 have Pb hazards, as do 43% of those built during 1940–1959 and 8% of those built during 1960–1977'' (ACCLPP, 2007). We are uncertain of the implications of these reductions for ambient air.

[54] We have not in the past used such an approach in developing risk assessments for other NAAQS reviews since other risk assessments (i.e., for ozone and PM) included a number of areas that did not meet the current NAAQS such that rolling up ambient pollutant concentrations was not needed to characterize risks associated with just meeting the current standard.

method to roll-up and roll-down Pb concentrations to simulate just meeting the current and alternative standards; [55] (3) the categorization and apportionment of policy-relevant exposure pathways and policy-relevant background, particularly with regard to exposures related to historically deposited Pb from leaded gasoline and from Pb paint; and (4) the weight to be given to risk estimates derived using various concentration-response functions. More broadly, we also solicit comment on the approach of considering exposures and risks resulting from the ingestion of historically emitted Pb that may now be present in indoor dust and outdoor soil (e.g., that associated with past use of Pb in gasoline or Pb paint) impacted by ambient air Pb as being policy-relevant for the purpose of setting a NAAQS.

3. Adequacy of the Current Standard

In considering the adequacy of the current standard, EPA will first consider 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 HAP 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.

EPA notes that CASAC specifically examined several scientific issues and related public health (and public welfare) policy issues that the CASAC Lead Review Panel [56] 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 of the Staff Paper), 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, the CASAC Lead Review Panel judged that the answer to each of these questions was ''no,'' leading the Panel to conclude that ''the existing state of science is consistent with continuing to list ambient lead as a criteria pollutant for which fully-protection 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 of the Staff Paper), CASAC strongly reiterated its opposition to any considered delisting of Pb, and expressed its unanimous support for maintaining fully-protective NAAQS (id., p. 2). The EPA seeks comment and supporting information on the issue of whether it would be appropriate for EPA to determine that emissions of Pb no longer contribute to air pollution that may reasonably be anticipated to endanger public heath. EPA also solicits comment and supporting information on the extent to which reductions in the ambient air Pb standard would benefit public health.

In considering the adequacy of the current standard, EPA will consider the available evidence and quantitative exposure- and risk-based information, summarized below.

a. Evidence-Based Considerations

In considering the broad array of health effects evidence assessed in the Criteria Document with respect to the adequacy of the current standard, EPA will focus on those health endpoints associated with the Pb exposure and blood levels most pertinent to ambient exposures. Additionally, we will 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). The prenatal period and early childhood are periods of increased susceptibility to Pb exposures, with evidence of adverse effects on the developing nervous system that generally appear to persist into later childhood and adolescence (CD, Section 6.2).[57] Thus, while the sensitivity of the elderly and other particular subgroups is recognized, 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. 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. Although CDC recognized a level of 30 μg/dL blood Pb as warranting individual intervention in 1978 when the Pb NAAQS was set, in 2005 they recognized 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, 1991; CDC, 2005).

The Criteria Document describes current evidence regarding the occurrence of a variety of adverse health

[55] There are other methods that might be used.

[56] 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.

[57] For example, the following statement is made in the Criteria Document ''Negative Pb impacts on neurocognitive ability and other neurobehavioral outcomes are robust in most recent studies even after adjustment for numerous potentially confounding factors (including quality of care giving, parental intelligence, and socioeconomic status). These effects generally appear to persist into adolescence and young adulthood.'' (CD, p.E–9)

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 (CD, Sections 8.4 and 8.5).[58] With regard to the evidence of effects on the developing nervous system at these low levels, EPA notes, 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 III (conducted from 1988–1994), 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).

As when the standard was set in 1978, EPA recognizes that there remain today contributions to blood Pb levels from nonair sources. Estimating contributions from nonair sources are 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 Criteria Document 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 different pathways (air-related and other) contributing to indoor dust Pb. As recognized in Section III.A. above (including footnote 13), some studies have found that dietary intake of Pb may be a predominant source of Pb exposure *among adults*, greater than consumption of water and beverages or inhalation (CD, p. 3–43). The exposure assessment for children performed for this review has employed available data and methods to develop estimates intended to inform a characterization of these pathways.

Consistent with reductions in air Pb concentrations[59] which contribute to blood Pb, nonair contributions have also been reduced. For example, the use of Pb paint in new houses has declined substantially over the 20th century, such that ''an estimated 68% of U.S. homes built before 1940 have Pb hazards, as do 43% of those built during 1940–1959 and 8% of those built during 1960–1977'' (ACCLPP, 2007). Additionally, 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 Criteria Document identifies a drop in dietary Pb intake by 2 to 5 year olds of 96% between the early 1980s and mid 1990s. The 1977 Criteria Document 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 Criteria Document 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. Specific measurements of Pb in blood that derived from Pb that had been in the air are not available. Rather, estimates are available for the relationship between Pb concentrations in air and Pb levels in blood, developed from populations in differing Pb exposure circumstances, which inform this issue. 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 higher[60] (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 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).[61] 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 1:5 with some higher estimates (see footnote 60) and some lower estimates (down to 1:1). EPA's consideration of this issue in 1986 indicated that ratios which consider both inhalation and ingestion pathways are ''necessarily higher than those estimates for inhaled air lead alone'' (USEPA, 1986a, p. 11–106). We solicit comment on data or studies that may help inform our understanding of this important parameter.

Based on this information, the Staff Paper concluded that young children remain the sensitive population of primary focus in this review, there is now no recognized safe level of Pb in

[58] For context, it is noted that the 2001–2004 median blood level for children aged 1–5 of all races and ethnic groups is 1.6 µg/dL, the median for the subset living below the poverty level is 2.3 µg/dL and 90th percentile values for these two groups are 4.0 µg/dL and 5.4 µg/dL, respectively. Similarly, the 2001–2004 median blood level for black, non-hispanic children aged 1–5 is 2.5 µg/dL, while the median level for the subset of that group living below the poverty level is 2.9 µg/dL and the median level for the subset living in a household with income more than 200% of the poverty level is 1.9 µg/dL. Associated 90th percentile values for 2001–2004 are 6.4 µg/dL (for black, non-hispanic children aged 1–5), 7.7 µg/dL (for the subset of that group living below the poverty level) and 4.1 µg/dL (for the subset living in a household with income more than 200% of the poverty level). (*http://www.epa.gov/envirohealth/children/body_burdens/b1-table.htm*—then click on ''Download a universal spreadsheet file of the Body Burdens data tables'').

[59] Air Pb concentrations nationally are estimated to have declined more than 90% since the early 1980s.

[60] For example, adjusted ratios from Brunekreef (1984, Table 1) ranged up to 1:8.5 and unadjusted ratios extended above 1:10.

[61] The CASAC Panel stated ''The Schwartz and Picher 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 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.'' (Henderson, 2007a, page D–2 to D–3).

children's blood, and studies appear to show adverse effects at mean concurrent blood Pb levels as low as 2 ug/dL (CD, pp. 6–31 to 6–32; Lanphear et al., 2000). 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. Using the framework employed in setting the standard in 1978, the more recently available evidence and more recently available estimates may suggest a level for the standard that is lower by an order of magnitude or more.

b. Exposure- and Risk-Based Considerations

In addition to the evidence-based considerations, EPA will also consider exposures and health risks estimated to occur upon 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 above, young children are the sensitive population of primary focus in this review. 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 were quantitatively estimated, EPA recognizes 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. As noted in section III.B above, the risk assessment results focus predominantly on risk estimates derived using the log-linear with low-exposure linearization (LLL) concentration-response function, with the range associated with the other three functions also being noted.

As noted in the Criteria Document, 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, Sections 8.6.1 and 8.6.2; Bellinger, 2004; Needleman *et al.*, 1982; Weiss, 1988; Weiss, 1990)). 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, 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). 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 this section, risk estimates for the median and for an upper percentile, the 95th are discussed. In setting the standard in 1978, EPA 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. Similarly, in their advice to EPA 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, EPA and CASAC also recognize uncertainties in the risk estimates at the edges of the distribution and consequently the 95th percentile is reported as the estimate of the high end of the risk distribution (Henderson, 2007b, p. 3). In so doing, however, EPA notes 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 when set in 1978 with regard to the 99.5th percentile.

In addition to estimating IQ loss associated with the combined exposure to Pb from all exposure pathways, EPA 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. There are various limitations associated with our modeling tools that affected the 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.[62] Accordingly, this notice presents these two sets of estimates as providing a range of interest, with regard to policy-relevant Pb, for 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. 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, indicating that most of the monitored areas of the country are well below the standard. Review of the current monitoring network in light of current information on Pb sources and emissions, however, indicated that monitors are not located near many of the larger sources. Therefore, the assessment may be underestimating Pb concentrations.

Using the current monitoring data, EPA estimated exposure and risk associated with *current conditions* 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.[63] Two current conditions scenarios were assessed for the general urban case study, one based on the 95th percentile of levels in large urban areas (0.87 $\mu g/m^3$, maximum quarterly mean) and one based on mean levels in such

[62] Comparisons of blood Pb levels estimated for individual case study populations (from all exposure sources in current conditions scenarios) to national population values from NHANES are noted in footnote 39 in Section III.B.3.a.

[63] Comparisons of median and 90th percentile blood Pb levels estimated for individual case study populations (from all exposure sources in current conditions scenarios) to national population values from NHANES are noted in footnote 39 in Section III.B.3.a. That comparison suggests that modeled estimates generated for the location-specific urban case studies for both population percentiles are somewhat larger than values cited in NHANES (for 2003–2004). However, as mentioned earlier, factors related to Pb exposure, including ambient air levels, are likely to differ for the urban case study populations compared with the national population underlying NHANES.

areas (0.14 μg/m^3, maximum quarterly. Levels in the three location-specific case studies ranged from 0.09 to 0.35 μg/m^3, in terms of maximum quarterly average. For the general urban case study, which is a simplified representation of urban areas, 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. 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. 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 areas, 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.[64] Median IQ loss associated with policy-relevant Pb (LLL function) is estimated to fall between 0.6 to 2.9 points IQ loss. 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. 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, Tables 5–9 and 5–10).

In order to consider exposure and risk associated with the *current standard,* EPA developed estimates for a case study based on air quality projected to just meet the standard in a location of the country where air concentrations do not meet the current standard (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 may exercise previously used capabilities as primary smelters) or by the congregation of multiple Pb sources in adjacent locations. We have simulated this scenario (increased Pb concentrations to just meet the current standard) in a general urban case study and 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, we note 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 Chicago and Los Angeles study areas for which current conditions are estimated to be at or below one tenth of the current NAAQS.

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 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 categories 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. 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.

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). 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). 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.

Further, in comparing current NAAQS scenario estimates to current conditions estimates for the three location-specific urban case studies, the estimated difference in total Pb-related IQ loss for the median is about 0.5 to 1.4 points using the LLL concentration-response function and a similar magnitude of difference is estimated for the 95th percentile. 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.

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 conditions simulated to just meet the current standard,[65] indicate levels of IQ loss that some may reasonably consider to be 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

[64] 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 μg/m^3), 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.

[65] As recognized in section III.B.2.d above, to simulate air concentrations associated with the current NAAQS, a proportional roll-up of concentrations from those for current conditions was performed for the location-specific urban case studies. This was not necessary for the primary Pb smelter case study in which air concentrations currently exceed the current standard.

current standard. Estimates of the additional number of children with IQ loss greater than one point (based on the LLL concentration-response function) in these three study areas with the current NAAQS scenario compared to current conditions range from 100 to 6,000 across the three locations. The corresponding estimates for the additional number of children with IQ loss greater than seven points, for the current NAAQS as compared to the current conditions scenario range from 600 to 35,000. These latter values for the change in incidence of children with greater than seven 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.

While the risk assessment has quantified risks associated with IQ impacts in childhood, 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 (CD, pp. 8–29 to 8–30). Additional impacts at low levels of childhood exposure 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.

c. CASAC Advice and Recommendations

Beyond the evidence- and risk/exposure-based information discussed above, in considering the adequacy of the current standard, EPA will also consider the advice and recommendations of CASAC, based on their review of the Criteria Document and the drafts of the Staff Paper and the related technical support document, as well as comments from the public on drafts of the Staff Paper and related technical support document.[66] 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 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 Criteria Document, of health effects occurring at dramatically lower blood Pb levels than those indicated by the evidence available when the standard was set.

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).

d. Policy Options

In considering the adequacy of the current standard, EPA first notes the dramatic changes in the basic patterns of air Pb emissions in the U.S. since the standard was set, reflecting the phase-out of Pb in gasoline, as well as changes to the CAA related to the inclusion of Pb compounds on the list of HAPs and associated requirements for technology- and risk-based standards for major stationary sources. We are aware that questions have been raised about the appropriateness of retaining Pb on the list of criteria pollutants and/or maintaining a NAAQS for Pb in light of these changed circumstances. We take note of the views of CASAC, summarized above, and the conclusions and recommendations in the OAQPS Staff Paper on these questions, which do not support delisting Pb or revoking the Pb NAAQS. We recognize, however, that there may be differing views on interpreting or weighing the available information. Thus, EPA solicits comment related to the questions of delisting and revocation. The EPA also solicits comment on whether the broad range of current multimedia Federal and State Pb control programs, summarized above in section II.C, are sufficient to provide appropriate public health protection in lieu of a Pb NAAQS.

In further considering the adequacy of the current standard, EPA will focus on the body of available evidence (summarized above in section III.A and discussed in the Criteria Document) that is much expanded from that available when the current standard was set. The presentation of the evidence in the Criteria Document describes 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. We recognize that the current health effects evidence together with findings from the exposure and risk assessments (summarized above in section III.B), like the information available at the time the standard was set, supports the conclusion that air-related Pb exposure pathways (by inhalation and ingestion) contribute to blood Pb levels in young children. Furthermore, we take note of the information that suggests that the air-to-blood relationship (i.e., the air-to-blood ratio), is likely larger, with regard to increase in blood Pb per unit air concentration, when air inhalation and ingestion are considered than that estimated when the standard was set using only inhalation and may be several times larger. EPA recognizes there is uncertainty in estimates of this relationship and solicits comment on on ratios supported by the current evidence.

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 some may consider to be significant from a public health perspective.[67] Further, although the current monitoring data indicate few areas with airborne Pb near or just exceeding the current standard, we recognize significant limitations with the current monitoring network and thus the potential that the prevalence of such levels of Pb

[66] All written comments submitted to the Agency will be available in the docket for this rulemaking, as will be transcripts of the public meeting held in conjunction with CASAC's review of the first draft of the Staff Paper and the first draft of the related technical support document, and of draft and final versions of the Criteria Document.

[67] As recognized in section III.B.2.d above, to simulate air concentrations associated with the current NAAQS, a proportional roll-up of concentrations from those for current conditions was performed for the location-specific urban case studies. This was not necessary for the primary Pb smelter case study in which air concentrations currently exceed the current standard.

concentrations may be underestimated by currently available data.

As summarized above, CASAC conclusions and recommendations and recommendations presented in the OAQPS Staff Paper reflect the view that the current standard is not adequate and support consideration of a revised standard to provide an adequate margin of safety for sensitive groups. Taking these views into account, we recognize that one approach is to consider a revised standard. We also recognize that there may be differing interpretations of the available information. Thus, EPA solicits comment on delisting, revocation, and the adequacy of the current standard and the rationale upon which such views are based.

4. 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 standard. In considering revisions to the current primary Pb standard, as discussed in the following sections, EPA will 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 with an adequate margin of safety.

a. Indicator

The indicator for the current standard is Pb–TSP. When the standard was set, the Agency considered identifying Pb in particles less than or equal to 10 μm in diameter (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.

More recently, in the 1990 Staff Paper, this issue was reconsidered in light of information regarding limitations of the high-volume sampler used for the Pb–TSP measurements and the continued use of total suspended particulate matter (TSP) as the indicator was supported by OAQPS staff (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 the current review, CASAC has recommended that EPA consider a change in the indicator to utilize low-volume PM_{10} sampling (Henderson, 2007a, b). In so doing, CASAC recognized that a scaling of the NAAQS level would be needed to accommodate the loss of very large coarse-mode Pb particles and concurrent Pb–PM_{10} and Pb–TSP sampling would be needed to inform development of scaling factors. 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 were regarding 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, which the Panel indicated 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 shorter averaging time that they recommend (Henderson, 2007b).

In considering the appropriate indicator, EPA takes note of and solicits comment on 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 current information does not support the derivation of a single scaling factor, which might be used to relate a level for Pb–TSP to a monitoring result using Pb–PM_{10} on a national scale. The EPA 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.

To the extent that Pb–PM_{10} exhibits less spatial variability and that a ''crosswalk'' can be developed between a level in terms of Pb–TSP, EPA recognizes that it is appropriate to 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.

The EPA will consider several options that might improve the available database and facilitate such a move in the future, while retaining Pb–TSP as the indicator for the NAAQS at this time, consistent with the recommendations in the Staff Paper. For example, we 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 an 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 were intended purely for purposes of illustrating the types of options the Agency might consider. Specific details of any options would need to be supported by appropriate data analyses. We solicit information and comments that would help inform such analyses and the Agency's views on the indicator for the primary Pb NAAQS.

b. Averaging Time and Form

The basis for the averaging time of the current standard 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 μg/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 Pb 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 above, the currently available health effects evidence [68] 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, EPA recognizes that today ''there is no level of Pb exposure that can yet be identified, with condfidence, as clearly not being associated with some risk of deleterious health effects'' (CD, p. 8–63). 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 Pb 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 Criteria Document and the risk assessment indicate that ingestion of dust can be a 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. As stated in the Criteria Document, ''given the large amount of time people spend indoors, exposure to Pb in dusts and indoor air can be significant'' (CD, p. 3–27). The Criteria Document further describes studies that evaluated the influence of dust Pb exposure on children's blood Pb: ''Using a structural equation model, Lanphear and Roghmann (1997) also found the exposure pathway most influential on blood Pb was interior dust Pb loading, directly or through its influence on hand Pb. Both soil and paint Pb influenced interior dust Pb; with the influence of paint Pb greater than that of soil Pb. Interior dust Pb loading also showed the strongest influence on blood Pb in a pooled multivariate regression analysis (Lanphear et al., 1998).'' (CD, p. 4–134).

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, EPA recognizes that there is also support for consideration of an averaging time shorter than a calendar quarter.

When the standard was set in 1978, 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 Pb 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. In this review, based on data from 2003–2005, the air quality analysis in Chapter 2 of the Staff Paper 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 three-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. 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, EPA will take into account analyses using air quality data for 2003–2005 that characterize maximum quarterly average and various monthly statistics for each year across the three year Pb-TSP dataset and also across the 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

[68] The differing evidence and associated strength of the evidence for these different effects is described in detail in the Criteria Document.

NAAQS subsequent to the promulgation of the Pb NAAQS. 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 was 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 Chapter 2 of the Staff Paper).

The analyses described in Chapter 2 of the Staff Paper 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.

In their advice to the Agency, CASAC has recommended that consideration be given to changing from a calendar quarter to a monthly averaging time (Henderson, 2007a, b). In making that recommendation, CASAC emphasizes 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, 2007b).

With regard to form of the standard, CASAC stated that one could ''consider having the lead standards based on the second highest monthly average, a form that appears to correlated well with using the maximum quarterly value'', while also indicating that ''the most protective form would be the highest monthly average in a year.''

The following observations support consideration of a monthly averaging time: (1) The health evidence indicates that very short exposures can lead to increases in blood Pb Pb 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 as prenatally and in developing infants. EPA also recognizes the limited available 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.

Based on the information and air quality analyses discussed above, EPA is requesting comment on a range of options, including the recommendations in the Staff Paper that include changing the averaging time to monthly, with a form of maximum or second maximum, as well as retaining the quarterly averaging time. The EPA is also requesting comment on, the options of changing the form to apply to a three-year period as well as retaining a single-year period. We solicit comments on these ranges of averaging times and forms as well as views and related rationales that might support alternative options.

c. Level

At this time, the Agency is interested in soliciting comment on a wide range of possible options for consideration when making a proposed decision on the level of the primary Pb NAAQS. These policy options range from lowering the standard, to the levels recommended by CASAC and the OAQPS Staff paper or lower, as well as on other alternative levels, up to and including the current level, and the rationale upon which such views are based.

i. Evidence-Based Considerations

The EPA recognizes that there are several aspects to the body of epidemiological evidence available in this review that complicate efforts to translate the evidence into the basis for selecting an appropriate level for an ambient air quality standard. As an initial matter, as summarized above and discussed in greater depth in the Criteria Document (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, not ambient air concentrations. 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).

In considering how this evidence can help inform the selection of the level of the standard, EPA will consider how the framework applied in the establishment of the standard may be applied to the much expanded body of evidence that is now available. This consideration builds upon the evidence-based considerations of the adequacy of the current standard, discussed above in Section III.C.3.a.

As noted above, this review focuses on young children as the key sensitive population for Pb exposures, the same population identified in 1978. In this sensitive population, 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. Some studies indicate 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 findings 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).[69] Further, the current evidence does not indicate a threshold for the more sensitive health endpoints such as adverse effects on the

[69] These findings include significant associations in the study sample subsets of children with blood Pb levels less than 10 μg/dL, less than 7.5 μg/dL and less than 5 μg/dL. A positive, but not statistically significant association, was observed in the less than 2.5 μg/dL subset, although the effect estimate for this subset was largest among all the subsets. The lack of statistical significance for this subset may be due to the smaller sample size of this subset which would lead to lower statistical power.

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 at that time, 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 substantially.

As when the standard was set in 1978, EPA recognizes that there remain today contributions to blood Pb levels from nonair sources. As discussed above, 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). The value of 1.4 µg/dL was the mean blood Pb level derived from a simulation of current nonair exposures using the IEUBK model (Henderson, 2007a, pp. F–60 to F–61). These current estimates are roughly an order of magnitude lower than the value of 12 µg/dL that was used in setting the 1978 standard.

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 precise 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 Criteria Document typically yield estimates in the range of 1:3 to 1:5, with some as high as 1:10 or higher (USEPA, 1986a; Brunekreef, 1984). Findings in a more recent study identified in the Criteria Document 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). These ratios are appreciably higher than the ratio of 1:2 that was used in setting the 1978 standard.

A standard setting approach being considered is to apply the framework relied upon in setting the standard in 1978 to the currently available information. In applying that framework, however, EPA recognizes that today ''there is no level of Pb exposure that can yet be identified, with confidence, as clearly not being associated with some risk of deleterious health effects'' (CD, p. 8–63). However, there is increasing uncertainty with regard to the magnitude and type of effects at levels below 5 µg/dL [70]. This is in contrast to the situation in 1978 when the Agency judged that the maximum safe blood Pb level (geometric mean) for a population of young children was 15 µg/dL based on its conclusion that the maximum safe blood Pb level of an individual child was 30 µg/dL. [71]

In illustrating the application of the 1978 framework, two blood Pb levels are used here for illustrative purposes. A level of 2 µg/dL was used because it represents some of the lowest population levels associated with adverse effect in the current evidence (e.g., CD, p. E–9; Lanphear et al., 2000). In addition, a level of 5 µg/dL has been used. This level has been associated with adverse health effects with a higher degree of certainty in the published literature, and is a level where cognitive deficits were identified with statistical significance (Lanphear et al., 2000).

Using a blood Pb target of 2 µg/dL as a substitute for the 1978 target of 15 µg/dL for the child population geometric mean, then subtracting 1 to 1.4 µg/dL for background, yields 0.6 to 1 µg/dL as a target for the air contribution to blood Pb. Dividing the air target by 5, consistent with currently available information on the ratio of air Pb to blood Pb, yields a potential standard level of 0.1 to 0.2 µg/m^3. Alternatively, using the same approach substituting 5 µg/dL for the child population geometric mean and subtracting 1 to 1.4 µg/dL for background, yields 3.6 to 4 µg/dL as a target for the air contribution to blood Pb. Dividing the air target by 5, consistent with currently available information on the ratio of air Pb to blood Pb, yields a level of 0.7 to 0.8 µg/m^3. Similarly, substitution of other blood Pb targets would result in still other levels.

In light of the current CDC blood Pb ''level of concern'' of 10 µg/dL, some might consider a blood Pb value of 10 µg/dL as a target blood Pb value for this calculation to derive a level for the primary standard. EPA notes, however, that the CDC does not consider this level of concern as a safe blood Pb level or one without evidence of adverse effects (CDC, 2005a). Rather, it is used by CDC to identify children with elevated blood Pb levels for follow-up activities [72] at the individual level and to trigger communitywide prevention activities (CDC, 2005a). The level of concern has been frequently misinterpreted as a definitive toxicologic threshold (CDC, 2005a). As summarized in Section III.A and above, and as described in detail in the Criteria Document, various adverse effects have been associated with children's blood Pb levels below 10 µg/dL. For example, the Criteria Document states that the currently available toxicologic and epidemiologic information ''includes assessment of new evidence substantiating risks of deleterious effects on certain health endpoints beng 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). Accordingly, EPA has not used a mean or an individual target blood Pb value of 10 µg/dL as the basis for an illustrative example of deriving a standard that is intended to protect public health with an adequate margin of safety. In recognition of differing views on this subject, however, we solicit comment on the appropriateness of using a mean or individual target blood Pb value of 10 µg/dL as the foundation for deriving a level for the primary Pb standard.

The above examples focus on the mean target blood Pb level for the sensitive population by way of illustrating application of the 1978 framework. The EPA solicits comment on mean target blood Pb levels as well as other factors that would be important in applying the 1978 framework. For example, the distribution of blood Pb levels within the sensitive population is an important aspect of the 1978 framework. When the standard was set in 1978, the Agency stated that the population mean, measured as the geometric mean, must be 15 µg/dL in order to ensure that 99.5 percent of children in the United States would

[70] As stated in the Criteria Document ''Some recent studies of Pb neurotoxicity in infants have observed effects at population average blood-Pb levels of only 1 or 2 µg/dL; and some cardiovascular, renal, and immune outcomes have been reported at blood-Pb levels below 5 µg/dL.'' (CD, p. E–16).

[71] More specifically, the 1978 target of 15 µg/dL 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 (43 FR 46247–49).

[72] Activities such as taking an environmental history, educating parents about Pb and conducting follow-up blood Pb monitoring were among those suggested for children with blood Pb levels greater than or equal to 10 µg/dL (CDC, 2005a). Recently, CDC's Advisory Committee on Childhood Lead Poisoning Prevention has also provided information and recommendations relevant to clinical management of children with blood Pb levels below 10 µg/dL (ACCLPP, 2007).

have a blood Pb level below 30 μg/dL, which was identified as the maximum safe blood Pb level for individual children based on the information available at that time (43 FR 46252). Target values for the mean of the population necessarily imply higher values for individuals associated with the upper percentiles of the blood Pb distribution. For example, the 2001–2002 NHANES information indicates that a geometric mean blood level of 1.7 μg/dL for children nationally, aged 1–5 years, is associated with a 95th percentile blood Pb level of 5.8 μg/dL (CDC, 2005b).

Additionally, the nonair (background) contribution to total blood Pb is an important input to the framework and we solicit comment on the definition and appropriate values for this parameter.[73] In the assessment presented in this notice, contributions attributed to ''recent air'' and to ''recent plus past air'' may include some Pb from the historic use of Pb in paint and gasoline and other sources.

Further, there are a range of estimates for the air-to-blood ratio that include estimates higher than that used in 1978 when the standard was set. We solicit comment and supporting information regarding the air-to-blood ratio and differences in the available estimates. All of these factors are important in applying a framework such as that used in 1978, and we solicit comment, along with supporting information, on all of these factors.

Beyond the 1978 framework illustrated above, EPA recognizes a variety of approaches can be used in translating the current evidence to a level for the standard. With this notice, EPA solicits comment on the 1978 standard setting framework and on alternate approaches and the factors that are relevant to those approaches.

ii. Exposure- and Risk-Based Considerations

To inform 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, EPA will also consider 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 in Section III.B. As discussed above, the risk assessment conducted by EPA is based on exposures that have been estimated for children of less than 7 years of age in six case studies. The assessment estimated the risk of adverse neurocognitive effects in terms of IQ decrements associated with total and policy-relevant Pb exposures, including incidence of different levels of IQ loss in three of the six case studies. In so doing, EPA is mindful of the important uncertainties and limitations that are associated with the exposure and risk assessments. 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 Criteria Document.

EPA also recognizes important limitations in the design of, and data and methods employed in, the exposure and risk analyses. For example, the available monitoring data for Pb, relied upon for estimating current conditions for the urban case studies is quite limited, in that monitors are not located near some of the larger known Pb sources, which provides the potential for underestimation of current conditions, and there is uncertainty about the proximity of existing monitors to other Pb sources potentially influencing 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, EPA recognizes that there is not sufficient information available 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. As noted above, however, coordinated intensive efforts over the last 20 years have yielded a substantial decline in blood Pb levels in the United States. Recent NHANES data (2003–2004) yield blood lead level estimates for children age 1 to 5 years of 1.6 μg/dL (median) and 3.9 μg/dL (90th percentile). These median and 90th percentile national-level data are lower than modeled values generated for the three location-specific urban case studies current conditions scenarios (see footnote 39). It is noted, however, that the urban case studies and the NHANES study are likely to differ with regard to factors related to Pb exposure, including ambient air levels.

EPA also recognizes 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) may fall between the estimates for ''recent air'' and those for ''recent'' plus ''past air''. However, there are limitations associated with the indoor dust Pb models that affect our ability to discern differences in the recent air category among different alternate air quality scenarios and both categories may include Pb in soil and dust from the historical use of Pb in paint.

With these limitations in mind, EPA will 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 $\mu g/m^3$ as a maximum quarterly average or 0.17 to 0.56 $\mu g/m^3$ 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) may fall between the estimates for ''recent air'' and those for ''recent'' plus ''past air'', EPA will consider ranges reflecting those two categories. Further, as noted above, we will focus on risk estimates derived using the LLL (log-linear with low exposure linearization) concentration-response function.

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 Tables 5–9 and 5–10 of the Risk Assessment Report)—estimated to fall between the estimates for recent air (0.6–0.7 points) and those for recent plus past air (2.9 points)—appears to be of a magnitude in the range that CASAC considered to be highly significant from a public health perspective (e.g., a

[73] As noted above, in 2001 when establishing standards for lead-based paint hazards in most pre-1978 housing and child-occupied facilities (66 FR 1206), the Agency grappled with the uncertainties in what environmental levels of historic Pb in soil and dust (from the historical use of Pb in paint and gasoline) in which specific medium may cause blood Pb levels that are associated with adverse effects (see Section II.C).

population IQ loss of 1–2 points). Comparable estimates for the current conditions scenarios in the general urban case study are still more significant with estimates for the general urban case study ranging from 1.3–1.8 for recent air and 3.2–3.6 for recent plus past air. For the primary Pb smelter case study, in which air quality exceeds the current NAAQS, IQ loss reductions in the recent plus past air category associated with the alternate NAAQS levels of 0.2 and 0.5 μg/m^3 ranging from 4.0 to 4.9 points IQ loss for the subarea.

Focusing only on the recent air estimates, estimates of IQ loss (based on the LLL function) associated with policy-relevant Pb at the 95th percentile of population total IQ loss are greater than 1 point for all current conditions scenarios in all three urban case studies for which the lowest air Pb concentrations are 0.09 μg/m^3 maximum quarterly average, and 0.17 μg/m^3 maximum monthly average.

EPA will also 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 in the Staff Paper), looking 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.[74]

[74] 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.

EPA will also 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 in the Staff Paper). For the general urban case study, estimated reductions in median Pb-related IQ loss 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).

Based on estimated reductions in Pb-associated IQ loss discussed above, EPA observes 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.

Reductions in air Pb concentrations from current conditions to meet the lower alternative NAAQS (0.02 and 0.05 μg/m^3, maximum monthly mean) are 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.

As discussed above, CASAC considered a population IQ loss of 1–2 points to be highly significant from a public health perspective. 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 μg/m^3, 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). Based on estimated changes in risk across the population associated with the two lower alternative levels (as compared to current conditions), reductions in the number of children with total Pb-related IQ loss greater than 1 or greater than 7 are estimated to be 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, EPA 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. EPA will therefore consider a different approach in this review.

In considering these risk estimates, EPA is mindful of CASAC's recommendation regarding the public health significance of a population loss of 1 to 2 IQ points, the significant implications of potential shifts in the distribution of IQ for the exposed population, and other unquantified Pb-related health effects. 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, we recognize that some may consider reducing the NAAQS as important from a public health perspective (from air-related ambient Pb) relative to that afforded by the current standard.

In considering the public health significance of IQ loss beyond CASAC's recommendation on this issue, we note that some may consider 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. On the other hand, we also recognize that some may hold different views. Thus, the magnitude of IQ loss that could be allowed by a standard that 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 lowest alternative levels considered, EPA will focus on total IQ loss and on the contribution to total IQ loss from policy-relevant pathways. In so doing, we recognize that nonair contributions to total Pb-related IQ loss are estimated to reach and exceed an IQ loss of 1–2 points, and we also recognize that air Pb contributions are generally of a much smaller magnitude. Thus, we recognize that it may be appropriate to consider smaller estimates of IQ loss from air Pb contributions (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. EPA 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. Thus, to the extent that incremental exposure reductions achieved through lowering the NAAQS might contribute to incremental reductions in children's blood Pb and to associated reductions in health effects, 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. On the other hand, to the extent that the uncertainties and limitations in the exposure and risk assessments are judged to be so great as to prevent meaningful conclusions from being drawn for these low alternative standard levels, consideration of such low levels may not be appropriate.

If the policy goal for the Pb NAAQS was to be defined, for example, so as to provide protection that limited estimates of IQ loss from policy-relevant exposures to no more than 1–2 points IQ loss at the population-level, EPA notes 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.

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.

iii. CASAC Advice and Recommendations

Beyond the evidence- and risk/exposure-based information discussed above, EPA's consideration of the level for the NAAQS will also take into account the advice and recommendations of CASAC, based on their review of the Criteria Document and drafts of the Staff Paper and the related technical support document, as well as comments from the public on drafts of the Staff Paper and related technical support document. Public comments pertaining to the level of the standard recommended appreciable reductions 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 judgement 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). In their March 2007 letter conveying comments on the pilot phase risk assessment, CASAC based their recommendation as to level on consideration of the health effects evidence they provided initial recommendations that the level should be substantially lower, reflecting their view of the evidence itself.

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.'' The Agency anticipates further advice from CASAC with regard to level at the time of their review of this ANPR.

iv. Policy Options

In considering alternative levels of the primary Pb standard, EPA will consider the health effects evidence and the exposure and risk assessment, as well as the important uncertainties and limitations in the evidence and the assessment results. To help inform public health policy judgments, we specifically solicit comment on levels of IQ loss considered to be significant from a public health perspective. Additionally, we solicit comment on the magnitude of IQ loss associated with exposures to ambient Pb by the pathways categorized as ''recent air'' in the risk assessment described in this notice that are considered to be significant from a public health perspective. We also solicit comment on the approach of adopting a public health policy goal of limiting policy-relevant air exposure such that the incremental blood Pb level (and the associated resulting IQ loss) are below a specified level (e.g., to a magnitude of 0.5 or 1 $\mu g/dL$, or other alternative values).

The EPA takes note of the views of CASAC on these matters, summarized above, the conclusions and recommendations in the OAQPS Staff Paper,[75] and the views of public commenters. We also note other views,

[75] The OAQPS Staff Paper recommends consideration of a range of alternative standard levels from as high as 0.1 to 0.2 $\mu g/m^3$ down to the lower levels evaluated in the risk assessment of 0.02 to 0.05 $\mu g/m^3$.

including retaining the current standard level or a range of alternative levels that includes the upper end of the alternative standards considered in the risk assessment (i.e., 0.5 $\mu g/m^3$ as a maximum monthly average). The EPA recognizes that there may be differing interpretations of the available evidence, the public health significance of various changes in population IQ loss, and various aspects of the evidence and exposure and risk assessments, including important uncertainties and limitations associated with the evidence and assessments. Thus, EPA solicits comment on the range of alternative standard levels identified above, as well as on other alternative levels, up to and including the current level, and the rationale upon which such views are based.

IV. The Secondary Standard

This section presents information relevant to the review of the secondary Pb NAAQS, including information on the welfare effects associated with Pb exposures, results of the screening-level ecological risk assessment, and considerations related to evaluating the adequacy of the current standard and alternative standards that might be appropriate for the Administrator to consider.

A. Welfare Effects Information

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. A qualitative assessment of welfare effects evidence related to ambient Pb is summarized in this section, drawing from Chapter 6 of the Staff Paper. The presentation here first 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, EPA recognizes that there have been a number of uses of Pb, especially as an ingredient in automobile fuel but also in other products such as paint, lead-acid batteries, and some pesticides, which have significantly contributed to widespread increases in Pb concentrations in the environment, a portion of which remains today (e.g., CD, Chapters 2 and 3).

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 and sulfur dioxide 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. Many sites receiving Pb predominantly through such long-range transport have accumulated large amounts of Pb in soils (CD, p.l AX7–98). There is little evidence that terrestrial 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, AX7.1.4.2).

Terrestrial ecosystems remain primarily sinks for Pb but amounts retained in various soil layers vary based on forest type, climate, and litter cycling (CD, Section 7.1). 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. 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, AX7.1.4.1).

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 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. Ecological soil screening levels (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.

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 United States (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).

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.

B. Screening Level Ecological Risk Assessment

This section presents a brief summary of the screening-level ecological risk assessment conducted by EPA for this review. The 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). The following summary focuses on key features of the approach used in the assessment and presents only a brief summary of the results of the assessment. A complete presentation of results is provided in the pilot phase Risk Assessment Report (ICF, 2006) and summarized in Chapter 6 of the Staff Paper.

1. Design Aspects of Assessment and Associated Uncertainties

The screening level risk assessment involved several location-specific 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 location near a nonurban roadway. An additional case study for an ecologically vulnerable location was identified and described (ICF, 2006), but resource constraints have precluded risk analysis for this location.

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. 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 surface water and sediment monitoring locations across the United States for the potential for ecological impacts associated with atmospheric deposition of Pb. 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.

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. The hazard quotient (HQ) approach was then used to compare Pb media concentrations with ecological screening values. The 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 (USGS, 2004). 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 ecological screening values used in this assessment were developed from the Eco-SSLs methodology, EPA's recommended ambient water quality criteria, and sediment screening values developed by MacDonald and others (2000, 2003). Soil screening values were derived for this assessment using the Eco-SSL methodology with the toxicity reference values for Pb (USEPA, 2005d, 2005e) and consideration of the inputs on diet composition, food intake rates, incidental soil ingestion, and contaminant uptake by prey (details are presented in Section 7.1.3.1 and Appendix L, of ICF, 2006). Hardness-specific surface water screening values were calculated for each site based on EPA's recommended ambient water quality criteria for Pb (USEPA, 1984). For sediment screening values, the assessment relied on sediment ''threshold effect concentrations'' and ''probable effect concentrations'' developed by MacDonald *et al.* (2000). 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).

The HQ is calculated as the ratio of the media concentration to the ecotoxicity screening value, and represented by the following equation:

HQ = (estimated Pb media concentration) / (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. HQ values greater than 1.0 indicate that the expected exposure exceeds the ecotoxicity screening value and that there is a potential for adverse effects.

There are several uncertainties that apply across case studies noted below:

- 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. In identifying sites for inclusion in the assessment, efforts were made to ensure that the Pb exposures assessed were attributable to airborne 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. New data and approaches (e.g., use of pH as indicator of bioavailability) may now be available to estimated the aquatic toxicity of Pb (CD, Sections AX7.2.1.2 and 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 (Appendix L, ICF, 2006).

2. Summary of Results

The following is a brief summary of key observations related to the results of the screening-level ecological risk assessment. A more complete discussion of the results is provided in Chapter 6 of the Staff Paper and the complete presentation of the assessment and results is presented in the pilot phase Risk Assessment Report (ICF, 2006).

- The national-scale screen of surface water data initial identified some 42 sample locations of which 15 were then identified as unrelated to mining sites and having 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.
- In the national-scale screen of sediment data associated with the 15 surface water sites described above, threshold effect concentration-based HQs at nine of these sites exceeded 1.0. Additionally, HQs based on probable effect concentrations exceeded 1.0 at five of the sites, indicating probable adverse effects to sediment dwelling organisms. Thus, sediment Pb concentrations at some sites are high enough that there is a likelihood that they would cause adverse effects to sediment dwelling organisms. However, the contribution of air emissions to these concentrations is unknown.
- In the primary Pb smelter case study, all three of the soil sampling clusters (including the ''reference areas'') had HQs that exceeded 1.0 for birds. Samples from one cluster also had HQs greater than 1.0 for plants and mammals. The surface water sampling clusters all had measurements below the detection limit of 3.0 μg/L. However, three sediment sample clusters had HQs greater than 1.0. 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 2007b; ICF 2006).
- In the secondary Pb smelter case study, the soil concentrations, developed from soil data for similar locations, resulted in avian HQs greater than 1.0 for all distance intervals evaluated. The scaled soil concentrations within 1 km of the facility also showed HQs greater than 1.0 for plants, birds, and mammals. These estimates indicate a potential for adverse effect to those receptor groups.
- In the nonurban, near roadway case study, HQs for birds and mammals were greater than 1.0 at all but one of the distances from the road. Plant HQs were greater than 1.0 at the closest distance. In summary, HQs above one were estimated for plants, birds and mammals, indicating potential for adverse effect to these receptor groups.

C. Considerations in Review of the Standard

This section presents an integrative synthesis of information in the Criteria Document together with EPA analyses and evaluations. EPA notes that the final decision on retaining or revising the current secondary Pb standard is a public policy judgment to be made by the Administrator. The Administrator's final decision will 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.

The NAAQS provisions of the Act require the Administrator to establish secondary standards 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.

The following discussion starts with background information on the current standard (Section IV.C.1). The general approach for this current review is summarized in Section IV.C.2. Considerations with regard to the adequacy of the current standard are discussed in section IV.C.3, with evidence and exposure-risk-based considerations in subsections IV.C.3.a and b, respectively, followed by a summary of CASAC advice and recommendations (section IV.C.3.c) and, lastly, solicitation of comment on the broad range of policy options (section IV.C.3.d). Considerations with regard to elements of alternative standards are discussed in Section IV.C.4.

1. 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 III.C.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 Criteria Document 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, it is generally 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 Criteria Document (USEPA, 1986a) and the 1990 Staff Paper (USEPA, 1990). In summarizing OAQPS staff conclusions and recommendations at that time, the 1990 Staff Paper 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, the 1990 Staff Paper highlighted concerns over potential ecosystem effects of Pb due to its persistence, but concluded that pending development of a stronger database that more accurately quantifies ecological effects of different Pb concentrations, consideration should be given to retaining a secondary standard at or below the level of the then-current secondary standard of 1.5 μg/m^3.

2. Approach for Current Review

To evaluate whether it is appropriate to consider retaining the current secondary Pb standard, or whether consideration of revisions is appropriate, EPA is considering an approach in this review like that used in the Staff Paper that considers the evidence and risk analyses. This approach 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.

3. Adequacy of the Current Standard

a. Evidence-Based Considerations

In considering the welfare effects evidence with respect to the adequacy of the current standard, EPA will consider not only the array of evidence newly assessed in the Criteria Document but also that assessed in the 1986 Criteria Document 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 (2006 Criteria Document). Some of the more relevant aspects of the evidence available since the standard was set include the following:

- A more quantitative determination of the mobility, distribution, uptake, speciation, and fluxes of atmospherically delivered Pb in terrestrial ecosystems shows 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, 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).
- There is 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).
- New methods for assessing the toxicity of metals to water column and sediment-dwelling organisms and data collection efforts (CD, Sections 7.2.1, 7.2.2, AX7.2.2, and AX7.2.2.2) have improved our understanding of Pb aquatic toxicity and findings include an indication 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 (such as co-occurring pollutants) have not been well addressed (CD, Section AX7.1.4.2).
- Evidence in ecological research generally indicates the value of a critical loads approach; however, 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, despite wide variations in Pb concentrations in soils throughout the country, Pb concentrations are likely 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). For areas influenced by point sources of air Pb that meet the current standard, concentrations of Pb in soil may exceed by many orders of magnitude the concentrations which are considered harmful to laboratory organisms (CD, Section 3.2 and AX7.1.2.3).

There are several difficulties in quantifying the role of current ambient Pb in the environment: Some Pb deposited before the standard was

enacted is still present in soils and sediments; 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. However, the evidence summarized above and in Section 4.2 of the Staff Paper and described in detail in the Criteria Document informs our understanding of Pb in the environment today and evidence of environmental Pb exposures of potential concern.

Conditions exist in which Pb-associated 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 contamination likely to be a factor for the foreseeable future. Based on this information, the Staff Paper concluded that the evidence suggests that the environmental levels of Pb occurring under the current standard, set nearly thirty years ago, may pose risk of adverse environmental effect.

b. Risk-based Considerations

In addition to the evidence-based considerations described in the previous section, the screening level ecological risk assessment is informative, 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.

The ecological screening levels employed in the screening level risk assessment for different media are drawn from different sources. 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 United States. Consideration of the environmental risks associated with the current standard is complicated by the environmental burden associated with air Pb concentrations that exceeded the current standard, predominantly in the past.

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 in terrestrial organisms (plants, birds and mammals) and in 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 2007b; ICF 2006). Additionally, estimates of Pb concentration in soils associated with the nonurban near roadway case study and 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 the secondary Pb smelter 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 United States today. Further the magnitude of Pb concentrations in several aquatic systems 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 (e.g., Section 2.8.1). 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). However, 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, pages AX7–141 to AX7–145).

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 at concentrations, particularly in soil, and aquatic sediment 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 and soil. Further, environmental concentrations may be related to emissions prior to establishment of the current standard and such concentrations appear to indicate a potential for harm to ecological receptors today.

c. CASAC Advice and Recommendations

In the CASAC letter transmitting advice and recommendations pertaining to the review of the first draft 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 Pb concentrations and deposition—combined with a large reservoir of historically deposited Pb 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 also cites the persistence of Pb in the environment, the possibility of some of the large amount of historically deposited Pb 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.

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 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.

d. Policy Options

In considering the adequacy of the current secondary standard, EPA will consider, for reasons discussed above in III.C.3.d on the primary standard, whether it is appropriate to maintain a NAAQS for Pb or to retain Pb on the list of criteria pollutants. We take note of the views of CASAC, summarized above, the conclusions and recommendations in the OAQPS Staff Paper, and the views of public commenters on these questions. We recognize that there may be differing views on interpreting or weighing the available information. Thus, EPA solicits comment related to the questions of delisting and revocation.

In further considering the adequacy of the current standard in providing requisite protection from Pb-related adverse effects on public welfare, EPA will focus on the body of available evidence (briefly summarized above in Section IV.A). Depending on the interpretation, the available data and evidence, primarily qualitative, may suggest 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, taking into account the available evidence and current media concentrations in a wide range of areas, EPA seeks comment on whether the evidence suggests 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 to be persistent in the environment. Historically deposited Pb has persisted, although location-specific dynamics of Pb in soil result in differences in the timeframe during which Pb is retained in surface soils or sediments where it may be available to ecological receptors (USEPA, 2007b, section 2.3.3). EPA seeks comment on the role of deposition of Pb from current sources and the availability of this Pb to ecological receptors.

There is only very limited information available pertinent to assessing whether groups of organisms which influence ecosystem function are subject to similar effects as those in humans. The screening-level risk information, while limited and accompanied by various uncertainties, also suggests occurrences of environmental Pb concentrations existing under the current standard that could have adverse environmental effects. Environmental Pb levels today are associated with atmospheric Pb concentrations and deposition that have combined with a large reservoir of historically deposited Pb in environmental media.

The EPA takes note of the views of CASAC, summarized above, the conclusions and recommendations in the OAQPS Staff Paper, and views of public commenters on the adequacy of the current standard. EPA solicits comment on the adequacy of the current standard and the rationale upon which such views are based.

4. Elements of the Standard

The secondary standard is defined in terms of four basic elements: indicator, averaging time, level and form, which serve to define the standard and must be considered collectively in evaluating the welfare protection afforded by the standards. In considering a revision to the current standard, EPA will 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, EPA notes that Pb is a persistent pollutant to which ecological receptors are exposed via multiple pathways. 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 are insufficient to identify an indicator other than total Pb that would provide protection against adverse environmental effect in all ecosystems nationally.

Lead 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 is currently insufficient for such use in this review.

There is a general lack of data that would indicate the appropriate level of Pb in environmental media that may be associated with adverse effects. The EPA notes the influence of airborne Pb on Pb in aquatic systems and of changes in airborne Pb on 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), as well as the comments of the CASAC Pb panel 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 Pb exposures in the environment and Pb 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). We concur 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 in indicator, averaging time, level or form. Thus, EPA solicits comment on the option of a reduction in the secondary standard consistent with any reduction of the primary standard that would provide increased protection against adverse environmental effect.

Beyond the views noted above, EPA recognizes that there may be differing interpretations of the available evidence and various aspects of the evidence and exposure and risk information, including on the important uncertainties and limitations associated with the evidence and assessment. Thus, EPA solicits additional information pertaining to and comment on the considerations described above, as well as on other views with regard to the elements of a secondary standard for Pb, and the rationale upon which such views are based.

V. Considerations for Ambient Monitoring

A determination of compliance with the Pb NAAQS for any given area is made based on ambient air monitoring data collected by State and local monitoring agencies. This section discusses aspects of the Pb surveillance monitoring requirements with regards to the adequacy under the current primary Pb NAAQS as well as under options being considered for a revised primary Pb NAAQS. These aspects include the sampling and analysis methods, network design, sampling schedule, and data handling methods. In addition, this section discusses the need for monitoring in support of the secondary Pb NAAQS.

A. Sampling and Analysis Methods

To be used in determination of compliance with the Pb NAAQS, the Pb data must be collected and analyzed using a Federal Reference Method (FRM), or a Federal Equivalent Method (FEM). The current FRM for Pb sampling and analyses is based on the use of a high-volume TSP sampler to collect the sample and the use of atomic absorption for the analysis of Pb in the sample (40 CFR 50 Appendix G). There are 21 FEMs currently approved for Pb-TSP (*http://www.epa.gov/ttn/amtic/criteria.html*). All 21 FEMs are based on the use of high-volume TSP samplers, but with a variety of different analysis methods (e.g., XRF and ICP/MS).

Concerns have been raised over the use of high-volume TSP samplers. CASAC has commented that TSP samplers have poor precision, that the upper particle cut size varies widely as a function of wind speed and direction, and that the spatial non-homogeneity of very coarse particles cannot be efficiently captured by a national monitoring network (Henderson, 2007b). For these reasons, CASAC recommended considering a revision to the Pb reference method to allow sample collection using PM_{10} samplers. CASAC suggested that it may be possible to develop a single quantitative adjustment factor from a short period of collocated sampling at multiple sites, or a Pb-PM_{10}/Pb-TSP equivalency ratio could be determined on a regional or site-specific basis.

The EPA evaluated the precision and bias of the high-volume Pb-TSP sampler based on data reported to AQS for collocated samplers and results of in-field sampler flow audits and laboratory audits for Pb (Camalier and Rice, 2007). In this evaluation, we found that the average precision of the high-volume Pb-TSP sampler was approximately 12%, with a standard deviation of 19%, and average sampling bias (based on flow audits) was -0.7%, with a standard deviation of 4.2%. We also estimated the average bias for the lab analyses at -1.1% (with a standard deviation of 5.5%) based on spiked filter audits. Total bias, which includes bias from both sampling and laboratory analysis, was estimated at -1.7%, with a standard deviation of 3.4%. This level of precision and bias is comparable to the goal of the FRM and FEM for other criteria pollutants (e.g., within 10% for $PM_{2.5}$, 40 CFR 58 Appendix A). We attempted to look at the precision of low-volume Pb-PM_{10} samplers based on data reported to AQS, however, we did not have enough data (18 paired data points for one site) to make any conclusions on the precision of this sampler.

Evaluations of the high-volume TSP sampler have demonstrated that the sampler's cutpoint can vary between 25 and 50 μm depending on wind speed and direction (Wedding *et al.*, 1977, McFarland and Rodes, 1979). A study was conducted during the last Pb NAAQS review to evaluate the effect of wind speed and direction on sampler efficiency (Purdue, 1988). This demonstration showed that the Pb collection efficiency of the high-volume TSP sampler ranged from 80% to 90% over a wide range of wind speeds and directions. In comparison, a study conducted near a primary Pb smelter indicated that the ratio of Pb-PM_{10} to Pb-TSP ranged from 17% to 186% for 22 collocated samples (Brion, 1988). We believe that the variability of the collection efficiency of the high-volume TSP sampler does not warrant the discontinuation of its use. However, with this notice, we are soliciting comments on this issue.

We analyzed data from a number of monitoring sites where collocated Pb-TSP and Pb-PM_{10} data have been collected in order to evaluate the appropriateness of using Pb-PM_{10} data as a surrogate for Pb-TSP (Cavender, 2007). From this analysis it is clear that a single relationship can not be made that would allow one to accurately estimate Pb-TSP concentrations from Pb-PM_{10} measurements at all sites. However, at many locations it does appear a strong linear relationship can be shown between Pb-TSP and Pb-PM_{10} concentrations. As such, it may be feasible for a monitoring agency to develop a site-specific relationship, using conservative assumptions, to estimate Pb-TSP based on Pb-PM_{10} measurements. We invite comments on the appropriateness of using Pb-PM_{10} data as a surrogate for Pb-TSP.

While all current FRM and FEM are based on the high-volume TSP sampler, several vendors market low-volume TSP samplers. These samplers are identical to low-volume PM_{10} samplers with the exception of the sampling head and corresponding cut size. These samplers have a number of advantages over the high-volume TSP sampler including the capability of sequential sampling (i.e., the ability to collect more than one sample between operator visits). Sequential sampling would be highly desirable if the sampling frequency is increased as part of a change to a monthly averaging period. Currently, the FEM demonstration requirements [40 CFR 53.33(i)] dictate that the FEM testing must be performed with an ambient Pb-TSP concentration between 0.5 $\mu g/m^3$ to 4.0 $\mu g/m^3$. Due to the dramatic decrease in ambient Pb concentrations, there are few (if any) areas in the country where a vendor could be assured that the average ambient Pb-TSP concentrations would meet the FEM demonstration requirements during the field testing period. If the Pb NAAQS is lowered, we believe it is appropriate to lower the FEM requirement to a level more consistent with current ambient Pb concentrations and the lowered NAAQS to allow for continued development and approval of Pb-TSP FEM. We invite comment on the appropriate range of concentrations for an FEM demonstration.

We also reviewed the method detection capabilities of the current lab methods for the FRM and FEM to ensure that these methods had the necessary sensitivity to accurately measure Pb-TSP at the low concentrations considered in the Risk Assessment Report and Staff Paper. Based on data submitted to AQS, the method detection limits for these methods are all 0.01 μg/m^3 or less (Rice, 2007). From these findings, we request comment on whether the current lab analysis methods are adequate for continued use even at the lowest alternative NAAQS levels considered in the Risk Assessment Report and Staff Paper.

B. Network Design

The existing Pb-TSP network has decreased substantially over the last few decades. In 1980 there were over 900 Pb-TSP sites, this number has been reduced to approximately 200 sites. These reductions were made because of substantially reduced ambient Pb concentrations and shifting priorities to other criteria pollutants. Now several states have no Pb-TSP monitors resulting in large portions of the country with no data on current ambient Pb-TSP concentrations. In addition, many of the largest Pb emitting sources in the country do not have nearby monitors, and there is substantial uncertainty about ambient air Pb levels resulting from historic Pb deposits near roadways. For these reasons, we request comment on whether the existing Pb-TSP network may not be adequate, and that additional monitoring sites may be needed to determine compliance with either the current or revised Pb NAAQS.

The minimum network design requirements are given in 40 CFR 58 Appendix D. The current network design requirements are for 2 FRM or FEM sites in any area where Pb concentrations exceed or have exceeded the NAAQS in the most recent 2 years. These requirements may make it difficult to persuade state and local monitoring agencies to add monitors in areas without existing monitors. As such, we believe that these requirements are not adequate and should be modified (as part of this rulemaking) to ensure monitoring is conducted in areas where NAAQS violations may occur.

We request comment on options for improving the coverage of the Pb network. One option would be to adopt network requirements similar to those recently promulgated for $PM_{2.5}$ and ozone which tie the number of required monitors to the population of the urban area and ambient Pb concentrations (40 CFR 58 Appendix D). Under this approach, more monitoring sites would be required in areas with larger populations and higher Pb concentrations. This approach would result in improved network coverage in urban areas. However, large Pb emitting sources that are not in urban areas may still not be monitored.

A second option would be to require one or more monitors near large Pb emitting sources. For example, a monitor could be required at the point near the maximum predicted concentrations for sources with a potential Pb emission rate of 1 ton per year or more (as provided by the most recent National Emissions Inventory, or permit data). Clearly, some effort would be necessary to identify an appropriate emissions threshold to ensure that all emission sources with the potential to exceed the NAAQS are monitored without creating undue burden where there is no potential to exceed the NAAQS. This option would ensure coverage of the highest Pb emitting sources, but may not provide adequate coverage in many populated areas where a combination of smaller emissions sources and re-entrained dust may result in Pb concentrations in excess of the NAAQS.

A third option could be created by the combination of the first two options discussed above: Establish a minimum number of required monitors in urban areas based on population and ambient Pb concentrations and require monitors near large Pb emission sources. This option would provide good monitoring coverage in urban areas and near Pb emissions sources. Again, care would need to be taken in establishing an emissions threshold.

A fourth option would be to utilize the current PM_{10} network if an acceptable regional or site-specific correlation of Pb-TSP and Pb-PM_{10} can be made. This option would provide a substantial increase in monitoring coverage without requiring a large investment in new monitoring stations. The current PM_{10} network has been carefully established to include both rural and urban ambient levels, though it was not designed to monitor near large Pb emitting sources. We invite comments on these options as well as suggestions for additional options to consider for improving the Pb network.

C. Sampling Schedule

The current sampling frequency requirement is for one 24-hour sample every six days [40 CFR 58.12(b)]. For the current NAAQS, which is based on a quarterly average, the 1-in-6 sampling schedule yields 15 samples per quarter on average with 100% completeness, or 12 samples with 75% completeness. A change to a monthly averaging period would result in between 4 and 6 samples per month at the current sampling frequency. If we change the averaging time to a monthly average, we would likely need to increase the sampling frequency as 4 samples would not result in a statistically valid estimate of the actual air quality for the period.

Incomplete sampling results in increased uncertainty in the estimate of actual ambient air quality. While some degree of uncertainty is unavoidable due to the precision and bias inherent to the sampling technique, it is important to understand the level of uncertainty for each sampling option being considered and to select a sampling frequency which achieves an acceptable level of uncertainty. We plan to go through the Data Quality Objectives (DQO) process in order to help us select an appropriate sampling option. The DQO process is a series of logical steps that guides decision makers to a plan for the resource-effective acquisition of environmental data. The DQO process is used to establish performance and acceptance criteria, which serve as the basis for designing a plan for collecting data of sufficient quality and quantity to support the goals of the study (EPA, 2006e, EPA/240/B–06/001).

We are considering several options for sampling frequency. These options include maintaining the current 1-in-6 day sampling schedule, increasing the sampling frequency to 1-in-3 day, or increasing the sampling frequency to 1-in-1 day sampling (i.e., complete sampling). In addition, we will be considering an option that relates sampling frequency to recent ambient Pb-TSP concentrations, such that an increased sampling frequency is required as the recent ambient Pb-TSP concentrations approach the NAAQS level. Other options that we will be considering include—

- Increasing sampling time duration (e.g., changing from a 24 hour sampling time duration to a 48 or 72 hour sampling time duration).
- Allowing for compositing of samples (i.e., analyzing sequential samples together).
- Allowing for multiple samplers at one site.

We invite comments on the appropriateness of these sampling options and suggestions for additional options for consideration.

D. Data Handling

A number of data handling conventions and computations are necessary when using ambient monitoring data to determine attainment

or non-attainment of the NAAQS. Recently, we have been codifying these data handling conventions and computations into a separate appendix for each NAAQS. As such, we intend to create an appendix for the interpretation of the Pb NAAQS as part of this rule making. Specific conventions we are considering and invite comments on at this time include the following—

- Design values will be developed based on the most recent 3 calendar year period.
- Design values will be rounded to two significant figures using conventional rounding methodology.
- 75% of the expected number of samples is needed for a quarter to be considered complete, or 50% for a month.
- Only one period (i.e., one month or one quarter depending on the final form of the standard) is needed to demonstrate non-attainment. Two periods would be needed if the NAAQS is based on the 2nd maximum.
- Three full consecutive years of complete data are needed to re-designate an area attainment from non-attainment.
- Incomplete periods can be used to demonstrate non-attainment, but not attainment.

E. Monitoring for the Secondary NAAQS

Currently, the secondary NAAQS is set equal to the primary NAAQS (1.5 μg/m^3, maximum quarterly average). We do not expect there to be ambient air concentrations in excess of the secondary NAAQS in rural areas that are not associated with a Pb emission source. If the secondary standard remains equal to the primary standard at the completion of the current review, we request comment on the option of developing Pb surveillance monitoring requirements for the primary NAAQS that will be sufficient to determine compliance with the secondary NAAQS.

While additional monitoring may not be necessary to demonstrate compliance with the secondary NAAQS, CASAC has recommended additional monitoring to gather information to better inform consideration of the secondary NAAQS in the next and future reviews. Specifically, CASAC stated that ''the EPA needs to initiate new measurement activities in rural areas—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'' (Henderson, 2007b).

We currently monitor ambient Pb in $PM_{2.5}$ as part of the IMPROVE network. There are 110 formally designated IMPROVE sites 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. While we believe it may not be appropriate to rely on either Pb-PM_{10} or Pb-$PM_{2.5}$ monitoring to demonstrate compliance with a Pb-TSP NAAQS, we believe the Pb-$PM_{2.5}$ measurements provided by the IMPROVE network can be used as a useful indicator to track changes in ambient Pb concentrations and resulting Pb deposition in rural areas that are not directly impacted by a Pb emission source. It may also be desirable to augment the IMPROVE network with a small ''sentinel'' network of collocated Pb-TSP monitors for a period of time in order to develop a better understanding of how Pb-$PM_{2.5}$ and Pb-TSP relate in these rural areas. Alternatively, since it is likely that at rural locations nearly all Pb is in the less than 10 μm size range, we could analyze the PM_{10} mass samples (which are already being collected) for Pb for a period of time to develop a better understanding of how Pb-$PM_{2.5}$ and Pb-PM_{10} relate in these rural areas. We welcome comments on the value and appropriateness of use of the IMPROVE Pb-$PM_{2.5}$ data for assessing trends in ambient air concentrations of Pb, and the need to collocate a small network of Pb-TSP or Pb-PM_{10} monitors at IMPROVE sites.

The National Water-Quality Assessment (NAWQA), conducted by the United States Geological Survey, contains data on Pb concentrations in surface water, bed sediment, and animal tissue for more than 50 river basins and aquifers throughout the country (CD, AX7.2.2.2). NAWQA data are collected during long-term, cyclical investigations wherein study units undergo intensive sampling for 3 to 4 years, followed by low-intensity monitoring and assessment of trends every 10 years. Similarly, the USGS is collaborating with Canadian and Mexican government agencies on a multi-national project called ''Geochemical Landscapes'' that has as its long-term goal a soil geochemical survey of North America (*http://minerals.cr.usgs.gov/projects/geochemical_landscapes/index.html*). The Geochemical Landscapes project has the potential to fill the need for periodic Pb soil sampling. We note the value of the NAWQA and Geochemical Landscapes data in the assessment of trends in Pb concentrations in both soil and aquatic systems, and support the continued collection of this data by the USGS.

VI. Solicitation of Comment

With the issuance of this ANPR, the Agency is soliciting broad public input to inform the Agency's proposed rulemaking related to the review of the Pb NAAQS. As noted in Section I above, this ANPR, as a consequence of the timing of the Pb NAAQS review relative to the Agency's initiation of the new NAAQS process, summarizes information from the OAQPS Staff Paper, and from the Agency's risk assessment and Criteria Document. In so doing, this notice presents OAQPS staff views on the adequacy of the current standard and on a range of policy options for the Administrator's consideration, together with the views of CASAC and the public as reflected in their comments on the related documents that have been previously made available for review. The Agency is soliciting comment on the range of views discussed above as well as any broader range of options that members of the public feel appropriate for the Administrator to consider. Comments are solicited together with the rationales for the views expressed in those comments. The Agency is also soliciting further advice from CASAC on the issues discussed in this notice at an upcoming public meeting (announced in a separate **Federal Register** notice).

In soliciting public comment in advance of reaching proposed decisions on whether to retain or revise the NAAQS under review, the Agency is interested in general, specific, and technical comments on all aspects of the rulemaking discussed in this notice and the related documents. These aspects generally include characterization of Pb in the ambient environment, characterization of the health effects evidence and the assessment of human exposure and health risk, characterization of the environmental effects evidence and consideration of environmental exposure and risk, as well as an assessment of the adequacy of the current primary and secondary standards and of alternative standards for the Administrator's consideration in reaching proposed decisions in this review of the Pb NAAQS. We solicit broad comment on these aspects of this rulemaking, informed by the discussion presented in this notice as well as the more comprehensive discussion in the Criteria Document, the Staff Paper, and related risk assessment reports.

Several types of information pertinent to the characterization of Pb in the

ambient environment are considered for this review. These include characterization of sources of Pb, including source distribution within the U.S. and associated estimates of the magnitude of air emissions. The currently available information on the magnitude, geographic distribution and variability of Pb levels in the ambient air is also considered. Further, given that Pb is a multimedia pollutant, characterization of Pb includes consideration of atmospheric deposition and Pb in ambient soil, surface waters and sediment. Comments, including information and views, are solicited in all of these areas as well as any other areas related to the characterization of Pb in the ambient environment that are relevant to this review.

The current health effects evidence for Pb, evaluated in the Agency's Criteria Document, encompasses a broad range of information regarding human exposure to ambient Pb, toxicokinetics of Pb, biological markers and models of Pb burden in humans, toxicological effects of Pb in laboratory animals and in vitro test systems, and epidemiologic studies of human health effects associated with Pb exposure. In addition, based on the information in the Criteria Documents, quantitative assessments of human exposures to Pb and associated health risks as well as environmental exposures and related risks have been conducted and are presented in related risk assessment reports. We are soliciting comments, including information and views, informed by the Criteria Document, Staff Paper, and risk assessment reports, on characterization of the health effects evidence and consideration of human exposure and health risk associated with Pb exposures. Similarly, the Agency is soliciting comment on the characterization of the environmental effects evidence and environmental risks of Pb relevant to this review.

With regard to the primary and secondary standards, a wide range of views have been expressed, reflecting differing conclusions about the scientific evidence and quantitative risk assessments and differing public health and welfare policy judgments about appropriate standards. These views range from asserting the need for significant strengthening of the standards to a recommendation in public comments that the Pb NAAQS should be revoked and/or Pb should be delisted as a criteria pollutant. We solicit comment on these views as well as on any other views that are thought to be appropriate for the Agency to consider, together with rationales for the views expressed. More specifically, we solicit comment, including views and associated rationale, informed by the Criteria Document, Staff Paper and related risk assessment reports, on the adequacy of the current primary and secondary standards. We also solicit comment on the range of alternative primary and secondary standards the Agency should consider, with a focus on the four basic elements of the standards, including indicator, averaging time, level, and form. Further, we are soliciting comment on the view that it is appropriate to revoke the NAAQS for Pb or to remove Pb from the list of criteria pollutants.

Issues related to Pb surveillance monitoring requirements relevant to this review are also discussed in this notice. These issues fall into several areas, including sampling and analysis methods related to Pb-TSP and Pb-PM_{10} measurements, monitoring network design, sampling schedule, and data handling. Specific aspects of monitoring in support of the secondary standard are also discussed. We are soliciting comments on the issues related to Pb surveillance monitoring requirements identified in this notice as well as on other issues relevant to these requirements in this review.

The Agency will consider comments received in response to this notice in reaching proposed decisions in this rulemaking. As noted above, the public will have an additional opportunity for comment on the proposed rulemaking, which will further inform the Administrator's final decisions on the Pb NAAQS.

VII. Statutory and Executive Order Reviews

Executive Order 12866: Regulatory Planning and Review

Under Executive Order (EO) 12866 (58 FR 51735, October 4, 1993), this action is a ''significant regulatory action.'' Accordingly, EPA submitted this action to the Office of Management and Budget (OMB) for review under EO 12866 and any changes made in response to OMB recommendations have been documented in the docket for this action.

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List of Subjects in 40 CFR Part 50

Environmental protection, Air pollution control, Carbon monoxide, Lead, Nitrogen dioxide, Ozone, Particulate matter, Sulfur oxides.

Dated: December 5, 2007.

Stephen L. Johnson,

Administrator.

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