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6560-50-P

ENVIRONMENTAL PROTECTION AGENCY

40 CFR Part 50

[EPA-HQ-OAR-2010-0108; FRL-9915-57-OAR]

RIN 2060-AQ44

National Ambient Air Quality Standards for Lead

AGENCY: Environmental Protection Agency.

ACTION: Proposed rule.

SUMMARY: Based on the Environmental Protection Agency's (EPA's) review of the air quality criteria and the national ambient air quality standards (NAAQS) for lead (Pb), the EPA is proposing to retain the current standards, without revision.

DATES: Comments must be received on or before **[INSERT DATE 90 DAYS AFTER DATE OF PUBLICATION IN THE FEDERAL REGISTER].**

Public Hearings: If, by **[INSERT DATE 20 DAYS AFTER PUBLICATION IN THE FEDERAL REGISTER]**, the EPA receives a request from a member of the public to speak at a public hearing concerning the proposed decision, we will hold a public hearing, with information about the hearing provided in a subsequent notice in the *Federal Register*.

ADDRESSES: Submit your comments, identified by Docket ID No. EPA-HQ-OAR-2010-0108 by one of the following methods:

- Federal eRulemaking Portal: http://www.regulations.gov: Follow the on-line instructions for submitting comments.
- Email: a-and-r-Docket@epa.gov. Include docket ID No. EPA-HQ-OAR-2010-0108 in the subject line of the message.

- Fax: 202-566-9744.
- Mail: Docket No. EPA-HQ-OAR-2010-0108, Environmental Protection Agency, Mail code 28221T, 1200 Pennsylvania Ave., NW, Washington, DC 20460.
- Hand Delivery: Docket No. EPA-HQ-OAR-2010-0108, Environmental Protection Agency, EPA WJC West Building, 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-2010-0108. 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 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 www.regulations.gov or email. The www.regulations.gov website is an "anonymous access" system, which means the EPA will not know your identity or contact information unless you provide it in the body of your comment. If you send an email comment directly to the EPA without going through www.regulations.gov, your email 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, the 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 the EPA cannot read your comment due to technical difficulties and cannot contact you for clarification, the 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 the EPA's public docket, visit the EPA Docket Center homepage at http://www.epa.gov/epahome/dockets.htm.

Public Hearing: To request a public hearing or information pertaining to a public hearing on this document, contact Ms. Eloise Shepherd, Health and Environmental Impacts Division, Office of Air Quality Planning and Standards (C504-02), U.S. Environmental Protection Agency, Research Triangle Park, NC 27711; telephone number (919) 541-5507; fax number (919) 541-0804; email address: shepherd.eloise@epa.gov. See the Supplementary Information for further information about a possible public hearing.

Docket: All documents in the docket are listed on the www.regulations.gov website. This includes documents in the rulemaking docket (Docket ID No. EPA-HQ-OAR-2010-0108) and a separate docket, established for the Integrated Science Assessment for this review (Docket ID No. EPA-HQ-ORD-2011-0051) that has been incorporated by reference into the rulemaking docket. All documents in these dockets are listed on the www.regulations.gov website. 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, is not placed on the Internet and may be viewed, with prior arrangement, at the EPA Docket Center. Publicly available docket materials are available either electronically in www.regulations.gov or in hard copy at the Air and Radiation Docket Information Center, EPA/DC, WJC West Building, 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 Information Center is (202) 566-1742.

FOR FURTHER INFORMATION CONTACT: Dr. Deirdre L. 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; email: murphy.deirdre@epa.gov. To request a public hearing or information pertaining to a public hearing on this document, contact Ms. Eloise Shepherd, Health and Environmental Impacts Division, Office of Air Quality Planning and Standards (C504-02), U.S. Environmental Protection Agency, Research Triangle Park, NC 27711; telephone number (919) 541-5507; fax number (919) 541-0804; email address: shepherd.eloise@epa.gov.

SUPPLEMENTARY INFORMATION:

General Information

Preparing Comments for the EPA

1. *Submitting CBI.* Do not submit this information to the EPA through www.regulations.gov or email. 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 the 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.
- 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 Information Related to this Action

A number of the documents that are relevant to this action are available through the EPA's Office of Air Quality Planning and Standards (OAQPS) Technology Transfer Network (TTN) website at http://www.epa.gov/ttn/naaqs/standards/pb/s_pb_index.html. These documents include the *Plan for Review of the National Ambient Air Quality Standards for Lead* (USEPA, 2011a), available at http://www.epa.gov/ttn/naaqs/standards/pb/s_pb_2010_pd.html, the *Integrated Science Assessment for Lead* (USEPA, 2013a), available at http://www.epa.gov/ttn/naaqs/standards/pb/s_pb_2010_isa.html, the *Review of the National Ambient Air Quality Standards for Lead: Risk and Exposure Assessment Planning Document* (USEPA, 2011b), available at http://www.epa.gov/ttn/naaqs/standards/pb/s_pb_2010_pd.html, and the *Policy Assessment for the Review of the Lead National Ambient Air Quality Standards* (USEPA, 2014), available at http://www.epa.gov/ttn/naaqs/standards/pb/s_pb_2010_pa.html. These and other related documents are also available for inspection and copying in the EPA

docket identified above.

Information about a Possible Public Hearing

To request a public hearing or information pertaining to a public hearing on this document, contact Ms. Eloise Shepherd, Health and Environmental Impacts Division, Office of Air Quality Planning and Standards (C504-02), U.S. Environmental Protection Agency, Research Triangle Park, NC 27711; telephone number (919) 541-5507; fax number (919) 541-0804; email address: shepherd.eloise@epa.gov.

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I. Background

A. Legislative Requirements

Two sections of the Clean Air Act (CAA or the Act) govern the establishment and revision of the NAAQS. Section 108 (42 U.S.C. 7408) directs the Administrator to identify and list certain air pollutants and then to issue air quality criteria for those pollutants. The Administrator is to list those air pollutants that in her "judgment, cause or contribute to air pollution which may reasonably be anticipated to endanger public health or welfare;" "the presence of which in the ambient air results from numerous or diverse mobile or stationary

sources;" and "for which . . . [the Administrator] plans to issue air quality criteria…" Air quality criteria are intended to "accurately reflect the latest scientific knowledge useful in indicating the kind and extent of all identifiable effects on public health or welfare which may be expected from the presence of [a] pollutant in the ambient air . . ." 42 U.S.C. 7408(b). Section 109 (42 U.S.C. 7409) directs the Administrator to propose and promulgate "primary" and "secondary" NAAQS for pollutants for which air quality criteria are issued. Section 109(b)(1) defines a primary standard as one "the attainment and maintenance of which in the judgment of the Administrator, based on such criteria and allowing an adequate margin of safety, are requisite to protect the public health."[1] 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 such criteria, is requisite to protect the public welfare from any known or anticipated adverse effects associated with the presence of [the] pollutant in the ambient air." [2]

The requirement that primary standards provide an adequate margin of safety was intended to address uncertainties associated with inconclusive scientific and technical information available at the time of standard setting. It was also intended to provide a reasonable degree of protection against hazards that research has not yet identified. *See Lead Industries Association v. EPA*, 647 F.2d 1130, 1154 (D.C. Cir 1980), *cert. denied*, 449 U.S. 1042 (1980); *American Petroleum Institute v. Costle*, 665 F.2d 1176, 1186 (D.C. Cir. 1981), *cert. denied*, 455 U.S. 1034 (1982); *American Farm Bureau Federation v. EPA*, 559 F. 3d 512, 533 (D.C. Cir.

[1] 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." See S. Rep. No. 91-1196, 91st Cong., 2d Sess. 10 (1970).

[2] 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."

2009); *Association of Battery Recyclers v. EPA*, 604 F. 3d 613, 617-18 (D.C. Cir. 2010). 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 provide 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. The CAA does not require the Administrator to establish a primary NAAQS at a zero-risk level or at background concentration levels, see *Lead Industries v. EPA*, 647 F.2d at 1156 n.51, but rather at a level that reduces risk sufficiently so as to protect public health with an adequate margin of safety.

In addressing the requirement for an adequate margin of safety, the EPA considers such factors as the nature and severity of the health effects involved, the size of sensitive population(s) at risk,[3] 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. See *Lead Industries Association v. EPA*, 647 F.2d at 1161-62.

In setting primary and secondary standards that are "requisite" to protect public health and welfare, respectively, as provided in section 109(b), the EPA's task is to establish standards that are neither more nor less stringent than necessary for these purposes. In so doing, the EPA may not consider the costs of implementing the standards. See generally, *Whitman v. American Trucking Associations*, 531 U.S. 457, 465-472, 475-76 (2001). Likewise, "[a]ttainability and technological feasibility are not relevant considerations in the promulgation of national ambient

[3] As used here and similarly throughout this notice, the term population (or group) refers to persons having a quality or characteristic in common, such as a specific pre-existing illness or a specific age or life stage. As discussed more fully in section II.B.4 below, the identification of sensitive groups (called at-risk groups or at-risk populations) involves consideration of susceptibility and vulnerability.

air quality standards." *American Petroleum Institute v. Costle*, 665 F. 2d at 1185.

Section 109(d)(1) requires that "not later than December 31, 1980, and at 5-year intervals thereafter, the Administrator shall complete a thorough review of the criteria published under section 108 and the national ambient air quality standards . . . and shall make such revisions in such criteria and standards and promulgate such new standards as may be appropriate . . .. " Section 109(d)(2) requires that an independent scientific review committee "shall complete a review of the criteria . . . and the national primary and secondary ambient air quality standards. . . and shall recommend to the Administrator any new . . . standards and revisions of existing criteria and standards as may be appropriate . . . ." Since the early 1980s, this independent review function has been performed by the Clean Air Scientific Advisory Committee (CASAC).[4]

B. Related Lead Control Programs

States are primarily responsible for ensuring attainment and maintenance of the NAAQS. Under section 110 of the Act (42 U.S.C. 7410) and related provisions, states are to submit, for EPA approval, state implementation plans (SIPs) 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 the EPA, also administer the Prevention of Significant Deterioration program (42 U.S.C. 7470–7479) for these pollutants.

The NAAQS is only one component of the EPA's programs to address Pb in the environment. Federal programs additionally provide for nationwide reductions in air 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

[4] Lists of CASAC members and of members of the CASAC Lead Review Panel are available at: http://yosemite.epa.gov/sab/sabproduct.nsf/WebCASAC/CommitteesandMembership?OpenDocument.

section 111 of the Act (42 U.S.C. 7411); emissions standards for solid waste incineration units and the national emission standards for hazardous air pollutants (NESHAP) under sections 129 (42 U.S.C. 7429) and 112 (42 U.S.C. 7412) of the Act, respectively.

The EPA has taken a number of actions associated with these air pollution control programs since the last review of the Pb NAAQS, including completion of several regulations which will result in reduced Pb emissions from stationary sources regulated under the CAA sections 112 and 129. For example, in January 2012, the EPA updated the NESHAP for the secondary lead smelting source category (77 FR 555, January 5, 2012). These amendments to the original maximum achievable control technology standards apply to facilities nationwide that use furnaces to recover Pb from Pb-bearing scrap, mainly from automobile batteries (15 existing facilities, one under construction). By the effective date in 2014, this action is estimated to result in a Pb emissions reduction of 13.6 tons per year (tpy) across the category (a 68% reduction). Somewhat lesser Pb emissions reductions are also expected from regulations completed in 2013 for commercial and industrial solid waste incineration units (78 FR 9112, February 7, 2013), as well as several other regulations since 2007 (72 FR 73179, December 26, 2007; 72 FR 74088, December 28, 2007; 73 FR 225, November 20, 2008; 78 FR 10006, February 12, 2013; 76 FR 15372, March 21, 2011; 78 FR 7138, January 31, 2013; 74 FR 51368, October 6, 2009; Policy Assessment, Appendix 2A).

The presentation below briefly summarizes additional ongoing activities that, although not directly pertinent to the review of the NAAQS, are associated with controlling environmental Pb levels and human Pb exposures more broadly. Among those identified are the EPA programs intended to encourage exposure reduction programs in other countries.

Reducing Pb exposures has long been recognized as a federal priority as environmental

and public health agencies continue to grapple with soil and dust Pb levels from the historical use of Pb in paint and gasoline and from other sources (Alliance to End Childhood Lead Poisoning, 1991; 62 FR 19885, April 23, 1997; 66 FR 52013, October 11, 2001; 68 FR 19931, April 23, 2003). A broad range of federal programs beyond those that focus on air pollution control provide for nationwide reductions in environmental releases and human exposures. For example, pursuant to section 1412 of the Safe Drinking Water Act (SDWA), the EPA regulates Pb in public drinking water systems through corrosion control and other utility actions which work together to minimize Pb levels at the tap (40 CFR 141.80-141.91). Under section 1417 of the SDWA, pipes, fittings and fixtures for potable water applications may not be used or introduced into commerce unless they are considered "lead free" as defined by that Act (40 CFR 141.43).[5] Additionally, federal Pb 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 (68 FR 36487, June 18, 2003).

Federal programs to reduce exposure to Pb in paint, dust, and soil are specified under the comprehensive federal regulatory framework developed under the Residential Lead-Based Paint Hazard Reduction Act (Title X). Under Title X (codified as Title IV of the Toxic Substances Control Act [TSCA]), the EPA has established regulations and associated programs in six categories: (1) training, certification and work practice requirements for persons engaged in Pb-based paint activities (abatement, inspection and risk assessment); accreditation of training providers; and authorization of state and tribal Pb-based paint programs; (2) training, certification, and work practice requirements for persons engaged in home renovation, repair and

[5] Effective in January 2014, the amount of Pb permitted in pipes, fittings, and fixtures was lowered (see "Summary of the Reduction of Lead in Drinking Water Act and Frequently Asked Questions" at http://water.epa.gov/drink/info/lead/index.cfm).

painting (RRP) activities; accreditation of RRP training providers; and authorization of state and tribal RRP programs; (3) ensuring that, for most housing constructed before 1978, information about Pb-based paint and Pb-based paint hazards flows from sellers to purchasers, from landlords to tenants, and from renovators to owners and occupants; (4) establishing standards for identifying dangerous levels of Pb in paint, dust and soil; (5) providing grant funding to establish and maintain state and tribal Pb-based paint programs; and (6) providing information on Pb hazards to the public, including steps that people can take to protect themselves and their families from Pb-based paint hazards. The most recent rule issued under Title IV of TSCA is for the Lead Renovation, Repair and Painting Program (73 FR 21692, April 22, 2008), which became fully effective in April 2010 and which applies to compensated renovators and maintenance professionals who perform RRP activities in housing and child-care facilities built prior to 1978. To foster adoption of the rule's measures, the EPA has been conducting an extensive education and outreach campaign to promote awareness of these new requirements among both the regulated entities and the consumers who hire them (http://www2.epa.gov/lead/renovation-repair-and-painting-program). In addition, the EPA is investigating whether Pb hazards are also created by RRP activities in public and commercial buildings, in which case the EPA plans to issue RRP requirements, where appropriate, for this class of buildings (79 FR 31072, May 30, 2014).

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, the 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., 66 FR 58258, November 20, 2001). Federal regulations concerning batteries in municipal solid waste facilitate the collection and recycling or proper disposal of batteries containing Pb.[6] 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 by encouraging pollution prevention, promotion of reuse and recycling, reduction of priority and toxic chemicals in products and waste, and conservation of 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 the 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).

The EPA's research program identifies, encourages and conducts research needed to locate and assess serious risks and to develop methods and tools to characterize and help reduce risks related to Pb exposure. For example, the EPA's Integrated Exposure Uptake Biokinetic Model for Lead in Children (IEUBK model) is widely used and accepted as a tool that informs the evaluation of site-specific data. More recently, in recognition of the need for a single model that predicts Pb concentrations in tissues for children and adults, the EPA has been developing the All Ages Lead Model (AALM) to provide researchers and risk assessors with a

[6] See, e.g., "Implementation of the Mercury-Containing and Rechargeable Battery Management Act" at http://www.epa.gov/epawaste/hazard/recycling/battery.pdf and "Municipal Solid Waste Generation, Recycling, and Disposal in the United States: Facts and Figures for 2005 http://www.epa.gov/epawaste/nonhaz/municipal/pubs/msw-2005.pdf.

pharmacokinetic model capable of estimating blood, tissue, and bone concentrations of Pb based on estimates of exposure over the lifetime of the individual (USEPA, 2006a, sections 4.4.5 and 4.4.8; USEPA, 2013a, section 3.6). The EPA's research activities on substances including Pb, such as those identified here, focus on improving our characterization of health and environmental effects, exposure, and control or management of environmental releases (see http://www.epa.gov/research/).

Other federal agencies also participate in programs intended to reduce Pb exposures. For example, programs of the Centers for Disease Control and Prevention (CDC) provide for the tracking of children's blood Pb levels in the U.S. and provide guidance on levels at which medical and environmental case management activities should be implemented (CDC, 2012; ACCLPP, 2012). As a result of coordinated, intensive efforts at the national, state and local levels, including those programs described above, blood Pb levels in all segments of the population have continued to decline from levels observed in the past. For example, blood Pb levels for the general population of children 1 to 5 years of age have dropped to a geometric mean level of 1.17 μg/dL in the 2009-2010 National Health and Nutrition Examination Survey (NHANES) as compared to the geometric mean in 1999-2000 of 2.23 μg/dL and in 1988-1991 of 3.6 μg/dL (USEPA, 2013a, section 3.4.1; USEPA, 2006a, AX4-2). Similarly, blood Pb levels in non-Hispanic black, Mexican American and lower socioeconomic groups, which are generally higher than those for the general population, have also declined (USEPA, 2013a, sections 3.4.1, 5.2.3 and 5.2.4; Jones et al., 2009).

The EPA also participates in a broad range of international programs focused on reducing environmental releases and human exposures in other countries. For example, the Partnership for Clean Fuels and Vehicles program engages governments and stakeholders in developing

countries to eliminate Pb in gasoline globally.[7] From 2007 to 2011, the number of countries known to still be using leaded gasoline was reduced from just over 20 to six, with three of the six also offering unleaded fuel. All six were expected to eliminate Pb from fuel in the near future (USEPA, 2011c). The EPA is a contributor to the Global Alliance to Eliminate Lead Paint, a cooperative initiative jointly led by the World Health Organization and the United Nations Environment Programme (UNEP) to focus and catalyze the efforts to achieve international goals to prevent children's Pb exposure from paints containing Pb and to minimize occupational exposures to Pb paint. This alliance has the broad objective of promoting a phase-out of the manufacture and sale of paints containing Pb and eventually to eliminate the risks that such paints pose. The UNEP is also engaged on the problem of managing wastes containing Pb, including Pb-containing batteries. The Governing Council of the UNEP, of which the U.S. is a member, has adopted decisions focused on promoting the environmentally sound management of products, wastes and contaminated sites containing Pb and reducing risks to human health and the environment from Pb and cadmium throughout the life cycles of those substances (UNEP Governing Council, 2011, 2013). The EPA is also engaged in the issue of environmental impacts of spent Pb-acid batteries internationally through the Commission for Environmental Cooperation (CEC), where the EPA Administrator along with the cabinet-level or equivalent representatives of Mexico and Canada comprise the CEC's senior governing body (CEC Council).[8]

C. Review of the Air Quality Criteria and Standards for Lead

[7] International programs in which the U.S. participates, including those identified here, are described at: http://epa.gov/international/air/pcfv.html, http://www.unep.org/transport/pcfv/, http://www.unep.org/hazardoussubstances/Home/tabid/197/hazardoussubstances/LeadCadmium/PrioritiesforAction/GAELP/tabid/6176/Default.aspx.

[8] The CEC was established to support cooperation among the North American Free Trade Agreement partners to address environmental issues of continental concern, including the environmental challenges and opportunities presented by continent-wide free trade.

Unlike pollutants such as particulate matter and carbon monoxide, air quality criteria had not been issued for Pb as of the enactment of the CAA of 1970, which first set forth the requirement to set NAAQS based on air quality criteria. In the years just after enactment of the CAA, the EPA did not list Pb under Section 108 of the Act, having determined to control Pb air pollution through regulations to phase out the use of Pb additives in gasoline (See 41 FR 14921, April 8, 1976). However, the decision not to list Pb under Section 108 was challenged by environmental and public health groups, and the U.S. District Court for the Southern District of New York concluded that the EPA was required to list Pb under Section 108. *Natural Resources Defense Council v. EPA*, 411 F. Supp. 864 21 (S.D. N.Y. 1976), affirmed, 545 F.2d 320 (2d Cir. 1978). Accordingly, on April 8, 1976, the EPA published a notice in the *Federal Register* that Pb had been listed under Section 108 as a criteria pollutant (41 FR 14921, April 8, 1976) and on October 5, 1978, the EPA promulgated primary and secondary NAAQS for Pb under Section 109 of the Act (43 FR 46246, October 5, 1978). 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 particles (Pb-TSP), not to be exceeded by the maximum arithmetic mean concentration averaged over a calendar quarter. These standards were based on the 1977 *Air Quality Criteria for Lead* (USEPA, 1977).

The first 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; henceforth referred to as the 1986 CD), 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 recommendations for consideration by the Administrator (USEPA, 1990b). After consideration of the documents developed during the review and the significantly changed circumstances since Pb was listed in 1976, 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, such as bringing more areas into compliance with the existing Pb NAAQS (USEPA, 1991). The EPA continues this broad, multi-program, multimedia approach to reducing Pb exposures today, as described in section I.B above.

The last review of the Pb air quality criteria and standards was initiated in November 2004 (69 FR 64926, November 9, 2004); the agency's plans for preparation of the Air Quality Criteria Document and conduct of the NAAQS review were presented in documents completed in 2005 and early 2006 (USEPA, 2005a; USEPA 2006b).[9] The schedule for completion of the review was governed by a judicial order in *Missouri Coalition for the Environment v. EPA* (No. 4:04CV00660 ERW, September 14, 2005; and amended on April 29, 2008 and July 1, 2008).

The scientific assessment for the review is described in the 2006 *Air Quality Criteria for Lead* (USEPA, 2006a; henceforth referred to as the 2006 CD), multiple drafts of which received review by CASAC and the public. The EPA also conducted human exposure and health risk assessments and a pilot ecological risk assessment for the review, after consultation with

[9] In the current review, these two documents have been combined in the *Integrated Review Plan for the National Ambient Air Quality Standards for Lead* (USEPA, 2011a).

CASAC and receiving public comment on a draft analysis plan (USEPA, 2006c). Drafts of these quantitative assessments were reviewed by CASAC and the public. The pilot ecological risk assessment was released in December 2006 (ICF International, 2006), and the final health risk assessment report was released in November 2007 (USEPA, 2007a). The policy assessment, based on both of these assessments, air quality analyses and key evidence from the 2006 CD, was presented in the Staff Paper (USEPA, 2007b), a draft of which also received CASAC and public review. The final Staff Paper presented OAQPS staff's evaluation of the public health and welfare policy implications of the key studies and scientific information contained in the 2006 CD and presented and interpreted results from the quantitative risk/exposure analyses conducted for this review. Based on this evaluation, the Staff Paper presented OAQPS staff recommendations that the Administrator give consideration to substantially revising the primary and secondary standards to a range of levels at or below 0.2 $\mu g/m^3$.

Immediately subsequent to completion of the Staff Paper, the EPA issued an advance notice of proposed rulemaking (ANPR) that was signed by the Administrator on December 5, 2007 (72 FR 71488, December 17, 2007).[10] CASAC provided advice and recommendations to the Administrator with regard to the Pb NAAQS based on its review of the ANPR and the previously released final Staff Paper and risk assessment reports. In 2008, the proposed decision on revisions to the Pb NAAQS was signed on May 1 and published in the *Federal Register* on May 20 (73 FR 29184, May 20, 2008). Members of the public provided comments and the CASAC Pb Panel also provided advice and recommendations to the Administrator based on its review of the proposal notice. The final decision on revisions to the Pb NAAQS was signed on October 15, 2008, and published in the *Federal Register* on November 12, 2008 (73 FR 66964,

[10] The ANPR, one of the features of the revised NAAQS review process that EPA instituted in 2006, was replaced by reinstatement of the Policy Assessment prepared by OAQPS staff (previously termed the OAQPS Staff Paper) in 2009 (Jackson, 2009).

November 12, 2008).

The November 2008 notice described the EPA's decision to revise the primary and secondary NAAQS for Pb, as discussed more fully in section II.A.1 below. In consideration of the much-expanded health effects evidence on neurocognitive effects of Pb in children, the EPA substantially revised the primary standard from a level of 1.5 $\mu g/m^3$ to a level of 0.15 $\mu g/m^3$. The averaging time was revised to a rolling 3-month period with a maximum (not-to-be-exceeded) form, evaluated over a 3-year period. The indicator of Pb-TSP was retained, reflecting the evidence that Pb particles of all sizes pose health risks. The secondary standard was revised to be identical in all respects to the revised primary standard (40 CFR 50.16). Revisions to the NAAQS were accompanied by revisions to the data handling procedures, the treatment of exceptional events and the ambient air monitoring and reporting requirements, as well as emissions inventory reporting requirements. One aspect of the revised data handling requirements is the allowance for the use of monitoring for particulate matter with mean diameter below 10 microns (Pb-PM_{10}) for Pb NAAQS attainment purposes in certain limited circumstances at non-source-oriented sites. Subsequent to the 2008 rulemaking, additional revisions were made to the monitoring network requirements (75 FR 81126, December 27, 2010). Guidance on the approach for implementation of the new standards was described in the *Federal Register* notices for the proposed and final rules (73 FR 29184, May 20, 2008; 73 FR 66964, November 12, 2008).

On February 26, 2010, the EPA formally initiated its current review of the air quality criteria and standards for Pb, requesting the submission of recent scientific information on specified topics (75 FR 8934, February 26, 2010). Soon after this, the EPA held a workshop to discuss the policy-relevant science, which informed identification of key policy issues and

questions to frame the review of the Pb NAAQS (75 FR 20843, April 21, 2010). Drawing from the workshop discussions, the EPA developed the draft Integrated Review Plan (draft IRP, USEPA, 2011d). The draft IRP was made available in late March 2011 for consultation with the CASAC Pb Review Panel and for public comment (76 FR 20347, April 12, 2011). This document was discussed by the Panel via a publicly accessible teleconference consultation on May 5, 2011 (76 FR 21346, April 15, 2011; Frey, 2011a). The final *Integrated Review Plan for the National Ambient Air Quality Standards for Lead* (IRP), developed in consideration of the CASAC consultation and public comment, was released in November 2011 (USEPA, 2011a; 76 FR 76972, December 9, 2011).

In developing the Integrated Science Assessment (ISA) for this review, the EPA held a workshop in December 2010 to discuss with invited scientific experts preliminary draft materials and released the first external review draft of the document for CASAC review and public comment in May 2011 (USEPA, 2011e; 76 FR 26284, May 6, 2011; 76 FR 36120, June 21, 2011). The CASAC Pb Review Panel met at a public meeting on July 20, 2011, to review the draft ISA (76 FR 36120, June 21, 2011). The CASAC provided comments in a December 9, 2011, letter to the EPA Administrator (Frey and Samet, 2011). The second external review draft ISA was released for CASAC review and public comment in February 2012 (USEPA, 2012a; 77 FR 5247, February 2, 2012) and was the subject of a public meeting on April 10-11, 2012 (77 FR 14783, March 13, 2012). The CASAC provided comments in a July 20, 2012, letter (Samet and Frey, 2012). The third external review draft was released for CASAC review and public comment in November 2012 (USEPA, 2012b; 77 FR 70776, November 27, 2012) and was the subject of a public meeting on February 5-6, 2013 (78 FR 938, January 7, 2013). The CASAC provided comments in a June 4, 2013, letter (Frey, 2013a). The final ISA was released in late

June 2013 (USEPA, 2013a, henceforth referred to as the ISA; 78 FR 38318, June 26, 2013).

In June 2011, the EPA developed and released the *Risk and Exposure Assessment Planning Document* (REA Planning Document) for consultation with CASAC and public comment (USEPA, 2011b; 76 FR 58509). This document presented a critical evaluation of the information related to Pb human and ecological exposure and risk (e.g., data, modeling approaches) newly available in this review, with a focus on consideration of the extent to which new or substantially revised REAs for health and ecological risk might be warranted by the newly available evidence. Evaluation of the newly available information with regard to designing and implementing health and ecological REAs for this review led us to conclude that the currently available information did not provide a basis for developing new quantitative risk and exposure assessments that would have substantially improved utility for informing the agency's consideration of health and welfare effects and evaluation of the adequacy of the current primary and secondary standards, respectively (REA Planning Document, sections 2.3 and 3.3, respectively). The CASAC Pb Review Panel provided consultative advice on that document and its conclusions at a public meeting on July 21, 2011 (76 FR 36120, June 21, 2011; Frey, 2011b). Based on their consideration of the REA Planning Document analysis, the CASAC Pb Review Panel generally concurred with the conclusion that a new REA was not warranted in this review (Frey, 2011b; Frey, 2013b). In consideration of the conclusions reached in the REA Planning Document and CASAC's consultative advice, the EPA has not developed REAs for health and ecological risk for this review. Accordingly, we consider the risk assessment findings from the last review for human exposure and health risk (USEPA, 2007a, henceforth referred to as the 2007 REA) and ecological risk (ICF International, 2006; henceforth referred to as the 2006 REA) with regard to any appropriate further interpretation in light of the evidence newly

available in this review.

A draft of the Policy Assessment (PA) was released for public comment and review by CASAC in January 2013 (USEPA, 2013b; 77 FR 70776, November 27, 2012) and was the subject of a public meeting on February 5-6, 2013 (78 FR 938, January 7, 2013). Comments provided by the CASAC in a June 4, 2013 letter (Frey, 2013b), as well as public comments received on the draft PA were considered in preparing the final PA, which was released in May 2014 (USEPA, 2014; 79 FR 26751, May 9, 2014).

D. Multimedia, Multipathway Aspects of Lead

Since Pb distributes from air to other media and is persistent, our review of the NAAQS for Pb considers the protection provided against such effects associated both with exposures to Pb in ambient air and with exposures to Pb that makes its way into other media from ambient air. Additionally, in assessing the adequacy of protection afforded by the current NAAQS, we are mindful of the long history of greater and more widespread atmospheric emissions that occurred in previous years (both before and after establishment of the 1978 NAAQS) and that contributed to the Pb that exists in human populations and ecosystems today. Likewise, we also recognize the role of other, nonair sources of Pb now and in the past that also contribute to the Pb that exists in human populations and ecosystems today.

Lead emitted to ambient air is transported through the air and is also distributed from air to other media. This multimedia distribution of Pb emitted into ambient air (air-related Pb) contributes to multiple air-related pathways of human and ecosystem exposure (ISA, sections 3.1.1 and 3.7.1). Air-related pathways may also involve media other than air, including indoor and outdoor dust, soil, surface water and sediments, vegetation and biota. Air-related Pb exposure pathways for humans include inhalation of ambient air or ingestion of food, water or

other materials, including dust and soil, that have been contaminated through a pathway involving Pb deposition from ambient air (ISA, section 3.1.1.1). Ambient air inhalation pathways include both inhalation of air outdoors and inhalation of ambient air that has infiltrated into indoor environments. The air-related ingestion pathways occur as a result of Pb passing through the ambient air, being distributed to other environmental media and contributing to human exposures via contact with and ingestion of indoor and outdoor dusts, outdoor soil, food and drinking water.

Lead exposures via the various inhalation and ingestion air-related pathways may vary with regard to the time in which they respond to changes in air Pb concentrations. For example, exposures resulting from human exposure pathways most directly involving Pb in ambient air and exchanges of ambient air with indoor air (e.g., inhalation) can respond most quickly, while those for pathways involving exposure to Pb deposited from ambient air into the environment (e.g., diet) may be expected to respond more slowly. The extent of this will be influenced by the magnitude of change, as well as – for deposition-related pathways – the extent of prior deposition and environment characteristics influencing availability of prior deposited Pb.

Lead currently occurring in nonair media may also derive from sources other than ambient air (nonair Pb sources) (ISA, sections 2.3 and 3.7.1). For example, Pb in dust inside some houses or outdoors in some urban areas may derive from the common past usage of leaded paint, while Pb in drinking water may derive from the use of leaded pipe or solder in drinking water distribution systems (ISA, section 3.1.3.3). We also recognize the history of much greater air emissions of Pb in the past, such as that associated with leaded gasoline usage and higher industrial emissions which have left a legacy of Pb in other (nonair) media.

The relative importance of different pathways of human exposure to Pb, as well as the

relative contributions from Pb resulting from recent and historic air emissions and from nonair sources, vary across the U.S. population as a result of both extrinsic factors, such as a home's proximity to industrial Pb sources or its history of leaded paint usage, and intrinsic factors, such as a person's age and nutritional status (ISA, sections 5.1, 5.2, 5.2.1, 5.2.5 and 5.2.6). Thus, the relative contributions from specific pathways is situation specific (ISA, p. 1-11), although a predominant Pb exposure pathway for very young children is the incidental ingestion of indoor dust by hand-to-mouth activity (ISA, section 3.1.1.1). For adults, however, diet may be the primary Pb exposure pathway (2006 CD, section 3.4). Similarly, the relative importance of air-related and nonair-related Pb also varies with the relative magnitudes of exposure by those pathways, which may vary with different circumstances.

The distribution of Pb from ambient air to other environmental media also influences the exposure pathways for organisms in terrestrial and aquatic ecosystems. Exposure of terrestrial animals and vegetation to air-related Pb can occur by contact with ambient air or by contact with soil, water or food items that have been contaminated by Pb from ambient air (ISA, section 6.2). Transport of Pb into aquatic systems similarly provides for exposure of biota in those systems, and exposures may vary among systems as a result of differences in sources and levels of contamination, as well as characteristics of the systems themselves, such as salinity, pH and turbidity (ISA, section 2.3.2). In addition to Pb contributed by current atmospheric deposition, Pb may occur in aquatic systems as a result of nonair sources such as industrial discharges or mine-related drainage, of historical air Pb emissions (e.g., contributing to deposition to a water body or via runoff from soils near historical air sources) or combinations of different types of sources (e.g., resuspension of sediments contaminated by urban runoff and surface water discharges).

The persistence of Pb contributes an important temporal aspect to lead's environmental

pathways, and the time (or lag) associated with realization of the impact of air Pb concentrations on concentrations in other media can vary with the media (e.g., ISA, section 6.2.2). For example, exposure pathways most directly involving Pb in ambient air or surface waters can respond more quickly to changes in ambient air Pb concentrations while pathways involving exposure to Pb in soil or sediments generally respond more slowly. An additional influence on the response time for nonair media is the environmental presence of Pb associated with past, generally higher, air concentrations. For example, after a reduction in air Pb concentrations, the time needed for sediment or surface soil concentrations to indicate a response to reduced air Pb concentrations might be expected to be longer in areas of more substantial past contamination than in areas with lesser past contamination. Thus, considering the Pb concentrations occurring in nonair environmental media as a result of air quality conditions that meet the current NAAQS is a complexity of this review, as it also was, although to a lesser degree, with regard to the prior standard in the last review.

E. Air Quality Monitoring

Lead emitted to the air is predominantly in particulate form. Once emitted, particle-bound Pb can be transported long or short distances depending on particle size, which influences the amount of time spent in the aerosol phase. In general, larger particles tend to deposit more quickly, within shorter distances from emissions points, while smaller particles remain in aerosol phase and travel longer distances before depositing (ISA, section 1.2.1). Accordingly, airborne concentrations of Pb near sources are much higher (and the representation of larger particles generally greater) than at sites not directly influenced by sources (PA, Figure 2-11; ISA sections 2.3.1 and 2.5.3).

Ambient air monitoring data for Pb, in terms of Pb-TSP, Pb-PM_{10} or Pb in particulate

matter with mean diameter smaller than 2.5 microns (Pb-$PM_{2.5}$), are currently collected in several national networks. Monitoring conducted for purposes of Pb NAAQS surveillance is regulated to ensure accurate and comparable data for determining compliance with the NAAQS. In order to be used in NAAQS attainment designations, ambient Pb concentration data must be obtained using either the federal reference method (FRM) or a federal equivalent method (FEM). The FRMs for sample collection and analysis are specified in 40 CFR part 50. The procedures for approval of FRMs and FEMs are specified in 40 CFR part 53. In 2013, after consultation with CASAC's Ambient Air Monitoring and Methods Subcommittee, the EPA adopted a new FRM for Pb-TSP, based on inductively coupled plasma-mass spectrometry (78 FR 40000, July 3, 2013). The previous FRM was retained as an FEM, and existing FEMs were retained as well.

The Pb monitoring network design requirements (40 CFR part 58, Appendix D, paragraph 4.5) include two types of monitoring sites – source-oriented monitoring sites and non-source-oriented monitoring sites – as well as the collection of a year of Pb-TSP measurements at 15 specific airports. The indicator for the current Pb NAAQS is Pb-TSP, although in some situations,[11] ambient Pb-PM_{10} concentrations may be used in judging nonattainment. Currently, approximately 260 Pb-TSP monitors are in operation; these are a mixture of source- and non-source-oriented monitors.

Since the phase-out of Pb in on-road gasoline, Pb is widely recognized as a source-oriented air pollutant. Variability in air Pb concentrations is highest in areas including a Pb source, "with high concentrations downwind of the sources and low concentration at areas far from sources" (ISA, p. 2-92). The current requirements for source-oriented monitoring include

[11] The Pb-PM_{10} measurements may be used for NAAQS monitoring as an alternative to Pb-TSP measurements in certain conditions defined in 40 CFR part 58, Appendix C, section 2.10.1.2. These conditions include where Pb concentrations are not expected to equal or exceed 0.10 $\mu g/m^3$ as an arithmetic 3-month mean and where the source of Pb emissions is expected to emit a substantial majority of its Pb in the size fraction captured by PM_{10} monitors.

placement of monitor sites near sources of air Pb emissions which are expected to or have been shown to contribute to ambient air Pb concentrations in excess of the NAAQS. At a minimum, there must be one source-oriented site located to measure the maximum Pb concentration in ambient air resulting from each non-airport Pb source which emits 0.50 or more tons of Pb per year and from each airport which emits 1.0 or more tons of Pb per year.[12] The EPA Regional Administrators may require additional monitoring beyond the minimum requirements where the likelihood of Pb air quality violations is significant. Such locations may include those near additional industrial Pb sources, recently closed industrial sources and other sources of resuspended Pb dust, as well as airports where piston-engine aircraft emit Pb associated with combustion of leaded aviation fuel (40 CFR part 58, Appendix D, section 4.5(c)). A single year of monitoring was also required near 15 specific airports[13] in order to gather additional information on the likelihood of NAAQS exceedances due to the combustion of leaded aviation gasoline (75 FR 81126, December 27, 2010; 40 CFR part 58, Appendix D, 4.5(a)(iii)). These airport monitoring data along with other data gathering and analyses will inform the EPA's ongoing investigation into the potential for Pb emissions from piston-engine aircraft to cause or contribute to air pollution that may reasonably be anticipated to endanger public health or welfare. This investigation is occurring under section 231 of the CAA, separate from the Pb NAAQS review. As a whole, the various data gathering and analyses are expected to improve

[12] The Regional Administrator may waive this requirement for monitoring near Pb sources if the state or, where appropriate, local agency can demonstrate the Pb source will not contribute to a maximum 3-month average Pb concentration in ambient air in excess of 50 percent of the NAAQS level based on historical monitoring data, modeling, or other means (40 CFR part 58, Appendix D, section 4.5(a)(ii)).

[13] These airports were selected based on three criteria: annual Pb inventory between 0.5 ton/year and 1.0 ton/year, ambient air within 150 meters of the location of maximum emissions (e.g., the end of the runway or run-up location), and airport configuration and meteorological scenario that leads to a greater frequency of operations from one runway. These criteria are expected, collectively, to identify airports with the highest potential to have ambient air Pb concentrations approaching or exceeding the Pb NAAQS (75 FR 81126).

our understanding of Pb concentrations in ambient air near airports and conditions influencing these concentrations.

Monitoring agencies are also required, under 40 CFR part 58, Appendix D, to conduct non-source-oriented Pb monitoring at the NCore sites[14] required in metropolitan areas with a population of 500,000 or more (as defined by the U.S. Census Bureau).[15] Either Pb-TSP or Pb-PM_{10} monitoring may be performed at these sites. Currently, all 50 NCore Pb sites are operational and measuring Pb concentrations, with 28 measuring Pb in TSP and 24 measuring Pb in PM_{10} (2 sites are measuring both Pb in TSP and Pb in PM_{10}). In a separate action addressing a range of issues related to monitoring requirements for criteria pollutants, the EPA is proposing to remove the requirement for Pb monitoring at NCore sites (79 FR 54395, September 11, 2014). This change is being proposed in consideration of current information indicating concentrations at these sites to be well below the Pb NAAQS and of the presence of other monitoring networks that provide information on Pb concentrations in urban areas not directly impacted by Pb sources. The data available for these sites indicate maximum 3-month average concentrations (of Pb-PM_{10} or Pb-TSP) well below the level of the Pb NAAQS, with the vast majority of sites showing concentrations less than 0.01 $\mu g/m^3$. Additionally, other monitoring networks provide data on Pb in PM_{10} or $PM_{2.5}$, at non-source-oriented urban, and some rural, sites. These include the National Air Toxics Trends Stations for PM_{10} and the Chemical Speciation Network for $PM_{2.5}$. Data on Pb in $PM_{2.5}$ are also provided at the rural sites of the Interagency Monitoring of Protected Visual Environments network.

[14] The NCore network, that formally began in January 2011, is a subset of the state and local air monitoring stations network that is intended to meet multiple monitoring objectives (e.g., long-term trends analysis, model evaluation, health and ecosystem studies, as well as NAAQS compliance). The complete NCore network consists of 63 urban and 15 rural stations, with each state containing at least one NCore station; 46 of the states plus Washington, DC and Puerto Rico have at least one urban station.

[15] http://www.census.gov/population/www/metroareas/metroarea.html.

The long-term record of Pb monitoring data documents the dramatic decline in atmospheric Pb concentrations that has occurred since the 1970s in response to reduced emissions (PA, Figures 2-1 and 2-7). Currently, the highest concentrations occur near some metals industries where some individual locations have concentrations that exceed the NAAQS (PA, Figure 2-10). Concentrations at non-source-oriented monitoring sites are much lower than those at source-oriented sites and well below the standard (PA, Figure 2-11).

II. Rationale for Proposed Decision on the Primary Standard

This section presents the rationale for the Administrator's proposed decision to retain the existing Pb primary standard. As discussed more fully below, this rationale is based on a thorough review, in the ISA, of the latest scientific information, generally published through September 2011,[16] on human health effects associated with Pb and pertaining to the presence of Pb in the ambient air. This proposal also takes into account: (1) the PA's staff assessments of the most policy-relevant information in the ISA and staff analyses of air quality, human exposure and health risks, upon which staff conclusions regarding appropriate considerations in this review are based; (2) CASAC advice and recommendations, as reflected in discussions of drafts of the ISA and PA at public meetings, in separate written comments, and in CASAC's letters to the Administrator; and (3) public comments received during the development of these documents, either in connection with CASAC meetings or separately.

In presenting the rationale and its foundations, section II.A provides background on the general approach for review of the primary NAAQS for Pb, including a summary of the approach used in the last review (section II.A.1) and the general approach for the current review

[16] In addition to the review's opening "call for information" (75 FR 8934), "literature searches were conducted routinely to identify studies published since the last review, focusing on studies published from 2006 (close of the previous scientific assessment) through September 2011," and references "that were considered for inclusion or actually cited in this ISA can be found at http://hero.epa.gov/lead" (ISA, p. 1-2).

(section II.A.2). Sections II.B and II.C summarize the body of evidence supporting this rationale, focusing on consideration of key policy-relevant questions, and section II.D summarizes the exposure/risk information for this review. Section II.E presents the Administrator's proposed conclusions on adequacy of the current standard, drawing on both evidence-based and exposure/risk-based considerations (sections II.E.1 and II.E.2), and advice from CASAC (section II.E.3).

A. General Approach

The past and current approaches described below are both based, most fundamentally, on using the EPA's assessment of the current scientific evidence and associated quantitative analyses to inform the Administrator's judgment regarding a primary standard for Pb that protects public health with an adequate margin of safety. We note that in drawing conclusions with regard to the primary standard, the final decision on the adequacy of the current standard is largely a public health policy judgment to be made by the Administrator. The Administrator's final decision must draw upon scientific information and analyses about health effects, population exposure and risks, as well as judgments about how to consider the range and magnitude of uncertainties that are inherent in the scientific evidence and analyses. Our approach to informing these judgments, discussed more fully below, is based on the recognition that the available health effects evidence generally reflects a continuum, consisting of 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 the EPA and the courts have historically interpreted the Act. These provisions require the Administrator to establish primary standards that, in the judgment of the Administrator, 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.[17] The four basic elements of the NAAQS (indicator, averaging time, level, and form) are considered collectively in evaluating the health protection afforded by the current standard.

1. Approach in the Last Review

The last review of the NAAQS for Pb was completed in 2008 (73 FR 66964, November 12, 2008). The 2008 decision to substantially revise the primary standard was based on the extensive body of scientific evidence published over almost three decades, from the time the standard was originally set in 1978 through 2005-2006. In so doing, the 2008 decision considered the body of evidence as assessed in the 2006 CD (USEPA, 2006a), as well as the 2007 Staff Paper assessment of the policy-relevant information contained in the CD and the quantitative risk/exposure assessment (USEPA, 2007a, 2007b), the advice and recommendations of CASAC (Henderson 2007a, 2007b, 2008a, 2008b), and public comment. While recognizing that Pb has been demonstrated to exert "a broad array of deleterious effects on multiple organ systems," the review focused on the effects most pertinent to ambient air exposures, which given ambient air Pb reductions over the past 30 years, are those associated with relatively lower exposures and associated blood Pb levels (73 FR 66975, November 12, 2008). In so doing, the EPA recognized the general consensus that the developing nervous system in children is among the most sensitive health endpoints associated with Pb exposure, if not the most sensitive one.

[17] The at-risk population groups identified in a NAAQS review may include low-income or minority groups. Where low-income/minority groups are among the at-risk populations, the rulemaking decision will be based on providing protection for these and other at-risk populations and lifestages (e.g., children, older adults, persons with pre-existing heart and lung disease). To the extent that low-income/minority groups are not among the at-risk populations identified in the ISA, a decision based on providing protection of the at-risk lifestages and populations would be expected to provide protection for the low-income/minority groups.

Thus, primary attention was given to consideration of nervous system effects, including neurocognitive and neurobehavioral effects, in children (73 FR 66976, November 12, 2008). The body of evidence included associations of such effects in study populations of variously aged children with mean blood Pb levels below 10 µg/dL, extending from 8 down to 2 µg/dL (73 FR 66976, November 12, 2008). The public health implications of effects of air-related Pb on cognitive function (e.g., IQ) in young children were given particular focus in the review.

The conclusions reached by the Administrator in the last review were based primarily on the scientific evidence, with the risk- and exposure-based information providing support for various aspects of the decision. In reaching his conclusion on the adequacy of the then-current standard, which was set in 1978, the Administrator placed primary consideration on the large body of scientific evidence available in the review including significant new evidence concerning effects at blood Pb concentrations substantially below those identified when the standard was initially set (73 FR 66987, November 12, 2008; 43 FR 46246, October 5, 1978). Given particular attention was the robust evidence of neurotoxic effects of Pb exposure in children, recognizing: (1) that while blood Pb levels in U.S. children had decreased notably since the late 1970s, newer epidemiological studies had investigated and reported associations of effects on the neurodevelopment of children with those more recent lower blood Pb levels and (2) that the toxicological evidence included extensive experimental laboratory animal evidence substantiating well the plausibility of the epidemiological findings observed in human children and expanding our understanding of likely mechanisms underlying the neurotoxic effects (73 FR 66987, November 12, 2008). Additionally, within the range of blood Pb levels investigated in the available evidence base, a threshold level for neurocognitive effects was not identified (73 FR 66984, November 12, 2008; 2006 CD, p. 8-67). Further, the evidence indicated a steeper

concentration-response (C-R) relationship for effects on cognitive function at those lower blood Pb levels than at higher blood Pb levels that were more common in the past, "indicating the potential for greater incremental impact associated with exposure at these lower levels" (73 FR 66987, November 12, 2008). As at the time when the standard was initially set in 1978, the health effects evidence and exposure/risk assessment available in the last review supported the conclusion that air-related Pb exposure pathways contribute to blood Pb levels in young children by inhalation and ingestion (73 FR 66987, November 12, 2008). The available information in the last review also indicated, however, a likely greater change in blood Pb per unit of air Pb than was estimated when the standard was initially set (73 FR 66987, November 12, 2008).

In the Administrator's decision on the adequacy of the 1978 standard, the Administrator considered the evidence using a very specifically defined framework, referred to as an air-related IQ loss evidence-based framework. This framework integrates evidence for the relationship between Pb in air and Pb in young children's blood with evidence for the relationship between Pb in young children's blood and IQ loss (73 FR 66987, November 12, 2008). This evidence-based approach considers air-related effects on neurocognitive function (using the quantitative metric of IQ loss) associated with exposure in those areas with elevated air concentrations equal to potential alternative levels for the Pb standard. In simplest terms, the framework focuses on children exposed to air-related Pb in those areas with elevated air Pb concentrations equal to specific potential standard levels, providing for estimation of a mean air-related IQ decrement for young children in the high end of the national distribution of air-related exposures. Thus, the conceptual context for the framework is that it provides estimates of air-related IQ loss for the subset of U.S. children living in close proximity to air Pb sources that contribute to such elevated air Pb concentrations. In such cases, when a standard of a particular level is just met at a monitor

sited to record the highest source-oriented concentration in an area, the large majority of children in the larger surrounding area would likely experience exposures to concentrations well below that level.

The two primary inputs to the evidence-based air-related IQ loss framework are air-to-blood ratios and C-R functions for the relationship between blood Pb and IQ response in young children. Additionally taken into consideration in applying and drawing conclusions from the framework were the uncertainties inherent in these inputs. Application of the framework also entailed consideration of an appropriate level of protection from air-related IQ loss to be used in conjunction with the framework. The framework estimates of mean air-related IQ loss are derived through multiplication of the following factors: standard level ($\mu g/m^3$), air-to-blood ratio (albeit in terms of $\mu g/dL$ blood Pb per $\mu g/m^3$ air concentration), and slope for the C-R function in terms of points IQ decrement per $\mu g/dL$ blood Pb.

Based on the application of the air-related IQ loss framework to the evidence, the Administrator concluded that, for exposures projected for air Pb concentrations at the level of the 1978 standard, the quantitative estimates of IQ loss associated with air-related Pb indicated risk of a magnitude that, in his judgment, was significant from a public health perspective, and that the evidence-based framework supported a conclusion that the 1978 standard did not protect public health with an adequate margin of safety (73 FR 66987, November 12, 2008). The Administrator further concluded that the evidence indicated the need for a substantially lower standard level to provide increased public health protection, especially for at-risk groups (most notably children), against an array of effects, most importantly including effects on the developing nervous system (73 FR 66987, November 12, 2008). In addition to giving primary consideration to the much expanded evidence base since the standard was set, the Administrator

also took into consideration the exposure/risk assessments. In so doing, he observed that, while taking into consideration their inherent uncertainties and limitations, the quantitative estimates of IQ loss associated with air-related Pb in air quality scenarios just meeting the then-current standard also indicated risk of a magnitude that, in his judgment, was significant from a public health perspective. Thus, the Administrator concluded the exposure/risk estimates provided additional support to the evidence-based conclusion that the standard needed revision (73 FR 66987, November 12, 2008).

In considering appropriate revisions to the prior standard in the review completed in 2008, each of the four basic elements of the NAAQS (indicator, averaging time, form and level) was evaluated. The rationale for decisions on those elements is summarized below.

With regard to indicator, consideration was given to replacing Pb-TSP with Pb-PM_{10}. The EPA recognized, however, that Pb in all particle sizes contributes to Pb in blood and associated health effects, additionally noting that the difference in particulate Pb captured by TSP and PM_{10} monitors may be on the order of a factor of two in some areas (73 FR 66991, November 12, 2008). Further, the Administrator recognized uncertainty with regard to whether a Pb-PM_{10}-based standard would also effectively control ultra-coarse[18] Pb particles, which may have a greater presence in areas near sources where Pb concentrations are highest (73 FR 66991, November 12, 2008). The Administrator decided to retain Pb-TSP as the indicator to provide sufficient public health protection from the range of particle sizes of ambient air Pb, including ultra-coarse particles (73 FR 66991, November 12, 2008). Additionally, a role was provided for Pb-PM_{10} in the monitoring required for a Pb-TSP standard (73 FR 66991, November 12, 2008)

[18] The term "ultra-coarse" refers to particles collected by a TSP sampler but not by a PM_{10} sampler. This terminology is consistent with the traditional usage of ''fine'' to refer to particles collected by a $PM_{2.5}$ sampler, and ''coarse'' to refer to particles collected by a PM_{10} sampler but not by a $PM_{2.5}$ sampler, recognizing that there will be some overlap in the particle sizes in the three types of collected material.

based on the conclusion that use of Pb-PM_{10} measurements at sites not influenced by sources of ultra-coarse Pb, and where Pb concentrations are well below the standard, would take advantage of the increased precision of these measurements and decreased spatial variation of Pb-PM_{10} concentrations, without raising the same concerns over a lack of protection against health risks from all particulate Pb emitted to the ambient air that support retention of Pb-TSP as the indicator (versus revision to Pb-PM_{10}) (73 FR 66991, November 12, 2008). Accordingly, allowance was made for the use of Pb-PM_{10} monitoring for Pb NAAQS attainment purposes in certain limited circumstances, at non-source-oriented sites, where the Pb concentrations are expected to be substantially below the standard and ultra-coarse particles are not expected to be present (73 FR 66991, November 12, 2008).

With regard to averaging time and form for the revised standard, consideration was given to a monthly averaging time, with a form of second maximum, and to 3-month and calendar quarter averaging times, with not-to-be exceeded forms. While the Administrator recognized that there were some factors that might imply support for a period as short as a month for averaging time, he also noted other factors supporting use of a longer time. He additionally took note of the complexity inherent in this consideration for the primary Pb standard, which is greater than in the case of other criteria pollutants due to the multimedia nature of Pb and its multiple pathways of human exposure. In this situation for Pb, the Administrator emphasized the importance of considering all of the relevant factors, both those pertaining to the human physiological response to changes in Pb exposures and those pertaining to the response of air-related Pb exposure pathways to changes in airborne Pb, in an integrated manner.

As discussed further in the PA, the evidence on human physiological response to changes in Pb exposure available in the last review indicated that children's blood Pb levels respond

quickly to increased Pb exposure, particularly during the time of leaded gasoline usage but likely with lessened immediacy since that time as children's exposure pathways have changed (PA, section 4.1.1.2). The Administrator also recognized limitations and uncertainties in the evidence and variability with regard to the information regarding the response time of indoor dust Pb to ambient airborne Pb. In consideration of the uncertainty associated with the evidence, the Administrator noted that the two changes in form for the standard (to a rolling 3-month average and to providing equal weighting to each month in deriving the 3-month average) both afford greater weight to each individual month than did the calendar quarter form of the 1978 standard, tending to control both the likelihood that any month will exceed the level of the standard and the magnitude of any such exceedance. Thus, based on an integrated consideration of the range of relevant factors, the averaging time was revised to a rolling 3-month period with a maximum (not-to-be-exceeded) form, evaluated over a 3-year period. As compared to the previous averaging time and form of calendar quarter (not-to-be exceeded), this revision was considered to be more scientifically appropriate and more health protective (73 FR 66996, November 12, 2008). The rolling average gives equal weight to all 3-month periods, and the new calculation method gives equal weight to each month within each 3-month period (73 FR 66996, November 12, 2008). Further, the rolling average yields twelve 3-month averages each year to be compared to the NAAQS versus four averages in each year for the block calendar quarters pertaining to the previous standard (73 FR 66996, November 12, 2008).

Lastly, based on the body of scientific evidence and information available, as well as CASAC recommendations and public comment, the Administrator decided on a standard level that, in combination with the specified choice of indicator, averaging time, and form, he judged requisite to protect public health, including the health of sensitive groups, with an adequate

margin of safety (73 FR 67006, November 12, 2008). In reaching the decision on level for the revised standard, the Administrator considered as a useful guide the evidence-based framework developed in that review. As described above, that framework integrates evidence for the relationship between Pb in air and Pb in children's blood and the relationship between Pb in children's blood and IQ loss. Application of the air-related IQ loss evidence-based framework was recognized, however, to provide "no evidence- or risk-based bright line that indicates a single appropriate level" for the standard (73 FR 67006, November 12, 2008). Rather, the framework was seen as a useful guide for consideration of health risks from exposure to ambient levels of Pb in the air, in the context of a specified averaging time and form, with regard to the Administrator's decision on a level for a revised NAAQS that provides public health protection that is sufficient but not more than necessary under the Act (73 FR 67004, November 12, 2008).

As noted above, use of the evidence-based air-related IQ loss framework to inform selection of a standard level involved consideration of the evidence with regard to two input parameters. The two input parameters are an air-to-blood ratio and a C-R function for population IQ response associated with blood Pb level (73 FR 67004, November 12, 2008). The evidence at the time of the last review indicated a broad range of air-to-blood ratio estimates,[19] each with limitations and associated uncertainties. Based on the then-available evidence, the Administrator concluded that 1:5 to 1:10 represented a reasonable range to consider and identified 1:7 as a generally central value on which to focus (73 FR 67004, November 12, 2008). With regard to C-R functions, in light of the evidence of nonlinearity and of steeper slopes at lower blood Pb levels, the Administrator concluded it was appropriate to focus on C-R analyses based on blood

[19] The term "air-to-blood ratio" describes the increase in blood Pb (in µg/dL) estimated to be associated with each unit increase of air Pb (in $\mu g/m^3$). Ratios are presented in the form of 1:x, with the 1 representing air Pb (in $\mu g/m^3$) and x representing blood Pb (in µg/dL). Description of ratios as higher or lower refers to the values for x (i.e., the change in blood Pb per unit of air Pb).

Pb levels that most closely reflected the then-current population of children in the U.S.,[20] recognizing the EPA's identification of four such analyses and giving weight to the central estimate or median of the resultant C-R functions (73 FR 67003, November 12, 2008, Table 3; 73 FR 67004, November 12, 2008). The median estimate for the four C-R slopes of -1.75 IQ points decrement per μg/dL blood Pb was selected for use with the framework. With the framework, potential alternative standard levels (μg/m^3) are multiplied by estimates of air-to-blood ratio (μg/dL blood Pb per μg/m^3 air Pb) and the median slope for the C-R function (points IQ decrement per μg/dL blood Pb), yielding estimates of a mean air-related IQ decrement for a specific subset of young children (i.e., those children exposed to air-related Pb in areas with elevated air Pb concentrations equal to specified alternative levels). As such, the application of the framework yields estimates for the mean air-related IQ decrements of the subset of children expected to experience air-related Pb exposures at the high end of the distribution of such exposures. The associated mean IQ loss estimate is the average for this highly exposed subset and is not the average air-related IQ loss projected for the entire U.S. population of children. Uncertainties and limitations were recognized in the use of the framework and in the resultant estimates (73 FR 67000, November 12, 2008).

In considering the use of the evidence-based air-related IQ loss framework to inform his judgment as to the appropriate degree of public health protection that should be afforded by the NAAQS to provide requisite protection against risk of neurocognitive effects in sensitive populations, such as IQ loss in children, the Administrator recognized in the 2008 review that there were no commonly accepted guidelines or criteria within the public health community that

[20] The geometric mean blood Pb level for U.S. children aged 5 years and below, reported for NHANES in 2003-04 (the most recent years for which such an estimate was available at the time of the 2008 decision) was 1.8 μg/dL and the 5th and 95th percentiles were 0.7 μg/dL and 5.1 μg/dL, respectively (73 FR 67002).

would provide a clear basis for such a judgment. During the 2008 review, CASAC commented regarding the significance from a public health perspective of a 1-2 point IQ loss in the entire population of children and along with some commenters, emphasized that the NAAQS should prevent air-related IQ loss of a significant magnitude, such as on the order of 1-2 IQ points, in all but a small percentile of the population. Similarly, the Administrator stated that "ideally air-related (as well as other) exposures to environmental Pb would be reduced to the point that no IQ impact in children would occur" (73 FR 66998, November 12, 2008). The Administrator further recognized that, in the case of setting a NAAQS, he was required to make a judgment as to what degree of protection is requisite to protect public health with an adequate margin of safety (73 FR 66998, November 12, 2008). The NAAQS must be sufficient but not more stringent than necessary to achieve that result, and the Act does not require a zero-risk standard (73 FR 66998, November 12, 2008). The Administrator additionally recognized that the evidence-based air-related IQ loss framework did not provide estimates pertaining to the U.S. population of children as a whole. Rather, the framework provided estimates (with associated uncertainties and limitations) for the mean of a subset of that population, the subset of children assumed to be exposed to the level of the standard. As described in the final decision "[t]he framework in effect focuses on the sensitive subpopulation that is the group of children living near sources and more likely to be exposed at the level of the standard" (73 FR 67000, November 12, 2008). As further noted in the final decision (73 FR 67000, November 12, 2008):

> *EPA is unable to quantify the percentile of the U.S. population of children that corresponds to the mean of this sensitive subpopulation. Nor is EPA confident in its ability to develop quantified estimates of air-related IQ loss for higher percentiles than the mean of this subpopulation. EPA expects that the mean of this subpopulation represents a high, but not quantifiable, percentile of the U.S. population of children. As a result, EPA expects that a standard based on consideration of this framework would provide the same or greater protection*

from estimated air-related IQ loss for a high, albeit unquantifiable, percentage of the entire population of U.S. children.

In reaching a judgment as to the appropriate degree of protection, the Administrator considered advice and recommendations from CASAC and public comments and recognized the uncertainties in the health effects evidence and related information as well as the role of, and context for, a selected air-related IQ loss in the application of the framework, as described above. Based on these considerations, the Administrator identified an air-related IQ loss of 2 points for use with the framework, as a tool for considering the evidence with regard to the level for the standard (73 FR 67005, November 12, 2008). In so doing, the Administrator was not determining that such an IQ decrement value was appropriate in other contexts (73 FR 67005, November 12, 2008). Given the various uncertainties associated with the framework and the scientific evidence base, and the focus of the framework on the sensitive subpopulation of children that are more highly exposed to air-related Pb, a standard level selected in this way, in combination with the selected averaging time and form, was expected to significantly reduce and limit for a high percentage of U.S. children the risk of experiencing an air-related IQ loss of that magnitude (73 FR 67005, November 12, 2008). At the standard level of 0.15 $\mu g/m^3$, with the combination of the generally central estimate of air-to-blood ratio of 1:7 and the median of the four C-R functions (-1.75 IQ point decrement per μg/dL blood Pb), the framework estimates of air-related IQ loss were below 2 IQ points (73 FR 67005, November 12, 2008, Table 4).

In reaching the decision in 2008 on a level for the revised standard, the Administrator also considered the results of the quantitative risk assessment to provide a useful perspective on risk from air-related Pb. In light of important uncertainties and limitations for purposes of evaluating potential standard levels, however, the Administrator placed less weight on the risk estimates than on the evidence-based assessment. Nevertheless, in recognition of the general

comparability of quantitative risk estimates for the case studies considered most conceptually similar to the scenario represented by the evidence-based framework, he judged the quantitative risk estimates to be "roughly consistent with and generally supportive" of the evidence-based framework estimates (73 FR 67006, November 12, 2008).

Based on consideration of the entire body of evidence and information available in the review, as well as the recommendations of CASAC and public comments, the Administrator decided that a level for the primary Pb standard of 0.15 $\mu g/m^3$, in combination with the specified choice of indicator, averaging time and form, was requisite to protect public health, including the health of sensitive groups, with an adequate margin of safety (73 FR 67006, November 12, 2008). In reaching decisions on level as well as the other elements of the revised standard, the Administrator took note of the complexity associated with consideration of health effects caused by different ambient air concentrations of Pb and with uncertainties with regard to the relationships between air concentrations, exposures, and health effects. For example, selection of a maximum, not to be exceeded, form in conjunction with a rolling 3-month averaging time over a 3-year span was expected to have the effect that the at-risk population of children would be exposed below the standard most of the time (73 FR 67005, November 12, 2008). The Administrator additionally considered the provision of an adequate margin of safety in making decisions on each of the elements of the standard, including, for example "selection of TSP as the indicator and the rejection of the use of PM10 scaling factors; selection of a maximum, not to be exceeded form, in conjunction with a 3-month averaging time that employs a rolling average, with the requirement that each month in the 3-month period be weighted equally (rather than being averaged by individual data) and that a 3-year span be used for comparison to the standard; and the use of a range of inputs for the evidence-based framework, that includes a focus on

higher air-to-blood ratios than the lowest ratio considered to be supportable, and steeper rather than shallower C-R functions, and the consideration of these inputs in selection of 0.15 $\mu g/m^3$ as the level of the standard" (73 FR 67007, November 12, 2008).

The Administrator additionally noted that a standard with this level would reduce the risk of a variety of health effects associated with exposure to Pb, including effects indicated in the epidemiological studies at lower blood Pb levels, particularly including neurological effects in children, and the potential for cardiovascular and renal effects in adults (73 FR 67006, November 12, 2008). The Administrator additionally considered higher and lower levels for the standard, concluding that a level of 0.15 $\mu g/m^3$ provided for a standard that was neither more or less stringent than necessary for this purpose, recognizing that the Act does not require that primary standards be set at a zero-risk level, but rather at a level that reduces risk sufficiently so as to protect public health with an adequate margin of safety (73 FR 67007, November 12, 2008). For example, the Administrator additionally considered potential public health protection provided by standard levels above 0.15 $\mu g/m^3$, which he concluded were insufficient to protect public health with an adequate margin of safety. The Administrator also noted that in light of all of the evidence, including the evidence-based framework, the degree of public health protection likely afforded by standard levels below 0.15 $\mu g/m^3$ would be greater than what is necessary to protect public safety with an adequate margin of safety.

The Administrator concluded, based on review of all of the evidence (including the evidence-based framework), that when taken as a whole the selected standard, including the indicator, averaging time, form, and level, would be "sufficient but not more than necessary to protect public health, including the health of sensitive subpopulations, with an adequate margin of safety" (73 FR 67007, November 12, 2008).

2. Approach for the Current Review

The approach in this review of the current primary standard takes into consideration the approach used in the last Pb NAAQS review, addressing key policy-relevant questions in light of currently available scientific and technical information. To evaluate whether it is appropriate to consider retaining the current primary Pb standard, or whether consideration of revision is appropriate, the EPA has adopted an approach in this review that builds upon the general approach used in the last review and reflects the broader body of evidence and information now available. As summarized above, the Administrator's decisions in the prior review were based on an integration of information on health effects associated with exposure to Pb with that on relationships between ambient air Pb and blood Pb; expert judgments on the adversity and public health significance of key health effects; and policy judgments as to when the standard is requisite to protect public health with an adequate margin of safety. These considerations were informed by air quality and related analyses, quantitative exposure and risk assessments, and qualitative assessment of impacts that could not be quantified.

Similarly in this review, as described in the PA, we draw on the current evidence and quantitative assessments of exposure pertaining to the public health risk of Pb in ambient air. In considering the scientific and technical information here as in the PA, we consider both the information available at the time of the last review and information newly available since the last review, including most particularly that which has been critically analyzed and characterized in the current ISA. We additionally consider the quantitative exposure/risk assessments from the last review that estimated Pb-related IQ decrements associated with different air quality conditions in simulated at-risk populations in multiple case studies (PA, section 3.4; 2007 REA). The evidence-based discussions presented below draw upon evidence from epidemiological

studies and experimental animal studies evaluating health effects related to exposures to Pb, as discussed in the ISA. The exposure/risk-based discussions have drawn from the quantitative health risk analyses for Pb performed in the last Pb NAAQS review in light of the currently available evidence (PA, section 3.4; 2007 REA; REA Planning Document). Sections II.B through II.D below summarize the current health effects and exposure/risk information with a focus on the specific policy-relevant questions identified for these categories of information in the PA (PA, chapter 3).

B. Health Effects Information

1. Array of Effects

Lead has been demonstrated to exert a broad array of deleterious effects on multiple organ systems as described in the assessment of the evidence available in this review and consistent with conclusions of past CDs (ISA, section 1.6; 2006 CD, section 8.4.1). A sizeable number of studies on Pb health effects are newly available in this review and are critically assessed in the ISA as part of the full body of evidence. The newly available evidence reaffirms conclusions on the broad array of effects recognized for Pb in the last review (see ISA, section 1.10).[21] Consistent with those conclusions, in the context of pollutant exposures considered

[21] Since the last Pb NAAQS review, the ISAs which have replaced CDs in documenting each review of the scientific evidence (or air quality criteria) employ a systematic framework for weighing the evidence and describing associated conclusions with regard to causality using established descriptors: "causal" relationship with relevant exposure, "likely" to be a causal relationship, evidence is "suggestive" of a causal relationship, "inadequate" evidence to infer a causal relationship, and "not likely" to be a causal relationship (ISA, Preamble).

relevant to the Pb NAAQS review,[22] the ISA determines that causal relationships[23] exist for Pb with effects on the nervous system in children (cognitive function decrements and the group of externalizing behaviors comprising attention, impulsivity and hyperactivity), the hematological system (altered heme synthesis and decreased red blood cell survival and function), and the cardiovascular system (hypertension and coronary heart disease), and on reproduction and development (postnatal development and male reproductive function) (ISA, Table 1-2). Additionally, the ISA describes relationships between Pb and effects on the nervous system in adults, on immune system function and with cancer[24] as likely to be causal[25] (ISA, Table 1-2, sections 1.6.4 and 1.6.7).

In some categories of health effects, there is newly available evidence regarding some aspects of the effects described in the last review or that strengthens our conclusions regarding aspects of Pb toxicity on a particular physiological system. Among the nervous system effects of

[22] In drawing judgments regarding causality for the criteria air pollutants, the ISA places emphasis "on evidence of effects at doses (e.g., blood Pb concentration) or exposures (e.g., air concentrations) that are relevant to, or somewhat above, those currently experienced by the population. The extent to which studies of higher concentrations are considered varies … but generally includes those with doses or exposures in the range of one to two orders of magnitude above current or ambient conditions. Studies that use higher doses or exposures may also be considered …[t]hus, a causality determination is based on weight of evidence evaluation …, focusing on the evidence from exposures or doses generally ranging from current levels to one or two orders of magnitude above current levels" (ISA, pp. lx-lxi).

[23] In determining a causal relationship to exist for Pb with specific health effects, the EPA concludes that "[e]vidence is sufficient to conclude that there is a causal relationship with relevant pollutant exposures (i.e., doses or exposures generally within one to two orders of magnitude of current levels)" (ISA, p. lxii).

[24] The EPA concludes that a causal relationship is likely to exist between Pb exposure and cancer, based primarily on consistent, strong evidence from experimental animal studies, but inconsistent epidemiological evidence (ISA, section 4.10.5). Lead has also 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 (ISA, section 4.10).

[25] In determining that there is likely to be a causal relationship for Pb with specific health effects, the EPA has concluded that "[e]vidence is sufficient to conclude that a causal relationship is likely to exist with relevant pollutant exposures, but important uncertainties remain" (ISA, p. lxii).

Pb, the newly available evidence is consistent with conclusions in the previous review which recognized that "[t]he neurotoxic effects of Pb exposure are among those most studied and most extensively documented among human population groups" (2006 CD, p. 8-25) and took note of the diversity of studies in which such effects of Pb exposure early in development (from fetal to postnatal childhood periods) have been observed (2006 CD, p. E-9). Nervous system effects that receive prominence in the current review, as in previous reviews, include those affecting cognitive function and behavior in children (ISA, section 4.3), with conclusions that are consistent with findings of the last review.

Across the broad array of Pb effects for systems and processes other than the nervous system, the evidence base has been augmented with additional epidemiological investigations in a number of areas, including developmental outcomes, such as puberty onset, and adult outcomes related to cardiovascular function, for which several large cohorts have been analyzed (ISA, Table 1-8 and sections 4.4 and 4.8). Conclusions on these other systems and processes are generally consistent with conclusions reached in the last review, while also extending our conclusions on some aspects of these effects (ISA, section 4.4 and Table 1-8).

Based on the extensive assessment of the full body of evidence available in this review, the major conclusions drawn by the ISA regarding health effects of Pb in children include the following (ISA, p. lxxxvii).

> *Multiple epidemiologic studies conducted in diverse populations of children consistently demonstrate the harmful effects of Pb exposure on cognitive function (as measured by IQ decrements, decreased academic performance and poorer performance on tests of executive function).… Evidence suggests that some Pb-related cognitive effects may be irreversible and that the neurodevelopmental effects of Pb exposure may persist into adulthood (Section 1.9.4). Epidemiologic studies also demonstrate that Pb exposure is associated with decreased attention, and increased impulsivity and hyperactivity in children (externalizing behaviors). This is supported by findings in animal studies demonstrating both analogous effects and biological plausibility at relevant exposure levels. Pb exposure can*

also exert harmful effects on blood cells and blood producing organs, and is likely to cause an increased risk of symptoms of depression and anxiety and withdrawn behavior (internalizing behaviors),decreases in auditory and motor function, asthma and allergy, as well as conduct disorders in children and young adults. There is some uncertainty about the Pb exposures contributing to the effects and blood Pb levels observed in epidemiologic studies; however, these uncertainties are greater in studies of older children and adults than in studies of young children (Section 1.9.5).

Based on the extensive assessment of the full body of evidence available in this review, the major conclusions drawn by the ISA regarding health effects of Pb in adults include the following (ISA, p. lxxxviii).

A large body of evidence from both epidemiologic studies of adults and experimental studies in animals demonstrates the effect of long-term Pb exposure on increased blood pressure (BP) and hypertension (Section 1.6.2). In addition to its effect on BP, Pb exposure can also lead to coronary heart disease and death from cardiovascular causes and is associated with cognitive function decrements, symptoms of depression and anxiety, and immune effects in adult humans. The extent to which the effects of Pb on the cardiovascular system are reversible is not well-characterized. Additionally, the frequency, timing, level, and duration of Pb exposure causing the effects observed in adults has not been pinpointed, and higher past exposures may contribute to the development of health effects measured later in life.

As in prior reviews of the Pb NAAQS, this review is focused on those effects most pertinent to ambient air Pb exposures. Given the reductions in ambient air Pb concentrations over the past decades, these effects are generally those associated with the lowest levels of Pb exposure that have been evaluated. Additionally, we recognize the limitations on our ability to draw conclusions regarding the exposure conditions contributing to the findings from epidemiological analyses of blood Pb levels in populations of older children and adults, particularly in light of their history of higher Pb exposures. Evidence available in future reviews may better inform this issue. In the last review, while recognizing the range of health effects in variously aged populations related to Pb exposure, we focused on the health effects for which the evidence was strongest with regard to relationships with the lowest exposure levels,

neurocognitive effects in young children.

As is the case for studies of nervous system effects in children (discussed in more detail in section II.B.3 below), newly available studies of other effects in child and adult cohorts include cohorts with similar or somewhat lower mean blood Pb levels than in previously available studies. Categories of effects for which a causal relationship has been concluded in the ISA and for which there are a few newly available epidemiological studies indicating blood Pb associations with effects in study groups with somewhat lower blood Pb levels than previously available for these effects include effects on development (delayed puberty onset) and reproduction (male reproductive function) and on the cardiovascular system (hypertension) (ISA, sections 4.4 and 4.8; 2006 CD, sections 6.5 and 6.6). With regard to the former category, study groups in the newly available studies include groups composed of older children ranging up to age 18 years, for which there is increased uncertainty regarding historical exposures and their role in the observed effects.[26] An additional factor that handicaps our consideration of exposure levels associated with these findings is the appreciable uncertainty associated with our understanding of Pb biokinetics during this lifestage (ISA, sections 3.2, 3.3, and 4.8.6). The evidence newly available for Pb relationships with cardiovascular effects in adults include some studies with somewhat lower blood Pb levels than in the last review. The long exposure histories of these cohorts, as well as the generally higher Pb exposures of the past, complicate conclusions regarding exposure levels that may be eliciting observed effects (ISA, sections 4.4.2.4 and 4.4.7).[27] Accordingly, as discussed further below, we focus in this review, as in the last, on neurocognitive effects in young children.

[26] Several of these studies involve NHANES III cohorts for which early childhood exposures were generally much higher than those common in the U.S. today (ISA, section 4.8.5).
[27] Studies from the late 1960s and 1970s suggest that adult blood Pb levels during that period ranged from roughly 13 to 16 μg/dL and from 15 to 30 μg/dL in children aged 6 and younger (ISA, section 4.4.1).

2. Critical Periods of Exposure

As in the last review, we base our current understanding of health effects associated with different Pb exposure circumstances at various stages of life or in different populations on the full body of available evidence and primarily on epidemiological studies of health effects associated with population Pb biomarker levels (discussed further in section II.B.3 below). The epidemiological evidence is overwhelmingly composed of studies that rely on blood Pb for the exposure metric, with the remainder largely including a focus on bone Pb. Because these metrics reflect Pb in the body (e.g., as compared to Pb exposure concentrations) and, in the case of blood Pb, reflect Pb available for distribution to target sites, they strengthen the evidence base for purposes of drawing causal conclusions with regard to Pb generally. The complexity of Pb exposure pathways and internal dosimetry, however, tends to limit the extent to which these types of studies inform our more specific understanding of the Pb exposure circumstances (e.g., timing within lifetime, duration, frequency and magnitude) eliciting the various effects.

As at the time of the last review (and discussed more fully in section II.B.3 below), assessment of the full evidence base, including evidence newly available in this review, demonstrates that Pb exposure prenatally and also in early childhood can contribute to neurocognitive impacts in childhood, with evidence also indicating the potential for effects persisting into adulthood (ISA, sections 1.9.4, 1.9.5, and 1.10). In addition to the observed associations of prenatal and childhood blood Pb with effects at various ages in childhood, there is also evidence of Pb-related cognitive function effects in non-occupationally exposed adults (ISA, section 4.3.11). This includes evidence of associations of such effects in adulthood with childhood blood Pb levels and in other cohorts, with concurrent (adult) blood Pb levels (ISA, sections 4.3.2.1, 4.3.2.7 and 4.3.11). As the studies finding associations of adult effects with

childhood blood Pb levels did not examine adult blood Pb levels, the relative influence of adult Pb exposure cannot be ascertained, and a corresponding lack of early life exposure or biomarker measurements for the latter studies limits our ability to draw conclusions regarding specific Pb exposure circumstances eliciting the observed effects (4.3.11). Findings of stronger associations for adult neurocognitive effects with bone Pb, however, indicate the role of historical or cumulative exposures for those effects (ISA, section 4.3).

A critical aspect of much of the epidemiological evidence, particularly studies focused on adults (and older children) in the U.S. today, is the backdrop of generally declining environmental Pb exposure (from higher exposures during their younger years) that is common across many study populations (ISA, p. 4-2).[28] An additional factor complicating the interpretation of health effect associations with blood Pb measurements in older children and younger adults is the common behaviors of younger children (e.g., hand-to-mouth contact) that generally contribute to relatively greater exposures earlier in life (ISA, sections 3.1.1, 4.2.1). Such exposure histories for adults and older children complicate our ability to draw conclusions regarding critical time periods and lifestages for Pb exposures eliciting the effects for which associations with Pb biomarkers have been observed in these populations (e.g., ISA, section 1.9.6).[29] Thus, our confidence is greatest in the role of early childhood exposure in contributing to Pb-related neurocognitive effects that have been associated with blood Pb levels in young

[28] The declines in Pb exposure concentrations occurring from the 1970s through the early 1990s (and experienced by middle aged and older adults of today), as indicated by NHANES blood Pb information, were particularly dramatic (ISA, section 3.4.1).

[29] The evidence from experimental animal studies can be informative with regard to key aspects of exposure circumstances in eliciting specific effects, thus informing our interpretation of epidemiological evidence. For example, the animal evidence base with regard to Pb effects on blood pressure demonstrates the etiologically-relevant role of long-term exposure (ISA, section 4.4.1). This finding then informs consideration of epidemiological studies of adult populations for whom historical exposures were likely more substantial than concurrent ones, suggesting that the observed effects may be related to the past exposure (ISA, section 4.4.1). For other health effects, the animal evidence base may or may not be informative in this manner.

children. This is due, in part, to the relatively short exposure histories of young children (ISA, sections 1.9.4, 1.9.6 and 4.3.11).

Epidemiological analyses evaluating risk of neurocognitive impacts (e.g., reduced IQ) associated with different blood Pb metrics in cohorts with differing exposure patterns (including those for which blood Pb levels at different ages were not highly correlated) also indicate associations with blood Pb measurements concurrent with full scale IQ (FSIQ) tests at ages of approximately 6-7 years. The analyses did not, however, conclusively demonstrate stronger findings for early (e.g., age 2 years) or concurrent blood Pb (ISA, section 4.3.11).[30] The experimental animal evidence additionally indicates early life susceptibility (ISA, section 4.3.15 and p. 5-21). Thus, while uncertainties remain with regard to the role of Pb exposures during a particular age of life in eliciting nervous system effects, such as cognitive function decrements, the full evidence base continues to indicate prenatal and early childhood lifestages as periods of increased Pb-related risk (ISA, sections 4.3.11 and 4.3.15). We recognize increasing uncertainty, however, in our understanding of the relative impact on neurocognitive function of additional Pb exposure of children by school age or later that is associated with limitations of the currently available evidence, including epidemiological cohorts with generally similar temporal patterns of exposure.

As in the last review, there is also substantial evidence of other neurobehavioral effects in children, including effects on externalizing behaviors (reduced attention span, increased

[30] In the collective body of evidence of nervous system effects in children, it is difficult to distinguish exposure in later lifestages (e.g., school age) and its associated risk from risks resulting from exposure in prenatal and early childhood (ISA, section 4.3.11). While early childhood is recognized as a time of increased susceptibility, a difficulty in identifying a discrete period of susceptibility from epidemiological studies has been that the period of peak exposure, reflected in peak blood Pb levels, is around 18-27 months when hand-to-mouth activity is at its maximum (ISA, section 3.4.1 and 5.2.1.1; 2006 CD, p. 6-60). The task is additionally complicated by the role of maternal exposure history in contributing Pb to the developing fetus (ISA, section 3.2.2.4.).

impulsivity, hyperactivity, and conduct disorders) and on internalizing behaviors. The evidence for many of these endpoints, as with neurocognitive effects, also includes associations of effects at various ages in childhood and for some effects, into adulthood, with blood Pb levels reflective of several different lifestages (e.g., prenatal and several different ages in childhood) (ISA, sections 4.3.3 and 4.3.4). There is similar or relatively less extensive evidence to inform our understanding of such effects associated with specific time periods of exposure at specific lifestages than is the case for effects on cognitive function.

Across the range of Pb effects on physiological systems and processes other than the nervous system, the evidence base for blood pressure and hypertension is somewhat more informative with regard to the circumstances of Pb exposure eliciting the observed effects than are the evidence bases for many other effects. In the case of Pb-induced increases in blood pressure, the evidence indicates an importance of long-term exposure (ISA, sections 1.6.2 and 4.4.7.1). The greater uncertainties regarding the time, duration and magnitude of exposure contributing to these observed health effects complicate identification of sensitive lifestages and associated exposure patterns that might be compared with our understanding of the sensitivity of young children to neurocognitive impacts of Pb. Thus, while augmenting the evidence base on these additional endpoints, the newly available evidence does not lead us to identify a health endpoint expected to be more sensitive to Pb exposure than neurocognitive endpoints in children, leading us to continue to conclude that the appropriate primary focus for our review is on neurocognitive endpoints in children.

In summary, as in the last review, we continue to recognize a number of uncertainties regarding the circumstances of Pb exposure, including timing or lifestages, eliciting specific health effects. Consideration of the evidence newly available in this review has not appreciably

changed our understanding on this topic. The relationship of long-term exposure to Pb with hypertension and increased blood pressure in adults is substantiated despite some uncertainty regarding the exposures circumstances (e.g., magnitude and timing) contributing to blood Pb levels measured in epidemiological studies. Across the full evidence base, the effects for which our understanding of relevant exposure circumstances is greatest are neurocognitive effects in young children. Moreover, available evidence does not suggest a more sensitive endpoint. Thus, we continue to recognize and give particular attention to the role of Pb exposures relatively early in childhood in contributing to neurocognitive effects, some of which may persist into adulthood.

3. Nervous System Effects in Children

In considering the question of levels of Pb exposure at which health effects occur, we recognize, as discussed in sections II.B.1 and II.B.2 above, that the epidemiological evidence base for our consideration in this review, as in the past, includes substantial focus on internal biomarkers of exposure, such as blood Pb, with relatively less information specific to exposure levels, including those derived from air-related pathways. Given that blood and bone Pb are integrated markers of aggregate exposure across all sources and exposure pathways, our interpretation of studies relying on them is informed by what is known regarding the historical context and exposure circumstances of the study populations. For example, a critical aspect of much of the epidemiological evidence is the backdrop of generally declining Pb exposure over the past several decades (e.g., ISA, sections 2.5 and 3.4.1; 2006 CD, section 3.4). Thus, as a generality, recent epidemiological studies of populations with similar characteristics as those studied in the past tend to involve lower overall Pb exposures and accordingly lower blood Pb levels. This has been of particular note in the evidence of blood Pb associations with nervous system effects, particularly impacts on cognitive function in children, for which we have seen

associations with progressively lower childhood blood Pb levels across past reviews (ISA, section 4.3.12; 1986 CD; USEPA, 1990a; 2006 CD; 73 FR 66976, November 12, 2008).

The evidence currently available with regard to the magnitude of blood Pb levels associated with neurocognitive effects in children is generally consistent with that available in the review completed in 2008. Nervous system effects in children, specifically effects on cognitive function, continue to be the effects that are best substantiated as occurring at the lowest blood Pb concentrations (ISA, pp. lxxxvii-lxxxviii). Associations of blood Pb with effects on cognitive function measures in children have been reported in many studies across a range of childhood blood Pb levels, including study group (mean/median) levels ranging down to 2 μg/dL (e.g., ISA, p. lxxxvii and section 4.3.2).[31]

Among the analyses of lowest study group blood Pb levels at the youngest ages are analyses available in the last review of Pb associations with neurocognitive function decrement in study groups with mean levels on the order of 3-4 μg/dL in children aged 24 months or ranging from 5 to 7 years (73 FR 66978-66979, November 12, 2008; ISA, sections 4.3.2.1 and 4.3.2.2; Bellinger and Needleman, 2003; Canfield et al., 2003; Lanphear et al., 2005; Tellez-Rojo et al., 2006; Bellinger, 2008; Canfield, 2008; Tellez-Rojo, 2008; Kirrane and Patel, 2014).[32] Newly available in this review are two studies reporting association of blood Pb levels prior to 3

[31] The value of 2 μg/dL refers to the regression analysis of blood Pb and end-of-grade test scores, in which blood Pb was represented by categories for integer values of blood Pb from 1 μg/dL to 9 and >10 μg/dL from large statewide database. A significant effect estimate was reported for test scores with all blood Pb categories in comparison to the reference category (1 μg/dL), which included results at and below the limit of detection. Mean levels are not provided for any of the categories (Miranda et al., 2009).

[32] The tests for cognitive function in these studies include age-appropriate Wechsler intelligence tests (Lanphear et al., 2005; Bellinger and Needleman, 2003), the Stanford-Binet intelligence test (Canfield et al., 2003), and the Bayley Scales of Infant Development (Tellez-Rojo et al., 2006). The Wechsler and Stanford-Binet tests are widely used to assess neurocognitive function in children and adults. These tests, however, are not appropriate for children under age 3. For such children, studies generally use the age-appropriate Bayley Scales of Infant Development as a measure of cognitive development.

years of age with academic performance on standardized tests in primary school; mean blood Pb levels in these studies were 4.2 and 4.8 μg/dL (ISA, section 4.3.2.5; Chandramouli et al., 2009; Miranda et al., 2009). One of these two studies, which represented integer blood Pb levels as categorical variables, indicated a small effect on end-of-grade reading score of blood Pb levels as low as 2 μg/dL, after adjustment for age of measurement, race, sex, enrollment in free or reduced lunch program, parental education, and school type (Miranda et al., 2009).

In a newly available study of blood Pb levels at primary school age, a significant association of blood Pb in children aged 8-11 years and concurrently measured FSIQ was reported for a cross-sectional cohort in Korea with a mean blood Pb level of 1.7 μg/dL and range of 0.43-4.91 μg/dL (Kim et al., 2009).[33] In considering the blood Pb levels in this study, we note that blood Pb levels in children aged 8-11 are generally lower than those in pre-school children, for reasons related to behavioral and other factors (ISA, sections 3.3.5, 3.4.1 and 5.2.1.1). It is likely that the blood Pb levels of this study group at earlier ages, e.g., prior to school entry, were higher and the available information does not provide a basis to judge whether the blood Pb levels in this study represent lower exposure levels than those experienced by the younger study groups. In still older children, a large cross-sectional investigation of blood Pb association with effects on memory and learning that was available in the last review was focused on children aged 6-16 years, born during 1972-1988, with a mean blood Pb of 1.9 μg/dL (Lanphear et al., 2000). A study newly available in this review, focused on a subset of the earlier study cohort (ages 12-16, born during 1975-1982), also reports a significant negative association of blood Pb with learning and memory test results with mean blood Pb levels of approximately 2 μg/dL (ISA, section 4.3.2.3; Lanphear et al., 2000; Krieg et al., 2010). In considering these study findings

[33] Limitations of this study included a lack of consideration of potential confounding by parental caregiving quality or IQ (ISA, Table 4-3).

with regard to the question of exposure levels eliciting effects, we recognize, however, that blood Pb levels are, in general, lower among teenagers than young children and also that, for these subjects specifically, the magnitude of blood Pb levels during the earlier childhood (e.g., pre-school ages) was much higher. For example, the mean blood Pb levels for the 1-5 year old age group in the NHANES 1976-80 sample was 15 μg/dL, declining to 3.6 μg/dL in the NHANES 1988-1991 sample (Pirkle et al., 1994; ISA, section 3.4.1). In summary, the available information is for population groups of ages for which the NHANES samples indicate exposure levels were higher earlier in childhood. Thus, in light of the NHANES information, although the blood Pb levels in the studies of cognitive effects in older child population groups are lower (at the time of the study) than the younger child study levels, the studies of older children do not provide a basis for concluding a role for lower Pb exposure levels than those experienced by the younger study groups.

With regard to other nervous system effects in children, the evidence base at lower blood Pb levels is somewhat extended since the last review with regard to the evidence on Pb and effects on externalizing behaviors, such as attention, impulsivity, hyperactivity and conduct disorders (ISA, section 4.3.3 and Table 4-17). Several newly available studies investigating the role of blood Pb levels in older children (primary school age and older) have reported significant associations for these effects with concurrent blood Pb levels, with mean levels generally on the order of 5 μg/dL or higher (ISA, section 4.3.3). One exception is the newly available cross-sectional, categorical analysis of the NHANES 2001-2004 sample of children aged 8-15 years, which found higher prevalence of conduct disorder in the subgroup with concurrent blood Pb levels of 0.8-1.0 μg/dL as compared to the <0.8 μg/dL group (ISA, section 4.3.4 and Table 4-12). As noted above, we recognize that many of these children, born between 1986 and 1996, are

likely to have had much higher Pb exposures (and associated blood Pb levels) in their earlier years than those commonly experienced by young children today, thus making this study relatively uninformative with regard to evidence of effects associated with lower exposure levels than provided by evidence previously available.

In summary, our conclusions regarding exposure levels at which Pb health effects occur, particularly with regard to such levels that might be common in the U.S. today, are complicated now, as in the last review, by several factors. These factors include the scarcity of information in epidemiological studies on cohort exposure histories, as well as by the backdrop of higher past exposure levels which frame the history of most, if not all, older study cohorts. Recognizing the complexity, as well as the potential role of higher exposure levels in the past, we continue to focus our consideration of this question on the evidence of effects in young children for which our understanding of exposure history is less uncertain.[34] Within this evidence base, we recognize the lowest study group blood Pb levels to be associated with effects on cognitive function measures, indicating that to be the most sensitive endpoint. As described above, the evidence available in this review is generally consistent with that available in the last review with regard to blood Pb levels at which such effects had been reported (ISA, section 4.3.2; 2006 CD, section 8.4.2.1; 73 FR 66976-66979, November 12, 2008). As blood Pb levels are a reflection of exposure history, particularly in early childhood (ISA, section 3.3.2), we conclude, by extension, that the currently available evidence does not indicate Pb effects at exposure levels appreciably lower than recognized in the last review.

We additionally note that, as in the last review, a threshold blood Pb level with which

[34] In focusing on effects associated with blood Pb levels in early childhood, however, we additionally recognize the evidence across categories of effects that relate to blood Pb levels in older child study groups (for which early childhood exposure may have had an influence) which provides additional support to an emphasis on nervous system effects (ISA, sections 4.3, 4.4, 4.5, 4.6, 4.7, 4.8).

nervous system effects, and specifically cognitive effects, occur in young children cannot be discerned from the currently available studies (ISA, sections 1.9.3 and 4.3.12). Epidemiological analyses have reported blood Pb associations with cognitive effects (FSIQ or BSID MDI[35]) for young child population subgroups (age 5 years or younger) with individual blood Pb measurements as low as approximately 1 µg/dL and mean concentrations as low as 2.9 to 3.8 µg/dL (ISA, section 4.3.12; Bellinger and Needleman, 2003; Bellinger, 2008; Canfield el al., 2003; Canfield, 2008; Tellez-Rojo et al., 2006; Tellez-Rojo, 2008). As concluded in the ISA, however, "the current evidence does not preclude the possibility of a threshold for neurodevelopmental effects in children existing with lower blood levels than those currently examined" (ISA, section 4.3.13).

Important uncertainties associated with the evidence of effects at low exposure levels are similar to those recognized in the last review, including the shape of the concentration-response relationship for effects on neurocognitive function at low blood Pb levels in today's young children. Also of note is our interpretation of associations between blood Pb levels and effects in epidemiological studies, with which we recognize uncertainty with regard to the specific exposure circumstances (timing, duration, magnitude and frequency) that have elicited the observed effects, as well as uncertainties in relating ambient air concentrations (and associated air-related exposures) to blood Pb levels in early childhood, as discussed in section II.B.2 above. We additionally recognize uncertainties associated with conclusions drawn with regard to the nature of the epidemiological associations with blood Pb (e.g., ISA, section 4.3.13), but note that, based on consideration of the full body of evidence for neurocognitive effects, the EPA has

[35] The Bayley Scales of Infant Development, Mental Development Index is a well-standardized and widely used assessment measure of infant cognitive development. Scores earlier than 24 months are not necessarily strongly correlated with later FSIQ scores in children with normal development (ISA, section 4.3.15.1).

determined a causal relationship to exist between relevant blood Pb levels and neurocognitive impacts in children (ISA, section 4.3.15.1).

Based primarily on studies of FSIQ, the assessment of the currently available studies, as was the case in the last review, continues to recognize a nonlinear relationship between blood Pb and effects on cognitive function, with a greater incremental effect (greater slope) at lower relative to higher blood Pb levels within the range thus far studied, extending from well above 10 µg/dL to below 5 µg/dL (ISA, section 4.3.12). This was supported by the evidence available in the last review, including the analysis of the large pooled international dataset comprised of blood Pb measurements and IQ test results from seven prospective cohorts (Lanphear et al., 2005; Rothenberg and Rothenberg, 2005; ISA, section 4.3.12). The blood Pb measurements in this pooled dataset that were concurrent with the IQ tests ranged from 2.5 µg/dL to 33.2 µg/dL. The study by Lanphear et al. (2005) additionally presented analyses that stratified the dataset based on peak blood Pb levels (e.g., with cutpoints of 7.5 µg/dL and 10 µg/dL peak blood Pb) and found that the coefficients from linear models of the association for IQ with concurrent blood Pb were higher in the lower peak blood Pb level subsets than the higher groups (ISA, section 4.3.12; Lanphear et al., 2005).

We note that since the completion of the ISA, two errors have been identified with the pooled dataset analyzed by Lanphear et al. (2005) (Kirrane and Patel, 2014). A recent publication and the EPA have separately recalculated the statistics and mathematical model parameters of Lanphear et al. (2005) using the corrected pooled dataset (see Kirrane and Patel, 2014). While the magnitude of the loglinear and linear regression coefficients are modified slightly based on the corrections, the conclusions drawn from these coefficients, including the finding of a steeper slope at lower (as compared to higher) blood Pb concentrations, are not

affected (Kirrane and Patel, 2014).

In other publications, stratified analyses of several individual cohorts also observed higher coefficients for blood Pb relationships with measures of neurocognitive function in lower as compared to higher blood Pb subgroups (ISA, section 4.3.12; Canfield et al., 2003; Bellinger and Needleman, 2003; Kordas et al., 2006; Tellez-Rojo et al., 2006). Of these subgroup analyses, those involving the lowest mean blood Pb levels and closest to the current mean for U.S. preschool children are listed in Table 1 (drawn from Table 3 of the 2008 final rulemaking notice [73 FR 67003, November 12, 2008], and Kirrane and Patel, 2014).[36] These analyses were important inputs for the evidence-based, air-related IQ loss framework which informed decisions on a revised standard in the last review (73 FR 67005, November 12, 2008), discussed in section II.A.1 above. As the framework focused on the median of the four slopes in Table 1, the change to the one from Lanphear et al. (2005) based on the recent recalculation described above has no impact on conclusions drawn from the framework.

Table 1. Summary of Quantitative Relationships of IQ and Blood Pb for Analyses with Blood Pb Levels Closest to Those of Young Children in the U.S. Today

Blood Pb Levels (µg/dL)		**Study/Analysis**	**Average Linear Slope**[A] (IQ[B] points per µg/dL)
Geometric Mean	**Range** (min-max)		
2.9	0.8 – 4.9	Tellez-Rojo et al (2006)[B], subgroup w. concurrent blood Pb <5 µg/dL	-1.71
3.3	0.9 – 7.4	Lanphear et al (2005)[C], subgroup w. peak blood Pb <7.5 µg/dL	-2.53
3.32	0.5 – 8.4	Canfield et al (2003) [C,D], subgroup w. peak blood Pb <10 µg/dL	-1.79

[36] One of these four is from the analysis of the lowest blood Pb subset of the pooled international study by Lanphear et al. (2005). The nonlinear model developed from the full pooled dataset is the basis of the C-R functions used in the 2007 REA, in which risk was estimated over a large range of blood Pb levels (PA, section 3.4.3.3). Given the narrower focus of the evidence-based framework on IQ response at the end of studied blood Pb levels (closer to U.S. mean level), the C-R functions in Table 1 are from linear analyses (each from separate publications) for the study group subsets with blood Pb levels closest to mean for children in the U.S. today.

3.8	1 – 9.3	Bellinger and Needleman (2003)[C,E], subgroup w. peak blood Pb <10 µg/dL	-1.56
		Median value	-1.75

A - Average linear slope estimates here are generally for relationship with IQ assessed concurrently with blood Pb measurement. As exceptions, Bellinger & Needleman (2003) slope is relationship for 10 year old IQ with blood Pb levels at 24 months, and the data for Boston cohort included in Lanphear et al. (2005) slope are relationship for 10 year old IQ with blood Pb levels at 5 years.
B - The slope for Tellez-Rojo et al. (2006) is for BSID (MDI), a measure of cognitive development appropriate to study population age (24-mos). The blood Pb levels for this subgroup are from Tellez-Rojo (2008).
C - The Lanphear et al. (2005) pooled international study also includes blood Pb data from the Rochester and Boston cohorts, although for different ages (6 and 5 years, respectively) than the ages analyzed in Canfield et al. (2003) and Bellinger and Needleman (2003). Thus, the ages at the blood Pb measurements used in derivation of the linear slope for the Lanphear et al. (2005) subgroup shown here are 5 to 7 years. The blood Pb levels and coefficient presented here for Lanphear et al (2005) study group reflect the recalculation using the corrected pooled dataset (Kirrane and Patel, 2014).
D - Blood Pb levels for this subgroup are from Canfield (2008).
E - Blood Pb levels for this subgroup are from Bellinger (2008).

Several studies newly available in the current review have, in all but one instance, also found a nonlinear blood Pb-cognitive function relationship in nonparametric regression analyses of the cohort blood Pb levels analyzed (ISA, section 4.3.12). These studies, however, used statistical approaches that did not produce quantitative results for each blood Pb group (ISA, section 4.3.12). Thus, newly available studies have not extended the range of observation for quantitative estimates of this relationship to lower blood Pb levels than those of the previous review. The ISA further notes that the potential for nonlinearity has not been examined in detail within a lower, narrower range of blood Pb levels than those of the full cohorts thus far studied in the currently available evidence base (ISA, section 4.3.12). Such an observation in the last review supported the consideration of linear slopes with regard to blood Pb levels at and below those represented in Table 1. In summary, the newly available evidence does not substantively alter our understanding of the C-R relationship (including quantitative aspects) for neurocognitive impact, such as IQ with blood Pb in young children.

4. At-Risk Populations

In this section, we use the term "at-risk populations"[37] to recognize populations that have a greater likelihood of experiencing Pb-related health effects, i.e., groups with characteristics that contribute to an increased risk of Pb-related health effects. These populations are also sometimes referred to as sensitive groups (as in section I.A above). In identifying factors that increase risk of Pb-related health effects, the EPA has considered evidence regarding factors contributing to increased susceptibility, generally including physiological or intrinsic factors contributing to a greater response for the same exposure, and those contributing to increased exposure, including that resulting from behavior leading to increased contact with contaminated media (ISA, Chapter 5). Physiological risk factors include both conditions contributing to a group'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 (e.g., ISA, sections 5.3 and 5.1, respectively).

The information newly available in this review has not substantially altered our previous understanding of at-risk populations for Pb in ambient air. As in the last review, the factor most prominently recognized to contribute to increased risk of Pb effects is childhood (ISA, section 1.9.6). As noted in section II.B.2 above, although the specific ages or lifestages of greatest susceptibility[38] or risk have not been established (e.g., ISA, section 4.3.11), the at-risk status of young children to the neurodevelopmental effects of Pb is well recognized (e.g., ISA, sections 1.9.6, 4.3, 5.2.1, 5.3.1, and 5.4). The evidence indicates that prenatal blood Pb levels are

[37] In the context of "at-risk populations," the term "population" refers to persons having one or more qualities or characteristics including, for example, a specific pre-existing illness or a specific age or lifestage, with lifestage referring to a distinguishable time frame in an individual's life characterized by unique and relatively stable behavioral and/or physiological characteristics that are associated with development and growth.

[38] As noted in the ISA, "in most instances, 'susceptibility' refers to biological or intrinsic factors (e.g., age and sex) while 'vulnerability' refers to nonbiological or extrinsic factors (e.g., socioeconomic status [SES])" and the terms "at-risk" and "sensitive" populations have in various instances been used to encompass these concepts more generally (ISA, p. 5-1). In providing detail regarding factors contributing to an "at-risk" status in this section, we have used the other terms in particular instances, with our usage consistent with these common definitions.

associated with nervous system effects, including mental development in very young children and can also be associated with cognitive decrements in older children (ISA, section 4.3). Additionally, the coincidence during early childhood of behaviors that increase exposure, such as hand-to-mouth contact by which children transfer Pb in settled particles to their mouths, and the development of the nervous system also contributes increased risk during this time (ISA, sections 3.7.1, 4.3.2.6, 5.2.1.1, 5.3.1.1 and 5.4). Collectively, however, the evidence indicates both the susceptibility of the developing fetus and early postnatal years, as well as the potential for continued susceptibility through childhood as the human central nervous system continues to mature and be vulnerable to neurotoxicants (ISA, sections 1.9.5 and 4.3.15; 2006 CD, section 6.2.12). As discussed in section II.B.2 above, while uncertainties remain with regard to the role of Pb exposures during a particular age of life in eliciting nervous system effects, such as cognitive function decrements, the full evidence base continues to indicate prenatal and early childhood lifestages as periods of increased Pb-related risk (ISA, sections 4.3.11 and 4.3.15).

Several physiological factors increase the risk of Pb-related health effects by contributing to increased blood Pb levels over those otherwise associated with a given Pb exposure (ISA, sections 3.2, 3.3 and 5.1). These include nutritional status, which plays a role in Pb absorption from the gastrointestinal tract (ISA, sections 3.2.1.2, 5.1, 5.3.10 and 5.4). For example, diets deficient in iron, calcium or zinc can contribute to increased Pb absorption and associated higher blood Pb levels (ISA, sections 3.2.1.2, and 5.1). Evidence is suggestive of some genetic characteristics as potential risk factors, such as presence of the δ-aminolevulinic acid dehydratase-2 (ALAD-2) allele which has been indicated to increase blood Pb levels or Pb-related risk of health effects in some studies (ISA, sections 3.3.2 and 5.1).

Risk factors based on increased exposure include spending time in proximity to sources

of Pb to ambient air or other environmental media (e.g., large active metals industries or locations of historical Pb contamination) (ISA, sections 1.9.6, 3.7.1, 5.2.5 and 5.4). Residential factors associated with other sources of Pb exposure (e.g., leaded paint or plumbing with Pb pipes or solder) are another exposure-related risk factor (ISA, sections 3.7.1, 5.2.6 and 5.4). Additionally, some races or ethnicities have been associated with higher blood Pb levels, with differential exposure indicated in some cases as the cause (ISA, sections 5.2.3 and 5.4). Lower socioeconomic status (SES) has been associated with higher Pb exposure and higher blood Pb concentration, leading the ISA to conclude the evidence is suggestive for low SES as a risk factor (ISA, sections 5.3.16, 5.2.4 and 5.4). Although the differences in blood Pb levels between children of lower and higher income levels (as well as among some races or ethnicities) have lessened, blood Pb levels continue to be higher among lower-income children indicating higher exposure and/or greater influence of factors independent of exposure, such as nutritional factors (ISA, sections 1.9.6, 5.2.1.1 and 5.4).

In considering risk factors associated with increased Pb exposure or increased blood Pb levels, we note that the currently available evidence continues to support a nonlinear relationship between neurocognitive effects and blood Pb that indicates incrementally greater impacts at lower as compared to higher blood Pb levels (ISA, section 4.3.12), as described in section II.B.3 above. An important implication of this finding is that while children with higher blood Pb levels are at greater risk of Pb-related effects than children with lower blood Pb levels, on an incremental basis (e.g., per μg/dL), the risk is greater for children at lower blood Pb levels. This was given particular attention in the last review of the Pb NAAQS, in which the standard was revised with consideration of the incremental impact of air-related Pb on young children in the U.S. and the recognition of greater impact for those children with lower absolute blood Pb levels

(73 FR 67002, November 12, 2008). Such consideration included a focus on those C-R studies involving the lowest blood Pb levels, as described in section II.A.1 above.

In summary, the information newly available in this review has not appreciably altered our understanding of human populations that are particularly sensitive to Pb exposures. In the current review, as at the time of the last review of the Pb NAAQS, we recognize young children as an important at-risk population, with sensitivity extending to prenatal exposures and into childhood development. Additional risk factors for increased blood Pb levels include deficiencies in dietary minerals (iron, calcium and zinc), some racial or ethnic backgrounds,[39] and spending time in proximity to environmental sources of Pb or residing in older houses with Pb exposure related to paint or plumbing.[40] The currently available evidence continues to additionally suggest a potential for increased risk associated with several other factors, including older adulthood,[41] pre-existing disease (e.g., hypertension), variants for certain genes and increased stress (ISA,

[39] The ISA concludes that studies of race/ethnicity provide adequate evidence that race/ethnicity is an at-risk factor based on the higher exposure observed among non-white populations and some modification observed in studies of associations between Pb levels and some health effects, such as hypertension (ISA, section 6.4).

[40] The evidence for SES continues to indicate increased blood Pb levels in lower income children, although its role with regard to an increased health risk for the same blood Pb level is unclear and its role generally with regard to Pb-related risk is somewhat complicated. SES often serves as a marker term for one or a combination of unspecified or unknown environmental or behavioral variables. Further, it is independently associated with an adverse impact on neurocognitive development, and a few studies have examined SES as a potential modifier of the association of childhood Pb exposure with cognitive function with inconsistent findings regarding low SES as a potential risk factor. The ISA concludes the evidence for SES as a Pb risk factor is suggestive, based on the greater exposures or blood Pb levels in some low SES groups (ISA, section 5.4).

[41] The ISA identifies older adulthood as a lifestage of potentially greater risk of Pb-related health effects based primarily on the evidence of increases in blood Pb levels during this lifestage (ISA, sections 5.2.1.2, 5.3.1.2, and 5.4), as well as observed associations of some cardiovascular and nervous system effects with bone and blood Pb in older populations, with biological plausibility for the role of Pb provided by experimental animal studies (ISA, sections 4.3.5,4.3.7 and 4.4). Exposure histories of older adult study populations, which included younger years during the time of leaded gasoline usage and other sources of Pb exposures which were more prevalent in the past than today, are likely contributors to their blood Pb levels (ISA, pp. lx-lxi; Figure 2-1 and sections 2.5.2, 3.3.5 and 5.2.1.2).

section 5.3.4). As discussed above, we recognize the sensitivity of the prenatal period and several lifestages of childhood to an array of neurocognitive and behavioral effects, and we particularly recognize young children as an important at-risk population in light of current environmental exposure levels. Age or lifestage was used to distinguish potential groups on which to focus in the last review in recognition of its role in exposure and susceptibility, and young children were the focus of the REA in consideration of the health effects evidence regarding endpoints of greatest public health concern and in recognition of effects on the developing nervous system as a sentinel endpoint for public health impacts of Pb. This identification continues to be supported by the evidence available in the current review.

5. Potential Impacts on Public Health

There are several potential public health impacts associated with Pb exposure in the current U.S. population. In recognition of effects causally related to blood Pb levels somewhat near those most recently reported for today's population and for which the weight of the evidence is greatest, the potential public health impacts most prominently recognized in the ISA are population IQ impacts associated with childhood Pb exposure and prevalence of cardiovascular effects in adults (ISA, section 1.9.1). With regard to the latter category, as discussed above, the full body of evidence indicates a role of long-term cumulative exposure, with uncertainty regarding the specific exposure circumstances contributing to the effects in the epidemiological studies of adult populations, for whom historical Pb exposures were likely much higher than exposures that commonly occur today (ISA, section 4.4). There is less uncertainty regarding the exposure patterns contributing to the blood Pb levels reported in studies of younger populations (ISA, sections 1.9.4 and 1.10). Accordingly, the discussion of public health implications relevant to this review is focused predominantly on nervous system effects, including IQ decrements, in children.

The magnitude of a public health impact is dependent upon the type or severity of the effect, as well as the size of populations affected. Intelligence quotient is a well-established, widely recognized and rigorously standardized measure of neurocognitive function, as well as a global measure reflecting the integration of numerous processes (ISA, section 4.3.2; 2006 CD, sections 6.2.2 and 8.4.2). Examples of other measures of cognitive function negatively associated with Pb exposure include other measures of intelligence and cognitive development and measures of other cognitive abilities, such as learning, memory, and executive functions, as well as academic performance and achievement (ISA, section 4.3.2). Although some neurocognitive effects of Pb in children may be transient, some may persist into adulthood (ISA, section 1.9.5).[42] We also note that deficits in neurodevelopment early in life may have lifetime consequences as "[n]eurodevelopmental deficits measured in childhood may set affected children on trajectories more prone toward lower educational attainment and financial well-being" (ISA, section 4.3.14). Thus, population groups for which neurodevelopment is affected by Pb exposure in early childhood are at risk of related impacts on their success later in life. Further, in considering population risk, the ISA notes that "[s]mall shifts in the population mean IQ can be highly significant from a public health perspective" (ISA, p. xciii). For example, if Pb-related decrements are manifested uniformly across the range of IQ scores in a population, "a small shift in the population mean IQ may be significant from a public health perspective because such a shift could yield a larger proportion of individuals functioning in the low range of the IQ distribution, which is associated with increased risk of educational, vocational, and social failure" as well as a decrease in the proportion with high IQ scores (ISA, section 1.9.1).

[42] The ISA states that the "persistence of effects appears to depend on the duration and window of exposure as well as other factors that may affect an individual's ability to recover from an insult," with some evidence of greater recovery in children reared in households with more optimal caregiving characteristics and low concurrent blood Pb levels (ISA, p. 1-77; Bellinger et al., 1990).

As summarized above, young children are the at-risk population that may be most at risk of health effects associated with exposure to Pb and children at greatest risk from *air-related* Pb are those children with highest air-related Pb exposure which we consider to be those living in areas of higher ambient air Pb concentrations. To inform our understanding of the extent of this population potentially at risk from air-related Pb, the PA includes two analyses. The first analysis is based on consideration of the available air Pb monitoring information. As the air quality data set available for the first analysis may not be inclusive of all of the newly sited monitors (as discussed in section 2.2.1 of the PA) and there may be other areas with elevated Pb concentrations, a second analysis was performed in consideration of emissions estimates from the National Emissions Inventory (NEI), although with recognition of uncertainties associated with inferences drawn from such estimates with regard to ambient air Pb concentrations and exposures (PA, pp. 3-36 to 3-38).[43]

The first PA analysis indicates that approximately one hundredth of one percent of the full population of children aged 5 or under in the U.S. reside within 0.5 km of monitors exceeding or within 10 percent of the level of the current standard (PA, section 2.2.2.2, pp. 3-36 to 3-37, 4-25 and Table 3-4). In the second analysis, the size of young child populations residing in areas near large Pb sources was approximately four hundredths of one percent of the full U.S. population of children aged 5 years or younger (PA, pp. 3-37 to 3-38, 4-25). The PA recognized uncertainties and potential limitations associated with the use of the emissions estimates in the second analysis to make inferences regarding ambient air Pb exposures, uncertainties both with regard to the accuracy of such estimates and also with regard to the role of specific source

[43] Such uncertainties include those with regard to specific source characteristics and meteorology, not explicitly considered in the analysis. In light of such uncertainties, the PA interprets the emissions-based analysis to provide a bounding estimate below which the true value is expected to fall (PA, p. 3-37).

characteristics and meteorology, not explicitly considered here, in influencing ambient air Pb concentrations and contributing to substantial variation in air Pb concentrations at source locations (e.g., PA, Figure 2-11). Accordingly, while the second analysis is considered informative with regard to the potential prevalence of airborne Pb emissions and potential exposure of human populations, it is limited with regard to its ability to identify populations living in areas of elevated ambient air Pb concentrations. The PA interprets the two analyses together to indicate that well below one tenth of one percent of the full population of children aged 5 years or younger in the U.S. today live in areas with air Pb concentrations near or above the current standard, with the current monitoring data indicating the size of this population to be approximately one hundredth of a percent of the full population of children aged 5 or younger (PA, pp. 3-36 to 3-38, 4-25, 4-32).

C. Blood Lead as a Biomarker of Exposure and Relationships with Air Lead

Blood Pb is well established as a biomarker of Pb exposure and of internal dose, with relationships between air Pb concentrations and blood Pb concentrations informing consideration of the NAAQS for Pb since its initial establishment in 1978. Lead associated with inhaled particles may, depending on particle size and Pb solubility, be absorbed into the systemic circulation or transported with particles to the gastrointestinal tract (ISA, section 3.2.1.1), where its absorption is influenced by a range of factors (ISA, section 3.2.1.2). Lead in the blood stream is quickly distributed throughout the body (e.g., within days), available for exchange with the soft and skeletal tissues, the latter of which serves as the largest storage compartment (ISA, section 3.2.2.2). Given the association with exposure and the relative ease of collection, blood Pb levels are extensively used as an index or biomarker of exposure by national and international health agencies, as well as in epidemiological and toxicological studies of Pb health effects and

dose-response relationships (ISA, sections 3.3.2, 3.4.1, 4.3, 4.4, 4.5, 4.6, 4.7, and 4.8). While bone Pb measurements are also used in epidemiological studies as an indicator of cumulative Pb exposure, blood Pb measurements remain the predominant, well-established and well-characterized exposure approach.

Since 1976, the CDC has been monitoring blood Pb levels nationally through the NHANES. This survey has documented the dramatic decline in mean blood Pb levels in all ages of the U.S. population that has occurred since the 1970s (PA, Figure 3-1), and that coincides with actions on leaded fuels, leaded paint, Pb in food packaging, and Pb-containing plumbing materials that have reduced Pb exposure in the U.S. (ISA, section 3.4.1; Pirkle et al., 1994; Schwemberger et al., 2005). This decline has continued over the more recent past. For example, the 2009-2010 geometric mean blood Pb level in U.S. children aged 1-5 years is 1.17 µg/dL, as compared to 1.51 µg/dL in 2007-2008 (ISA, section 3.4.1) and 1.8 µg/dL in 2003-2004, the most recent data available at the time of the last review (73 FR 67002, November 12, 2008). Somewhat less dramatic declines have been reported in the upper tails of the distribution and in different groups with higher blood Pb levels than the general child population (ISA, Figures 3-17 and 3-19).

The blood Pb concentration in childhood (particularly early childhood) can more quickly (than in adulthood) reflect changes in total body burden (associated with the shorter exposure history) and can also reflect changes in recent exposures (ISA, section 3.3.5). The relationship of children's blood Pb to recent exposure may reflect their labile bone pool, with their rapid bone turnover in response to rapid childhood growth rates (ISA, section 3.3.5). The relatively smaller skeletal compartment of Pb in children (particularly very young children) compared to adults is subject to more rapid turnover. The distribution of Pb in the body is dynamic throughout life,

with Pb in the body being exchanged between blood and bone and between blood and soft tissues (ISA, sections 3.3.5 and 3.2.2; 2006 CD, section 4.3.2). The rates of these exchanges vary with age, exposure and various physiological variables. For example, resorption of bone, which results in the mobilization of Pb from bone into the blood, is a somewhat rapid and ongoing process during childhood and a more gradual process in later adulthood (ISA, sections 3.2.2.2, 3.3.5 and 3.7.2; PA, pp. 3-2 to 3-3).

Lead in ambient air contributes to Pb in blood by multiple exposure pathways by both inhalation and ingestion exposure routes (ISA, section 3.1.1). Multiple studies have demonstrated young children's blood Pb levels to reflect Pb exposures, including exposures to Pb in surface dust (e.g., Lanphear and Roghmann, 1997; Lanphear et al., 1998). These and studies of child populations near sources of air Pb emissions, such as metal smelters, have further demonstrated the effect of airborne Pb on interior dust and on blood Pb (ISA, sections 3.4.1, 3.5.1 and 3.5.3; Hilts, 2003; Gulson et al., 2004).

As blood Pb is an integrated marker of aggregate Pb exposure across all pathways, the blood Pb C-R relationships described in epidemiological studies of Pb-exposed populations do not distinguish among different sources of Pb or pathways of Pb exposure (e.g., inhalation, ingestion of indoor dust, ingestion of dust containing leaded paint). Thus, our interpretation of the health effects evidence for purposes of this review necessitates characterization of the relationships between Pb from those sources and pathways of interest in this review (i.e., those related to Pb emitted into the air) and blood Pb.

The evidence for air-to-blood relationships derives from analyses of datasets for populations residing in areas with differing air Pb concentrations, including datasets for circumstances in which blood Pb levels have changed in response to changes in air Pb. The

control for variables other than air Pb that can affect blood Pb varies across these analyses. At the conclusion of the last review in 2008, the EPA interpreted the evidence as providing support for use (in informing the Administrator's decision on standard level) of a range of air-to-blood ratios[44] "inclusive at the upper end of estimates on the order of 1:10 and at the lower end on the order of 1:5" (73 FR 67002, November 12, 2008). This conclusion reflected consideration of the air-to-blood ratios presented in the 1986 CD[45] and associated observations regarding factors contributing to variation in such ratios, ratios reported subsequently and ratios estimated based on modeling performed in the REA, as well as advice from CASAC (73 FR 66973-66975, 67001-67002, November 12, 2008). The information available in this review, which is assessed in the ISA and largely, although not completely, comprises studies that were available in the last review, does not alter the primary scientific conclusions drawn in the last review regarding the relationships between Pb in ambient air and Pb in children's blood. The ratios summarized in the ISA in this review span a range generally consistent with the range concluded in 2008 (ISA, section 3.5.1).

The evidence pertaining to the quantitative relationship between air Pb and children's blood Pb is now, as in the past, limited by the circumstances in which the data are collected. These estimates are generally developed from studies of populations in a variety of Pb exposure circumstances. Accordingly, there is significant variability in air-to-blood ratios among the different study populations exposed to Pb through different air-related exposure pathways and at different exposure levels. This variability in air-to-blood estimates can relate to the

[44] The quantitative relationship between ambient air Pb and blood Pb, often termed a slope or ratio, describes the increase in blood Pb (in µg/dL) estimated to be associated with each unit increase of air Pb (in µg/m^3). Ratios are presented in the form of 1:x, with the 1 representing air Pb (in µg/m^3) and x representing blood Pb (in µg/dL). Description of ratios as higher or lower refers to the values for x (i.e., the change in blood Pb per unit of air Pb). Slopes are presented as simply the value of x.

[45] The 2006 CD did not include an assessment of then-current evidence on air-to-blood ratios.

representation of air-related pathways and study populations, including, for example, relatively narrow age ranges for the population in order to reduce age-related variability in blood Pb, or including populations with narrowly specified dietary sources. It can relate to the study population exposure and blood Pb levels (ISA, section 3.7.4). It can also relate to the precision of air and blood measurements and of the study circumstances, such as with regard to spatial and temporal aspects. Additionally, in situations where exposure to nonair sources covaries with air-related exposures that are not accounted for in deriving ratio estimates, uncertainties may relate to the potential for confounding by nonair exposure covariance (ISA, section 3.5). Most of the studies assessed in the ISA and PA have reported ratios for which the relationship is linear, while a subset are derived from nonlinear models (PA, Table 3-1; ISA, section 3.7.4).

As was noted in the last review, age is an important influence on the magnitude of air-to-blood ratio estimates derived. Ratios for children are generally higher than those for adults, and higher for young children than older children, perhaps due to behavioral differences between the age groups, as well as their shorter exposure history. Similarly, given the common pattern of higher blood Pb levels in pre-school-aged children than during the rest of childhood, related to behaviors that increase environmental exposures (e.g., hand-to-mouth activity), ratios would be expected to be highest in earlier childhood. Additionally, estimates of air-to-blood ratios that include air-related ingestion pathways in addition to the inhalation pathway are "necessarily higher," in terms of blood Pb response, than those estimates based on inhalation alone (1986 CD, p. 11-106). Thus, the extent to which studies account for the full set of air-related inhalation and ingestion exposure pathways affects the magnitude of the resultant air-to-blood estimates, such that including fewer pathways as "air-related" yields lower ratios. Estimates of air-to-blood ratios can also be influenced by population characteristics that may influence blood Pb;

accordingly, some analyses include adjustments.

Given the recognition of young children as a key at-risk population in this review, as in the last (as discussed in section II.B.3 above), as well as the influence of age on blood Pb levels, we have considered the available studies in groups based on the extent of their inclusion of children younger than or barely school age (less than or equal to 5 years of age). Among the first group of studies, focused exclusively on young children, only one study dates from the end of or after the phase-out of leaded gasoline usage (Hilts, 2003). This study reports changes in children's blood Pb levels associated with reduced Pb emissions and associated air concentrations near a Pb smelter in Canada (for children through age 5). Given the timing of this study, after the leaded gasoline phase-out, and its setting near a smelter, the ambient air Pb in this study may be somewhat more comparable to that near sources in the U.S. today than other studies discussed herein. The study authors report an air-to-blood ratio of 1:6.[46] An EPA analysis of the air and blood data reported for 1996, 1999 and 2001 results in a ratio of 1:6.5, and an analysis focused only on the 1996 and 1999 data (pre- and post- the new technology) yields a

[46] Sources of uncertainty include the role of factors other than ambient air Pb reduction in influencing decreases in blood Pb (ISA, section 3.5.1). The author cited remedial programs (e.g., community and home-based dust control and education) as potentially responsible for some of the blood Pb reduction seen during the study period (1997 to 2001), although the author notes that these programs were in place in 1992, suggesting they are unlikely to have contributed to the sudden drop in blood Pb levels occurring after 1997 (Hilts, 2003). Other aspects with potential implications for ratios include the potential for children with lower blood Pb levels not to return for subsequent testing, and the age range of 6 to 36 months in the 2001 blood screening compared to ages up to 60 months in earlier years of the study (Hilts, 2003).

ratio of 1:7 (ISA, section 3.5.1; Hilts, 2003).[47] The two other studies that focused on children of age 5 or younger analyzed variations in air Pb as a result of variations in leaded gasoline usage in Chicago, Illinois and reported somewhat higher ratios of 1:8 and 1:8.6 (Hayes et al., 1994; Schwartz and Pitcher, 1989). We note, however, the blood Pb concentrations in the two leaded gasoline studies are appreciably higher (a factor of two or more) than those in the study near the smelter (Hilts, 2003), and also than those commonly reported in the U.S. today.

The second group of studies includes but is not limited to children less than or equal to 5 years of age. This group includes a complex statistical analysis and associated dataset for a cohort of children born in Mexico City from 1987 through 1992 (Schnaas et al., 2004). Although this study, which was not assessed in the last review, encompasses the period of leaded gasoline usage, it further informs our understanding of factors influencing the quantitative relationship between air Pb and children's blood Pb. Air-to-blood ratios developed from this study are influenced by a number of factors and appear to range from roughly 1:2 to 1:6, in addition to an estimate of 1:9 (ISA, section 3.5.1), although the latter is derived from a data set restricted to the latter years of the study when little change in air Pb concentration occurred, such that the role of air Pb may be more uncertain. Estimates associated with the developmental period of highest exposure (e.g., age 2 years) range up to approximately 1:6, illustrating the influence of age on the ratio (ISA, section 3.5.1). Also in the second group of studies are two much older studies of

[47] This study considered changes in ambient air Pb levels and associated blood Pb levels over a 5-year period which included closure of an older Pb smelter and subsequent opening of a newer facility in 1997 and a temporary (3-month) shutdown of all smelting activity in the summer of 2001. The author observed that the air-to-blood ratio for children in the area over the full period was approximately 1:6. The author noted limitations in the dataset associated with exposures in the second time period, after the temporary shutdown of the facility in 2001, including sampling of a different age group at that time and a shorter time period (3 months) at these lower ambient air Pb levels prior to collection of blood Pb levels. Consequently, the EPA calculated an alternate air-to-blood Pb ratio based on ambient air Pb and blood Pb reductions in the first time period, after opening of the new facility in 1997 (ISA, section 3.5.1).

populations with age ranges extending well beyond 6 years. The first is the review and meta-analysis by Brunekreef (1984) using datasets available at the time for variously aged children as old as 18 years with identified air monitoring methods and reliable blood Pb data for 18 locations in the U.S. and internationally.[48] Two air-to-blood ratio estimates derived from this study based on log-log models both round to 1:5 (for air concentrations corresponding to the geometric means of the two sets of data pairs [1.5 and 0.54 μg/m^3]). A ratio on the order of 1:9 was derived based on the study by Schwartz and Pitcher (1989) of the relationship between U.S. NHANES II blood Pb levels for white subjects, aged ≤74 years, and national usage of leaded gasoline, adjusted for age and other covariates (Henderson, 2007a, pp. D-2 to D-3; ISA, Table 3-12).

The last two studies are focused on older children, ages 6-11 in India and Germany (Tripathi et al., 2001; Ranft et al., 2008) and employed methods to characterize media Pb concentrations that differed from the other studies assessed (PA, p. 3-11). The location-specific geometric mean blood Pb levels in the Indian study (8.6-14.4 μg/dL) indicate blood Pb distributions in this age group much higher than those pertinent to similarly aged children in the U.S. today and the air-to-blood ratio estimate reported was 1:3.6 (Tripathi et al., 2001). The more recent German study by Ranft et al. (2008) analyzed data from a nearly 20-year period associated with the leaded gasoline phase-out, during which average blood Pb levels declined from 9 μg/dL in 1983 (345 children, average age of 9 years) to 3 μg/dL in 2000 (162 children, average of 6

[48] In the dataset reviewed by Brunekreef (1984), air-to-blood ratios from the subset of those studies that used quality control protocols and presented adjusted slopes include values of 3.6, (Zielhuis et al., 1979), 5.2 (Billick et al., 1979, 1980); 2.9 (Billick, 1983), and 8.5 (Brunekreef et al., 1983). The studies cited here adjusted for parental education (Zielhuis et al., 1979), age and race (Billick et al., 1979, 1980) and air Pb monitor height (Billick, 1983); Brunekreef (1984) used multiple regression to control for several confounders (73 FR 66974).

years).[49] Average air Pb concentration declined from 0.45 μg/m^3 to 0.06 μg/m^3 over the same period, with the largest reduction occurring between the first study year (derived from two monitoring sites for full study area) and the second study year, 1991, for which air concentrations were derived from a combination of dispersion modeling and the two monitoring sites.[50] For a mean air Pb concentration of 0.1 μg/m^3, the study's multivariate loglinear regression model predicted air-to-blood ratios of 3.2 and 6.4 for "background" blood Pb concentrations of 1.5 and 3 μg/dL, respectively. In this study, background referred to Pb in blood from other sources; the blood Pb distribution over the study period, including levels when air Pb concentrations are lowest, indicates 3 μg/dL may be the better estimate of background for this study population. Inclusion of soil Pb as a variable in the model may have contributed to an underestimation of the blood Pb-air Pb ratios for this study because some of the Pb in soil likely originated in air and the blood Pb-air Pb slope does not include the portion of the soil/dust Pb ingestion pathway that derives from air Pb. Using univariate linear, log-log and loglinear models on the median air and blood Pb concentrations reported for the 5 years included in this study, the ISA also derived air-to-blood ratio estimates for data from this study ranging from 9 to 17 (ISA, p. 3-126; Ranft et al., 2008, Table 2). Uncertainties related to this study's estimates include those related to the bulk of air concentration reduction occurring between the first two time points (1983 and 1991) and the difference among the year's air datasets (e.g., two data sources [air monitors] in 1983 and multiple geographical points from a combination of the monitors and

[49] Blood Pb measurements were available on a total of 843 children across five time periods, in the first of which the average child age was 9 years while it was approximately 6 years in each of the latter years: 1983 (n=356), 1991 (n=147), 1994 (n=122), 1997 (n=56), and 2000 (n=162) (Ranft et al., 2008).

[50] The 1983 air Pb concentrations were based on two monitoring stations, while a combination of dispersion modeling and monitoring data was used in the later years. Surface soil Pb measurements were from 2000-2001, but geo-matched to blood Pb measurements across full study period (Ranft et al., 2008).

modeling in subsequent years).

In this review, as in the 2008 Pb NAAQS review, in addition to considering the evidence presented in the published literature and that reviewed in the 1986 CD, we also consider air-to-blood ratios derived from the exposure assessment (PA, p. 3-14; 73 FR 66974, November 12, 2008; 2007 REA, section 5.2.5.2). In the exposure assessment (summarized in section II.D below), current modeling tools and information on children's activity patterns, behavior and physiology were used to estimate blood Pb levels associated with multimedia and multipathway Pb exposure. The results from the various case studies assessed, with consideration of the context in which they were derived (e.g., the extent to which the range of air-related pathways was simulated, and the limitations associated with those simulations), and the multiple sources of uncertainty are also informative to our understanding of air-to-blood ratios. Estimates of air-to-blood ratios for the two REA case studies that represent localized population exposures exhibited an increasing trend across air quality scenarios representing decreasing air concentrations. For example, across the alternative standard levels assessed, which ranged from a calendar quarter average of 1.5 $\mu g/m^3$ down to a monthly average of 0.02 $\mu g/m^3$, the ratios ranged from 1:2 to 1:9 for the generalized (local) urban case study, with a similar trend, although of generally higher ratio, for the primary smelter case study subarea. This pattern of model-derived ratios is generally consistent with the range of ratios obtained from the literature, briefly discussed above. We continue to recognize a number of sources of uncertainty associated with these model-derived ratios which may contribute to high or low biases (as discussed further in section 3.1 of the PA).

The evidence on the quantitative relationship between air Pb and air-related Pb in blood is now, as in the past, limited by the circumstances (such as those related to Pb exposure) in

which the data were collected. Previous reviews have recognized the significant variability in air-to-blood ratios for different populations exposed to Pb through different air-related exposure pathways and at different air and blood levels, with the 1986 CD noting that ratios derived from studies involving the higher blood and air Pb levels pertaining to occupationally exposed workers are generally smaller than ratios from studies involving lower blood and air Pb levels (ISA, p. 3-132; 1986 CD, p. 11–99). Consistent with this observation, slopes in the range of 3 to 5 were estimated for child population datasets assessed in the 1986 CD (ISA, p. 3-132; 1986 CD p. 11–100; Brunekreef, 1984). Additional studies considered in the last review and those assessed in the ISA provide evidence of ratios above this older range (ISA, p. 3-133). For example, a ratio of 1:6.5-1:7 is indicated by the study by Hilts (2003), one of the few studies that evaluate the air Pb-blood Pb relationship in conditions that are closer to the current state in the U.S. (ISA, p. 3-132). We additionally note the variety of factors identified in the ISA that may potentially affect estimates of various ratios (including potentially coincident reductions in nonair Pb sources during the course of the studies), and for which a lack of complete information may preclude any adjustment of estimates to account for their role (ISA, section 3.5).

In summary, as at the time of the last review of the NAAQS for Pb, the currently available evidence includes estimates of air-to-blood ratios, both empirical and model-derived, with associated limitations and related uncertainties. These limitations and uncertainties, which are summarized here and also noted in the ISA, usually include uncertainty associated with reductions in other Pb sources during the study period. The limited amount of new information available in this review has not appreciably altered the scientific conclusions reached in the last review regarding relationships between Pb in ambient air and Pb in children's blood or with regard to the range of ratios. The currently available evidence continues to indicate ratios

relevant to the population of young children in the U.S. today, reflecting multiple air-related pathways in addition to inhalation, to be generally consistent with the approximate range of 1:5 to 1:10 given particular attention in the 2008 NAAQS decision, including the "generally central estimate" of 1:7 (73 FR 67002, 67004, November 12, 2008; ISA, pp. 3-132 to 3-133).

D. Summary of Risk and Exposure Assessment Information

The risk information available for this review and summarized here is based primarily on the exposure and risk assessment developed in the last review of the Pb NAAQS, described in the 2007 REA, the 2007 Staff Paper and the 2008 notice of final decision (USEPA, 2007a; USEPA, 2007b; 73 FR 66964, November 12, 2008), as considered in the context of the evidence newly available in this review (PA, section 3.4). As described in the REA Planning Document, careful consideration of the information newly available in this review, with regard to designing and implementing a full REA for this review, led to the conclusion that performance of a new REA for this review was not warranted. We did not find the information newly available in this review to provide the means by which to develop an updated or enhanced risk model that would substantially improve the utility of risk estimates in informing the current Pb NAAQS review (REA Planning Document, section 2.3). Based on their consideration of the REA Planning Document analysis, the CASAC Pb Review Panel generally concurred with the conclusion that a new REA was not warranted in this review (Frey, 2011b).[51] Accordingly, the risk/exposure information considered in this review is drawn primarily from the 2007 REA, augmented by a limited new computation for one case study focused on risk associated with the current standard, as described below (PA, section 3.4 and Appendix 3A).

1. Overview

[51] In their review of the draft PA, the CASAC Pb Review Panel reinforced their concurrence with the EPA's decision not to develop a new REA (Frey, 2013).

The focus for the risk assessment and associated estimates is on Pb derived from sources emitting Pb to ambient air. As discussed in section I.D above, the multimedia and persistent nature of Pb, the role of multiple exposure pathways, and the contributions of nonair sources of Pb to human exposure media all present challenges and contribute significant additional complexity to the health risk assessment that goes far beyond the situation for similar assessments typically performed for other NAAQS pollutants (e.g., that focus only on the inhalation pathway). The conceptual model that informed planning for the 2007 REA identified sources, pathways, routes, exposed populations, and health endpoints, focusing on those aspects of Pb exposure most relevant to the review, while also recognizing the role of Pb exposure pathways unrelated to Pb in ambient air (2007 REA, section 2.1). Limitations in the available data and models affected our characterization of the various complexities associated with exposure to ambient air Pb. As a result, the assessment included a number of simplifying assumptions in a number of areas and the estimates of air-related Pb risk produced are approximate and are characterized by upper and lower bounds.

As recognized in I.D above, sources of human Pb exposure include current and historical air emissions sources, as well as miscellaneous nonair sources, which can contribute to multiple exposure media and associated pathways (e.g., inhalation of ambient air, ingestion of indoor dust, outdoor soil/dust and diet or drinking water). In addition to airborne emissions (recent or those in the past), sources of Pb to these pathways also include old leaded paint, including Pb mobilized indoors during renovation/repair activities, and contaminated soils. Lead in diet and drinking water may have air pathway-related contributions as well as contributions from nonair sources (e.g., Pb solder on water distribution pipes and Pb in materials used in food processing). Limitations in our data and modeling tools handicapped our ability to fully separate the nonair

contributions to Pb exposure from estimates of air-related Pb exposure and risk. As a result, we have developed bounds within which we estimate air-related Pb risk to fall. The lower bound is based on a combination of pathway-specific estimates that do not completely represent all air-related pathways, while the upper bound is based on a combination of pathway-specific estimates that includes pathways that are not air-related but the separating out of which is precluded by modeling and data limitations.

Inclusion of exposure populations, exposure/dose metric, health effects endpoint and risk metric in the 2007 REA were based on consideration of the then-currently available evidence as assessed in detail in the 2006 CD. As discussed in the REA Planning Document (USEPA, 2011b), these selections continue to be supported by the evidence now available in this review as described in the ISA. The REA focused on risk to the central nervous system in childhood as the most sensitive effect that could be quantitatively assessed, with decrement in IQ used as the risk metric. Exposure and biokinetic modeling was used to estimate blood Pb concentrations in children exposed to Pb up to age 7 years.[52] This focus reflected the evidence for young children with regard to air-related exposure pathways and susceptibility to Pb health impacts (e.g., ISA, sections 3.1.1, 4.3, 5.2.1.1, 5.3.1.1, and 5.4). For example, the hand-to-mouth activity of young children contributes to their Pb exposure (i.e., incidental soil and indoor dust ingestion) and ambient air-related Pb has been shown to contribute to Pb in outdoor soil and indoor house dust (ISA, sections 3.1.1 and 3.4.1; 2006 CD, section 3.2.3).

The 2007 REA relied on a case study approach to provide estimates that inform our understanding of air-related exposure and risk in different types of air Pb exposure situations. Lead exposure and associated risk were estimated for multiple case studies that generally

[52] The pathways represented in this modeling included childhood inhalation and ingestion pathways, as well as maternal contributions to newborn body burden (2007 REA, Appendix H, Exhibit H-6).

represent two types of residential population exposures to air-related Pb: (1) location-specific urban populations of children with a broad range of air-related exposures, reflecting existence of urban concentration gradients; and (2) children residing in localized areas with air-related exposures representing air concentrations specifically reflecting the standard level being evaluated (see PA, Table 3-6). Thus, the two types of case studies differed with regard to the extent to which they represented population variability in air-related Pb exposure.

In drawing on the 2007 REA for our purposes in this review, we focused on two case studies, one from each of these two categories: (1) the location-specific urban case study for Chicago and (2) the generalized (local) urban case study (PA, Table 3-6). Accordingly, our summary of analysis details below focuses on details particular to these two case studies. The generalized (local) urban case study (also referred to as *general urban case study*) was not based on a specific geographic location and reflected several simplifying assumptions in representing exposure including uniform ambient air Pb levels associated with the standard of interest across the hypothetical study area and a uniform study population. Based on the nature of the population exposures represented by the two categories of case study, the generalized (local) urban case study includes populations that are relatively more highly exposed by way of air pathways to air Pb concentrations near the standard level evaluated, compared with the populations in the location-specific urban case. The location-specific urban case studies provided representations of urban populations with a broad range of air-related exposures due to spatial gradients in both ambient air Pb levels and population density. For example, the highest air concentrations in these case studies (i.e., those closest to the standard being assessed) were found in very small parts of the study areas, while a large majority of the case study populations resided in areas with much lower air concentrations.

2. Summary of Design Aspects

The approach to assessing exposure and risk for the two categories of case studies was comprised of four main analytical steps: (1) estimation of ambient air Pb concentrations, (2) estimation of Pb concentrations in other key exposure media, including outdoor soil and indoor dust, (3) use of exposure media Pb concentrations, with other pathway Pb intake rates (e.g., diet), to estimate blood Pb levels in children using biokinetic modeling, and (4) use of C-R functions derived from epidemiological studies to estimate IQ loss associated with the blood Pb levels.

Concentrations of Pb were estimated in ambient media and indoor dust using a combination of empirical data and modeling projections. The use of empirical data brings with it uncertainty related to the potential inclusion of nonair source signals in these measurements (e.g., house paint contributions to indoor dust and outdoor soil Pb). Conversely, the use of modeling tools introduces other uncertainties (e.g., model and parameter uncertainties).

Characterization of Pb in ambient air relied on (1) the use of ambient monitor data for the location-specific urban case studies and (2) an assumption of uniform ambient air Pb levels (matching the standard level being considered) for the generalized (local) urban case study. For the location-specific urban case studies, we used Pb monitors within each study area to characterize spatial gradients. By contrast, the generalized (local) urban case study is designed to assess exposure and risk for a smaller group of residents (e.g., neighborhood) exposed at the level of the standard and, therefore, did not rely on monitor data; rather, ambient air Pb concentration was fixed at the standard being assessed. For the generalized (local) urban case study, which has a single exposure zone in which air Pb concentrations do not vary spatially, we derived a single air Pb concentration estimate to meet the standard assessed. Concentrations in the location-specific urban study areas, which relied on empirical (monitor-based) data to define ambient air Pb concentrations, reflected contributions from all sources affecting the

concentrations in those locations, be they currently active stationary or mobile sources, resuspension of previously deposited Pb or other.[53]

The air quality scenarios assessed in the 2007 REA included conditions just meeting the NAAQS that was current at the time of the last review (1.5 $\mu g/m^3$, as a calendar quarter average), conditions meeting several alternative, lower standards,[54] and current conditions in the three location-specific urban case studies (PA, section 3.4.3.2). The full impact of changes in air Pb conditions associated with attainment of lower standards was not simulated, however, due to limitations in the available data and modeling tools that precluded simulation of linkages between some media and air Pb. Specifically, while Pb concentrations in indoor dust were simulated to change with the different air quality scenarios for which there were differing ambient air Pb concentrations (outdoors and indoors), dietary and drinking water Pb concentrations, as well as soil Pb concentrations, were not varied across the air quality scenarios in any case study (see PA, Table 3-7).[55]

In estimating blood Pb levels using the IEUBK model, Pb concentrations in exposure media (e.g., ambient air, diet, water, indoor dust) were held constant throughout the 7-year simulation period, while behavioral and physiological variables were changed with age of child (2007 REA, sections 3.2.1.1 and 5.2.4). Detail on methods used to characterize media Pb concentrations and all IEUBK inputs for each case study are in the 2007 REA, sections 3.1, 3.2,

[53] Additional detail on estimation of ambient (outdoor) and indoor air concentrations is presented in section 5.2.2 and Appendices A through D of the 2007 REA.
[54] The alternatives lower than the NAAQS at the time of the last review for which air quality scenarios were assessed were a maximum calendar quarter average of 0.2 $\mu g/m^3$ and maximum monthly averages of 0.5, 0.2, 0.05 and 0.02 $\mu g/m^3$ (PA, Table 3-8).
[55] Characterization of Pb concentrations in outdoor surface soil/dust for the generalized (local) and location-specific urban cases studies was based on the use of nationally representative residential soil measurements obtained from the literature (2007 REA, sections 3.1.3 and 5.2.2.2 and Appendix F). Diet and drinking water intake and concentrations, as well as other model inputs, were based on the most current information (2007 REA, Appendix H).

5.2.3 and 5.2.4, and appendices C through H. Population variability in Pb intake and uptake was simulated through use of the IEUBK model to first generate a central-tendency estimate of the blood Pb levels for the group of children within a given exposure zone of a study area, coupled with use of a geometric standard deviation (GSD) and for the location-specific case studies, Monte Carlo-based population sampling (PA, section 3.4; 2007 REA, Appendix H). The risk characterization step employed in the 2007 REA generated a distribution of IQ loss estimates for the set of children simulated in the assessment.

Specifically, blood Pb estimates for the concurrent blood Pb metric[56] were combined with four C-R functions for blood Pb concentration with IQ loss based on the analysis by Lanphear et al. (2005) of a pooled international dataset of blood Pb and IQ (see the 2007 REA, section 5.3.1.1). We used the four different C-R functions to provide different characterizations of behavior at low exposures in recognition of uncertainty related to modeling this endpoint, particularly at lower blood Pb levels for which there is limited representation in the Lanphear et al. (2005) pooled dataset.[57] In considering the risk estimates here (as in the last review), we focus on estimates for one of the four functions (referred to as the loglinear with low-exposure linearization C-R function [PA, section 3.4.3.3]). The range of risk estimates reflecting all four C-R functions provide perspective on the impact of uncertainty in this key modeling step. Additional detail on the C-R functions is provided in the PA and the 2007 Pb Staff Paper (PA,

[56] As in the last review, we give primary emphasis to estimates based on the concurrent blood Pb metric, consistent with CASAC advice in the last review (Henderson, 2007b).
[57] The 5th percentile for the concurrent blood Pb measurements in that dataset is 2.5 μg/dL, and the median is 9.7 μg/dL (Lanphear et al., 2005).

section 3.4.3.3; USEPA, 2007b, section 4.2.1).[58] We focus on the median IQ loss estimates, as in the last review, due to increased confidence in these estimates relative to the higher percentile estimates, for which we recognize significant uncertainty (PA, sections 3.4.5, 3.4.6 and 3.4.7; 2007 Staff Paper, p. 4-20).

As the 2007 REA did not include an air quality scenario simulated to just meet the standard selected by the 2008 decision,[59] we employed two different approaches to estimate risk pertaining to conditions just meeting the current Pb standard (set in 2008) for our purposes in this review. First, given the similarity to the current standard of the then-current conditions scenario for the Chicago case study (among all the 2007 REA scenarios), we consider the risk estimates for that scenario as informative with regard to risk associated with the current standard.[60] To augment the risk information available in this current review and in recognition of the variation among specific locations and urban areas with regard to air quality patterns and exposed

[58] As noted in section II.B.3 above, since the completion of the ISA in the current review, two errors have been identified with the pooled dataset analyzed by Lanphear et al., (2005) (Kirrane and Patel, 2014). The EPA and a recent publication have separately recalculated the statistics and mathematical models of Lanphear et al., (2005) using the corrected pooled dataset (Kirrane and Patel, 2014). While the conclusions drawn from these coefficients, including the finding of a steeper slope at lower (as compared to higher) blood Pb concentrations, are unaffected, the magnitude of the loglinear and linear regression coefficients are somewhat lower based on the corrections. For example, the loglinear model coefficient used for the C-R function, on which the EPA focused in the last review and also focuses on here, changed only negligibly from -2.7 to -2.65 when recalculated using the corrected pooled dataset (Kirrane and Patel, 2014). As a result, the risk estimates for this function would be expected to be very similar although slightly lower if derived using the recalculated loglinear model coefficient for the corrected dataset. Since the loglinear model coefficient calculated from the corrected dataset is unchanged at two significant figures from that original reported, any change to the risk estimates would be very small and, particularly in light of other uncertainties in the analysis, does not materially affect staff's consideration of the results.

[59] The 2008 decision on the level for the revised NAAQS was based primarily on consideration of the evidence-based air-related IQ loss framework; risk estimates available for scenarios simulated in the 2007 REA were concluded to be roughly consistent with and generally supportive of the evidence-based air-related IQ loss estimates (see section II.A.1 above).

[60] In the Chicago urban case study, the maximum monthly average concentration was 0.31 $\mu g/m^3$, and the maximum calendar quarter average concentration was 0.14 $\mu g/m^3$ (2003-2005 data; 2007 REA, Appendix O).

population, we have also newly developed estimates for an air quality scenario just meeting the current Pb NAAQS in the context of the generalized (local) urban case study. These estimates were derived based on interpolation from the risk estimates available for scenarios previously assessed for the generalized (local) urban case study. Such interpolated estimates were only developed for the generalized urban case study due to its use of a single exposure zone which greatly simplified the method employed.[61]

The general approach we followed to newly develop estimates for the current standard in the generalized (local) urban case study was to identify the two alternative standard scenarios simulated in the 2007 REA which represented air quality conditions bracketing those for the current standard and then linearly interpolate an estimate of risk for the current standard based on the slope created from the two bracketing estimates (PA, section 3.4.3.3.2 and Appendix 3A). By this method, the air quality scenario for the current standard (0.15 $\mu g/m^3$, as a not-to-be-exceeded 3-month average) was found to be bracketed by the scenarios for alternative standards of 0.20 $\mu g/m^3$ (maximum calendar quarter average) and 0.20 $\mu g/m^3$ (maximum monthly average). Using interpolation between the risk estimates for these two scenarios, we developed median risk estimates for the current standard (PA, Appendix 3A).

3. Key Limitations and Uncertainties

In characterizing risk associated with Pb from air-related exposure pathways, we faced a variety of challenges and employed a number of methods. The challenges related to significant data and modeling limitations which affected our ability to parse out the portion of total (all-

[61] We did not interpolate risk estimates for the current standard for the other case studies (i.e., the primary Pb smelter and location-specific urban case studies) because those case studies utilized a more complex, spatially-differentiated and population-based approach (see 2007 REA) which precludes application of the simple linear interpolation approach described, without introduction of substantial added uncertainty (relative to the other estimates for the same case study). The simplicity of the generalized (local) urban study area, however, with its single exposure zone, is amenable to the linear interpolation of risk described here.

pathway) blood Pb and IQ loss attributable to air-related pathways, as well as our representation of key sources of variability and characterization of uncertainty. Although we separated total estimates into risk estimates for diet/drinking water and two air-related categories ("recent air" and "past air"), significant limitations in our modeling tools and data resulted in an inability to parse risk estimates specific to the air-related pathways. For example, we recognize that Pb in diet and drinking water sources may include some Pb derived from Pb in the ambient air, as well as Pb from nonair sources, but limitations precluded explicit modeling of the contribution from air pathways to these exposure pathways, such that the air-related component of these exposures was not estimated. Rather, we focused on estimates from the two air-related categories, which we considered to under- and over-estimate air-related risk, respectively, to create bounds within which we consider air-related risk to fall.

The first air-related category ("recent") included Pb exposure pathways tied most directly to ambient air, which consequently have the potential to respond relatively more quickly to changes in air Pb (i.e., inhalation and ingestion of indoor dust Pb derived from the infiltration of ambient air Pb indoors). Importantly, media concentrations associated with the pathways in this category were simulated to change in response to air concentrations (as noted in section II.D.2 above and described in section 3.4.3.1 of the PA). The air-related Pb exposure pathways in the second air-related category ("past air"), all of which are associated with atmospheric deposition, included ingestion of Pb in outdoor dust/soil and ingestion of the portion of Pb in indoor dust that after deposition from ambient air outdoors is carried indoors with humans. While there is the potential for these other air-related exposures to be affected (over some time frame) by changes in air Pb concentrations (associated with an adjustment to the Pb standard), limitations in our data and tools precluded simulation of that relationship. Consequently, risk estimated for this

category reflects media measurements available for the 2007 REA and is identical for all air quality scenarios. Further, although paint is not an air-related source of Pb exposure, it may be reflected somewhat in estimates developed for the "past air" category, due to modeling constraints (2007 Staff Paper, section 4.2.4). Thus, as exposures included in the first air-related category ("recent") do not completely capture all air-related pathways, we consider risk for this category an underestimate of air-related risk. Yet, as exposures included in the second air-related category include pathways that are not air-related, we consider the summed risk across both categories to include a slight over-estimate of air-related risk.

In summary, because of limitations in the assessment design, data and modeling tools, we consider our estimates of risk attributable to air-related exposure pathways to be approximate and to be 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. With regard to the latter, we are additionally cognizant of the modeling and data limitations which reduce the extent to which the upper end of these bounds reflects impacts of alternative air quality conditions simulated. We note that this limitation will tend to contribute to estimates for the "past air" category representing relatively greater overestimates with relatively lower air Pb air quality scenarios.

We recognize several important sources of variability in air-related Pb exposures and associated risk, for which the approaches by which they were addressed in the 2007 REA are summarized here (PA, section 3.4.6).

- Variation in distributions of potential urban residential exposure and risk across U.S. urban residential areas is addressed by the inclusion of location-specific urban study areas that reflect a diverse set of urban areas in the U.S.

- Representation of a more highly exposed subset of urban residents potentially exposed at the level of the standard is addressed by the inclusion of the generalized (local) urban study area.
- Variation in residential exposure to ambient air Pb within an urban area of the location-specific case studies is addressed through the partitioning of these study areas into exposure zones to provide some representation of spatial gradients in ambient air Pb and their interaction with population distribution and demographics.
- Inter-individual variability in blood Pb levels is addressed through the use of empirically derived GSDs to develop blood Pb distribution for the child population in each exposure zone, with GSDs selected particular to each case study population.
- Inter-individual variability in IQ response to blood Pb is addressed through the use of C-R functions for IQ loss based on a pooled analysis reflecting studies of diverse populations.

With regard to uncertainties, we recognize one overarching area concerning the precision of our estimation of the neurocognitive risk (as represented by IQ loss) associated with ambient air Pb. For reasons related to the evidence of nonlinear responses of blood Pb to Pb exposure and of Pb-associated IQ response to blood Pb, the 2007 REA first estimated blood Pb levels and associated risk for total Pb exposure (i.e., including Pb from air-related and nonair exposure pathways) and then separated out estimates for pathways of interest (PA, section 3.4.4). However, as described above, significant limitations in our modeling tools affected our ability to develop precise estimates for air-related exposure pathways. We believe these limitations led to a slight overestimation of the risks for the "past air" category and to an under-representation of air-related pathways for the "recent air" category. Thus, we characterized the risk attributable to air-

related exposure pathways to be bounded by the estimates developed for the "past air" category and the sum of estimates for the "recent air" and "past air" categories. For air quality scenarios other than those for the previous NAAQS, this upper bound is recognized as having a potential upward bias with regard to its reflection of the simulated air quality conditions because modeling and data limitations precluded simulation of the influence of lower air Pb concentrations on the outdoor dust and soil exposure pathways (PA, section 3.4.4).

We recognize a range of additional uncertainties, limitations, and assumptions that are reflected in various ways in the 2007 REA and associated results (PA, section 3.4.7), which include the following.

- *Temporal Aspects:* During the 7-year exposure period, media concentrations remain fixed and the simulated child resides at the same residence (although exposure factors, including behavioral and physiological parameters, are adjusted to match the aging of the child). These aspects introduce uncertainty into the risk estimates, although the existence of a directional bias is unclear.
- *Generalized (local) Urban Case Study:* The design for this case study employs assumptions regarding uniformity that are reasonable in the context of a general description of a small neighborhood population but would contribute significant uncertainty to extrapolation of these estimates to a specific urban location, particularly a relatively large one. An additional area of uncertainty concerns the representation of variability in air quality. Given the relatively greater variability common in areas of high Pb concentrations, the approach used to reflect variability may bias the estimates high.
- *Location-specific Urban Case Studies:* Limitations in the spatial density of ambient air monitors in the simulated areas limit our characterization of spatial gradients of ambient

air Pb levels in these case studies. This factor introduces uncertainty into the risk estimates for this category of case study; the existence of a directional bias is unclear.

- *Air Quality Simulation:* Focus on only then-current conditions (2003-2005) scenario for the Chicago urban case study in this review precludes uncertainty associated with simulations of alternative air quality scenarios in the 2007 REA.
- *Outdoor Soil/Dust Pb Concentrations*: Limitations in datasets on Pb levels in surface soil/dust Pb in urban areas and in our ability to simulate the impact of reduced air Pb levels related to lowering the NAAQS in the 2007 REA contribute uncertainty to air-related risk estimates for the current standard in the generalized (local) urban case study. The likely impact is a high bias on these risk estimates (related to low bias on estimating risk reduction for lower standard levels in the 2007 REA) given lack of simulated changes in soil Pb related to changes in ambient air Pb.
- *Indoor Dust Pb Concentrations:* Limitations and uncertainty in modeling of indoor dust Pb levels, including the impact of reductions in ambient air Pb levels, contributes uncertainty to air-related risk estimates. Although the indoor dust modeling does link changes in ambient air Pb to changes in indoor dust Pb, it does not include a link between ambient air Pb, outdoor soil Pb and subsequent changes in the level of Pb carried (or "tracked") into the house. This could introduce low bias into the total estimates of air-related Pb exposure and risk.
- *Interindividual Variability in Blood Pb Levels:* Uncertainty related to population variability in blood Pb levels related to interindividual variability in factors other than media concentration and limitations in modeling of this introduces significant uncertainty

into blood Pb and IQ loss estimates for the 95th percentile of the population. The extent of any systematic bias from this source of uncertainty is unknown.

- *Pathway Apportionment for Higher Percentile Blood Pb and Risks:* Limitations, primarily in data, prevented us from characterizing the degree of correlation among high-end Pb exposures for the various pathways (e.g., the degree to which an individual experiencing high drinking water Pb exposure would also experience high Pb paint exposure and high ambient air-related Pb exposure). Our inability to characterize potential correlations between exposure pathways (particularly at the higher percentile exposure levels) limited our ability to (1) effectively model high-end Pb risk and (2) apportion that risk between different exposure pathways, including ambient air-related pathways.
- *IQ Loss C-R Functions:* Specification of the quantitative relationship between blood Pb level and IQ loss is subject to greater uncertainty at lower blood Pb levels. The use of four C-R functions models (which each treat the response at low blood Pb levels in a different manner) is considered to provide a reasonable characterization of this source of uncertainty and its impact on risk estimates. Comparison of risk estimates from the four models indicates this source of uncertainty to have a potentially significant impact on risk.

4. Summary of Risk Estimates and Key Observations

In this summary of risk estimates, drawn from the PA, we focus on the estimates of air-related IQ loss derived using the C-R function in which we have greatest confidence (see PA, sections 3.4.3.3.1 and 3.4.7) for the median child in a given case study (exposure modeled through age 7 years), given the substantially greater uncertainty associated with air-related risk

estimates for extremes of the risk distribution, such as the 95th percentile (PA, section 3.4). Estimates for other risk metrics and the full range of case studies and air quality scenarios are described elsewhere in detail (e.g., 2007 REA, sections 4.2 and 5.3.2 and appendices; 2007 Staff Paper, chapter 4; 73 FR 66964, November 12, 2008). Based on results from the 2007 REA for a location-specific urban study area (Chicago case study) and on those newly derived in this review based on interpolation from the 2007 REA results (for the generalized [local] urban case study), median air-related IQ loss for the current standard is estimated, with rounding, to generally fall near or somewhat above a rough lower bound of 1 point IQ loss and below a rough upper bound of 3 points IQ loss. As would be expected by the use of interpolation, the newly derived estimates are consistent with the estimates for similar air quality scenarios that were available in the last review (PA, section 3.4.5). For example, the generalized (local) urban case study current standard scenario estimates for median air-related IQ loss are identical to those for the scenario of just meeting a potential alternative of 0.2 $\mu g/m^3$ maximum calendar quarter average for that case study (PA, Table 3-11). Further, the upper bound below which the median IQ loss is estimated to fall is also approximately 3 IQ points in the generalized (local) urban case study scenarios for just meeting potential alternatives of 0.2 $\mu g/m^3$, 0.05 and 0.02 $\mu g/m^3$ maximum monthly average, providing an indication of the limitations associated with estimating air-related Pb exposures and risk for lower air Pb scenarios (PA, sections 3.4.4 and 3.4.5).

As summarized in section II.D.3 above, a range of limitations and areas of uncertainty were associated with the information available in the last review (PA, sections 3.4.4, 3.4.6 and 3.4.7). In this review, the REA Planning Document concluded that none of the primary sources of uncertainty identified to have the greatest impact on risk estimates would be substantially reduced through the use of newly available information (USEPA, 2011b). Thus, the key

observations regarding air-related Pb risk modeled for the set of standard levels assessed in the 2007 REA, as well as the risk estimates interpolated for the current standard, are not significantly affected by the new information. Further, our overall characterization of uncertainty and variability associated with those estimates (as summarized above and in sections 3.4.6 and 3.4.7 of the PA) is not appreciably affected by new information. As recognized at the time of the last review, exposure and risk modeling conducted for this analysis was complex and subject to significant uncertainties due to limitations in the data and models, among other aspects. Of particular note, limitations in the assessment design, data and modeling tools handicapped us from sharply separating Pb linked to ambient air from Pb that is not air related.

In summary, the estimates of risk attributable to air-related exposures, with which we recognize a variety of sources of uncertainty, are considered to be approximate, falling within upper and lower bounds. These bounds for scenarios just meeting the current standard are roughly estimated, with rounding, as 3 and 1 IQ points, which over- and underestimate risk, respectively. In characterizing the magnitude of air-related risk associated with the current standard, we focus on median estimates, for which we have appreciably greater confidence than estimates for outer ends of the risk distribution (see PA, section 3.4.7) and on risks derived using the C-R function in which we have greatest confidence (see PA, sections 3.4.3.3.1 and 3.4.7). These risk results for the current standard, both those estimated in the last review for one of the location-specific urban study area populations and those newly derived in this review using interpolation of the estimates from the last review for the generalized (local) urban case study, which is recognized to reflect a generalized high end of air-related exposure for localized populations, provide approximate bounds for air-related risk, with attendant uncertainties described above. Focusing on the results for the generalized (local) urban case study, the

interpolated estimates for the scenario representing the current standard are very similar to estimates for the two 0.2 $\mu g/m^3$ scenarios (maximum monthly and calendar quarter averages) simulated in the 2007 REA[62] and are appreciably lower than those associated with the previous standard. For this case study, across the two 0.2 $\mu g/m^3$ scenarios, the current standard scenario and the more restrictive air quality scenarios, the upper bound below which air-related risk is estimated to fall rounds to the same value, reflecting the significant limitations associated with developing precise estimates of air-related risk, particularly for the lower air Pb scenarios (PA, sections 3.4.4, 3.4.5, and 3.4.7).

E. Conclusions on Adequacy of the Current Primary Standard

In evaluating whether, in view of the advances in scientific knowledge and additional information now available, it is appropriate to retain or revise the current standard, the Administrator builds upon the last review and reflects upon the body of evidence and information now available. The Administrator has taken into account both evidence-based and quantitative exposure- and risk-based considerations in developing conclusions on the adequacy of the current primary Pb standard. Evidence-based considerations draw upon the EPA's assessment and integrated synthesis of the scientific evidence from epidemiological studies and experimental animal studies evaluating health effects related to exposures to Pb, with a focus on policy-relevant considerations as discussed in the PA. The exposure/risk-based considerations draw from the results of the quantitative analyses presented in the 2007 REA, augmented as described in the PA, and summarized in section II.D above, and consideration of those results in the PA. More specifically, estimates of the magnitude of ambient Pb-related exposures for young children and associated impacts on IQ associated with just meeting the current primary Pb

[62] There is uncertainty associated with judging differences between the current standard and these potential alternative standards due to the difference in air quality datasets used to estimate air concentration variability of the 2007 REA estimates versus the interpolated risk estimate.

NAAQS have been considered. Together the evidence-based and risk-based considerations have informed the Administrator's proposed conclusions related to the adequacy of the current Pb standard in light of the currently available scientific evidence.

As described in section II.A.2 above, consideration of the evidence and the exposure/risk information in the PA and by the Administrator is framed by consideration of a series of key policy-relevant questions. The following sections describe the consideration of these questions in the PA, the advice received from CASAC, as well as the comments received from various parties, and then present the Administrator's proposed conclusions regarding the adequacy of the current primary standard.

1. Evidence-based Considerations in the Policy Assessment

In considering the evidence with regard to the issue of adequacy of the current standard, the PA addresses several questions that build on the information summarized in sections II.B and II.C above (and sections 3.1 through 3.3 of the PA) to more broadly address the extent to which the current evidence base supports the adequacy of the public health protection afforded by the current primary standard. The first question addresses the integrated consideration of the health effects evidence, in light of aspects described in sections II.A.1 and II.A.2 above. The second question focuses on consideration of associated areas of uncertainty. The third question then integrates consideration of the prior two questions with a focus on the standard, including each of the four elements. The PA considerations and conclusions with regard to these questions are summarized below.

In considering the extent to which information newly available in this review may have altered scientific support for the occurrence of health effects associated with Pb in ambient air, the PA concludes that the current evidence continues to support the EPA's conclusions from the previous review regarding key aspects of the health effects evidence for Pb and the health effects

of multimedia exposure associated with levels of Pb occurring in ambient air in the U.S. (PA, section 4.2.1). The conclusions in this regard are based on consideration of the assessment of the currently available evidence in the ISA, particularly with regard to key aspects summarized in Chapter 3 of the PA, in light of the assessment of the evidence in the last review as described in the 2006 CD and summarized in the notice of final rulemaking (73 FR 66964, November 12, 2008). Key aspects of these conclusions are summarized below.

As at the time of the last review, blood Pb continues to be the predominant biomarker employed to assess exposure and health risk of Pb (ISA, Chapters 3 and 4), as discussed in section II.C above. This widely accepted role of blood Pb in assessing exposure and risk is illustrated by its established use in programs to prevent both occupational Pb poisoning and childhood Pb poisoning, with the latter program, implemented by the CDC, recently issuing updated guidance on blood Pb measurement interpretation (CDC, 2012). As in the past, the current evidence continues to indicate the close linkage of blood Pb levels in young children to their body burden; this linkage is associated with the ongoing bone remodeling during that lifestage (ISA, section 3.3.5). This tight linkage plays a role in the somewhat rapid response of children's blood Pb to changes in exposure (particularly to exposure increases), which contributes to its usefulness as an exposure biomarker (ISA, sections 3.2.2, 3.3.5, and 3.3.5.1). Additionally, the weight of evidence documenting relationships between children's blood Pb and health effects, most particularly those on the nervous and hematological systems (e.g., ISA, sections 4.3 and 4.7), speaks to its usefulness in assessing health risk.

As in the last review, the evidence on air-to-blood relationships available today continues to be composed of studies based on an array of circumstances and population groups (of different age ranges), analyzed by a variety of techniques, which together contribute to appreciable

variability in the associated quantitative estimates and uncertainty with regard to the relationships existing in the U.S. today. Accordingly, interpretation of this evidence base, as discussed in section II.C above, also includes consideration of factors that may be influencing various study estimates. We consider the study estimates in light of such factors both with regard to the extent to which the factors affect the usefulness of specific study estimates for the general purpose here of quantitatively characterizing relationships between Pb in ambient air and air-related Pb in children's blood and also with regard to the pertinence of such factors more specifically to conditions and populations in the U.S. today. As noted in the PA, the current evidence, while including two additional studies not available at the time of the last review, is not appreciably changed from that available in the last review (PA, section 3.1). The range of estimates that can be derived from the full dataset is broad and not changed by the inclusion of the newly available estimates. Further, the PA recognizes significant uncertainties regarding the air Pb to air-related blood Pb relationship for the current conditions where concentrations of Pb in both ambient air and children's blood are substantially lower than they have been in the past. In considering the strengths, limitations and uncertainties associated with the full dataset, the currently available evidence appears to continue to support a range of estimates for the purpose at hand that is generally consistent with the range given weight in the last review, 1:5 to 1:10 (ISA, section 3.7.4 and Table 3-12; 73 FR 67001-2, 67004, November 12, 2008). The PA additionally notes that the generally central estimate of 1:7 identified for this range in the last review is consistent with the study involving blood Pb for pre-school children and air Pb conditions near a large source of Pb to ambient air with concentrations near (and/or previously above) the level of the current Pb standard (ISA, section 3.5.1; Hilts, 2003).[63] In so noting, the

[63] The older study by Hayes et al. (1994) during time of leaded gasoline indicated a generally similar ratio of 1:8, although the blood Pb levels in that study were much higher than those in the

PA also recognizes the general overlap of such circumstances with those represented by the evidence-based, air-related IQ loss framework,[64] for which air-to-blood ratio is a key input. In characterizing the range of air-to-blood ratio estimates, we recognize uncertainty inherent in such estimates as well as the variation in currently available estimates resulting from a variety of factors, including differences in the populations examined, as well as in the Pb sources or exposure pathways addressed in those study analyses (ISA, section 3.7.4).

The scientific evidence continues to recognize a broad array of health effects on multiple organ systems or biological processes related to blood Pb, including Pb in blood prenatally (ISA, section 1.6). The currently available evidence continues to support identification of neurocognitive effects in young children as the most sensitive endpoint associated with blood Pb concentrations (ISA, section 1.6.1), which as an integrated index of exposure reflects the aggregate exposure to all sources of Pb through multiple pathways (inhalation and ingestion). Evidence continues to indicate that some neurocognitive effects in young children may not be reversible and may have effects that persist into adulthood (ISA, section 1.9.5). Thus, as discussed in section II.B. above, the evidence of Pb effects at the low end of the studied blood Pb levels (closest to those common in the U.S. today) continues to be strongest and of greatest concern for effects on the nervous system, most particularly those on cognitive function in children.

As in the last review, evidence on risk factors continues to support the identification of young children as an important at-risk population for Pb health effects (ISA, section 5.4). The current evidence also continues to indicate important roles as factors that increase risk of Pb-

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study by Hilts (2003). Among the studies focused on this age group, the latter study includes blood Pb levels closest to those in U.S. today.

[64] Concentrations near air sources are higher than those at more distant sites (as described in PA, section 2.2.2); it is near-source locations where there is the potential for concentrations at or near the current standard.

related health effects for the following: nutritional factors, such as iron and calcium intake; elevated blood Pb levels; and proximity to sources of Pb exposure, such as industrial releases or buildings with old, deteriorating, leaded paint. Further, some races or ethnic groups continue to demonstrate increased blood Pb levels relative to others, which may be related to these and other factors (ISA, sections 5.1, 5.2 and 5.4).

With regard to our understanding of the relationship between exposure or blood Pb levels in young children and neurocognitive effects, the PA notes that the evidence in this review, as in the last, does not establish a threshold blood Pb level for neurocognitive effects in young children (ISA, sections 1.9.4 and 4.3.12). The lowest blood Pb levels at which associations with neurocognitive impacts have been observed in pre-school and school age children continue to range down below 5 μg/dL, with the lowest group levels that have been associated with such effects ranging down to 2 μg/dL (ISA, sections 1.6.1 and 4.3.15.1). Additionally, as in the last review, there is evidence that the relationship of young children's blood Pb with neurocognitive impacts, such as IQ, is nonlinear across a wide range of blood Pb, with greater incremental impacts at lower versus higher blood Pb levels (ISA, sections 1.9.4 and 4.3.12). Accordingly, as in the last review, the PA focuses on C-R relationships from study groups with blood Pb levels closest to those in children in the U.S. today, which are generally lower than epidemiological study groups. The currently available evidence does not identify additional C-R slopes for study groups of young children (e.g., $\leq$ 7 years) with mean blood Pb levels below that of groups identified in the last review, 2.9 – 3.8 μg/dL, as discussed in section II.B.3 above (ISA, section 4.3.12). Thus, the blood Pb concentration - IQ response functions or slopes identified in this review for epidemiological study groups of young children with mean blood Pb levels closest to that of children in the U.S. today include the same set recognized at the time of the last review

(see Table 1 above), the median of which is 1.75 IQ points decrement per μg/dL blood Pb (73 FR 67003, November 12, 2008).

In considering the evidence with regard to the extent to which important uncertainties identified in the last review have been reduced or to which new uncertainties have emerged, as summarized in discussing the previous question and in section II.B above, the PA concludes that no new uncertainties were identified as emerging since the last review. However, the PA recognizes important uncertainties identified in the last review that remain today. Importantly, given our focus in this review, as in the last review, on neurocognitive impacts associated with Pb exposure in early childhood, the PA recognizes remaining uncertainties in our understanding of the C-R relationship of neurocognitive impacts, such as IQ decrements, with blood Pb level in young children, particularly across the range of blood Pb levels common in the U.S. today. With regard to C-R relationships for IQ, the evidence available in this review does not include studies that appreciably extend the range of blood Pb levels studied beyond those available in the last review. As in the last review, the early childhood (e.g., 2 to 7 years of age) blood Pb levels for which associations with IQ response have been reported continue to extend at the low end of the range to study group mean blood Pb levels of 2.9 to 3.8 μg/dL (e.g., 73 FR 67003, November 12, 2008, Table 3). The studies examining C-R relationships down to these blood Pb levels, as summarized in section II.B.3 above, continue to indicate higher C-R slopes in those groups with lower blood Pb levels than in study groups with higher blood Pb levels (ISA, section 4.3.12). The lack of studies considering C-R relationships for Pb effects on IQ at still lower blood Pb levels contributes to uncertainty regarding the quantitative relationship between blood Pb and IQ response in populations with mean blood Pb levels closer to the most recently available mean for children aged 1 to 5 years of age (e.g., 1.17 μg/dL in 2009-2010 [ISA, p. 3-85]).

Further, the PA recognizes important uncertainties in our understanding of the relationship between ambient air Pb concentrations and air-related Pb in children's blood. The evidence newly available in this review has not reduced such key uncertainties. As in the last review, air-to-blood ratios based on the available evidence continue to vary, with our conclusions based on the current evidence generally consistent with the range of 1:5 to 1:10 given emphasis in the last review (73 FR 67002, November 12, 2008; ISA, section 3.7.4). There continues to be uncertainty regarding the extent to which this range represents the relationship between ambient air Pb and Pb in children's blood (derived from the full set of air-related exposure pathways) and with regard to its reflection of exposures associated with ambient air Pb levels common in the U.S. today and to circumstances reflecting just meeting the current Pb standard (ISA, section 3.7.4). The PA additionally notes the significant uncertainty remaining with regard to the temporal relationships of ambient Pb levels and associated exposure with occurrence of a health effect (73 FR 67005, November 12, 2008).

In integrating consideration of the prior two questions with a focus on the standard, the PA then addresses the question regarding the extent to which newly available information supports or calls into question any of the basic elements of the current Pb standard. The PA addresses this question for each of the elements of the standard in light of the health effects evidence and other relevant information available in this review (and summarized in sections II.B and II.C above). As an initial matter, the PA recognizes the weight of the scientific evidence available in this review that continues to support our focus on effects on the nervous system of young children, specifically neurocognitive decrements, as the most sensitive endpoint. Consistent with the evidence available in the last review, the currently available evidence continues to indicate that a standard that provides requisite public health protection against the

occurrence of such effects in at-risk populations would also provide the requisite public health protection against the full array of health effects of Pb. Accordingly, the discussion of the elements below is framed by that background.

Indicator

The indicator for the current Pb standard is Pb-TSP. Key considerations in retaining this indicator in the last review are summarized in section II.A.1. Exposure to Pb in all sizes of particles passing through ambient air can contribute to Pb in blood and associated health effects by a wide array of exposure pathways (ISA, section 3.1). These pathways include the ingestion route, as well as inhalation (ISA, section 3.1), and a wide array of particle sizes play a role in these pathways (ISA, section 3.1.1.1). As at the time of the last review, the PA recognizes the variability of the Pb-TSP FRM in its capture of airborne Pb particles (as discussed in section 2.2.1.3.1 of the PA). As in the last review, the PA also notes that an alternative approach for collection of a conceptually comparable range of particle sizes, including ultra-coarse particles, is not yet available. Additionally, the limited available information regarding relationships between Pb-TSP and Pb in other size fractions indicates appreciable variation in this relationship, particularly near sources of Pb emissions where concentrations and potential exposures are greatest. Thus, the PA concludes that the information available in this review does not address previously identified limitations and uncertainties for the current indicator. Nor does the newly available information identify additional limitations or uncertainties.

The PA notes that the evidence available in this review continues to indicate the role of a range of air Pb particle sizes in contributing to Pb exposure (e.g., ISA, section 3.1.1.1) that contributes to Pb in blood and associated health effects. For example, the evidence indicates larger particle sizes for Pb that occurs in soil and house dust and may be ingested as compared to

Pb particles commonly occurring in the atmosphere and the size fraction of the latter that may be inhaled (ISA, section 3.1.1.1). Taken together, the PA concludes that the evidence currently available reinforces the appropriateness of an indicator for the Pb standard that reflects a wide range of airborne Pb particles.

Averaging time and form

The averaging time and form of the standard were revised in the last Pb NAAQS review, based on considerations summarized in section II.A.1 above. The current standard is a not-to-be-exceeded rolling 3-month average (40 CFR 50.16), derived from three monthly averages calculated in accordance with the current data handling procedures (40 CFR part 50, Appendix R). The form is a maximum, evaluated within a 3-year period (40 CFR 50.16). As at the time of the last review, the PA notes that evidence continues to support the importance of periods on the order of 3 months and the prominent role of deposition-related exposure pathways, with uncertainty associated with characterization of precise time periods associating ambient air Pb with air-related health effects. The PA concludes that relevant factors continue to be those pertaining to the human physiological response to changes in Pb exposures and those pertaining to the response of air-related Pb exposure pathways to changes in airborne Pb. The PA concludes that the newly available evidence in this review does not appreciably improve our understanding of the period of time in which air Pb concentrations would lead to the health effects most at issue in this review (PA, section 4.2.1). Newly available evidence accordingly also does not appreciably improve our understanding of the period of time for which control of air Pb concentrations would protect against exposures most pertinent to the health effects most at issue in this review. Thus, while there continue to be limitations in the evidence to inform our consideration of these elements of the standard and associated uncertainty, the available evidence

continues to provide support for the decisions made in the last review regarding these elements of the current Pb standard.

Level

The level of the current standard is 0.15 μg/m^3 (40 CFR 50.16). As described in section II.A.1 above, this level was selected in 2008 with consideration of, among other factors, an evidence-based air-related IQ loss framework, for which there are two primary inputs: air-to-blood ratios and C-R functions for blood Pb – IQ response in young children. Additionally taken into consideration were the uncertainties inherent in these inputs.[65] Application of the framework also entailed consideration of a magnitude of air-related IQ loss, which as further described in section II.A.1 above, is used in conjunction with this specific framework in light of the framework context, limitations and uncertainties. Additionally, selection of a level for the standard in 2008 was made in conjunction with decisions on indicator, averaging time and form.

As an initial matter, the PA considers the extent to which the evidence-based, air-related IQ loss framework which informed the Administrator's decision in the last review is supported by the currently available evidence and information. In so doing, the PA recognizes the support provided by the currently available evidence for the key conclusions drawn in the last review with regard to health effects of greatest concern, at-risk populations, the influence of Pb in ambient air on Pb in children's blood and the association between children's blood Pb and decrements in neurocognitive function (e.g., IQ). The PA additionally notes the complexity associated with interpreting the scientific evidence with regard to specific levels of Pb in ambient air, given the focus of the evidence on blood Pb as the key biomarker of children's aggregate exposure. The need to make such interpretations in the face of the associated complexity

[65] As discussed further below, the Administrator also considered the exposure/risk-based information, which he found to be roughly consistent and generally supportive of the framework estimates (73 FR 67004).

supported use of the evidence-based framework in the last review. In considering the currently available evidence for the same purposes in this review, the PA concludes that the evidence-based framework continues to provide a useful tool for consideration of the evidence with regard to the level of the standard.

The PA next turned to consideration of the primary inputs to the framework: air-to-blood ratios and C-R functions for blood Pb – IQ response in young children. With regard to the former, the PA concludes the limited newly available information assessed in the ISA, and discussed in section II.C above, to be generally consistent with the information in this area that was available at the time of the last review. The PA additionally recognizes the variability and uncertainty associated with quantitative air-to-blood ratios based on this information, as also existed in the last review. As in the last review, factors contributing to the variability and uncertainty of these estimates are varied and include aspects of the study populations (e.g., age and Pb exposure pathways) and the study circumstances (e.g., length of study period and variations in sources of Pb exposure during the study period). The PA notes that the full range of estimates associated with the available evidence is wide and considers it appropriate to give emphasis to estimates pertaining to circumstances closest to those in the U.S. today with regard to ambient air Pb and children's blood Pb concentrations, while recognizing the limitations associated with the available information. With that in mind, the PA considers the currently available evidence to continue to support the range of estimates for air-to-blood ratios concluded in the last review to be most appropriate for the current population of young children in the U.S., in light of the multiple air-related exposure pathways by which children are exposed and of the levels of air and blood Pb common today. Identification of this range also included consideration of the limitations associated with the available information and inherent uncertainties. This range

of air-to-blood ratios included 1:10 at the upper end and 1:5 at the lower end. The PA further recognizes that the limited evidence for air Pb and children's blood Pb concentrations closest to those in U.S. today continues to provide support for the Administrator's emphasis in the 2008 decision on the relatively central estimate of 1:7.

With regard to the second input to the evidence-based framework, C-R functions for the relationship of young children's blood Pb with neurocognitive impacts (e.g., IQ decrements), the PA considers several aspects of the evidence. First, as discussed in section II.B.3 above, the currently available information continues to provide evidence that this C-R relationship is nonlinear across the range of blood Pb levels from the higher concentrations more prevalent in the past to lower concentrations more common today. Thus, the PA continues to consider it particularly appropriate to focus on the evidence from studies with blood Pb levels closest to those of today's population which, as in the last review, includes studies with study group mean blood Pb levels ranging roughly from 3 to 4 μg/dL in children aged 24 months to 7 years (PA, Table 3-3). As discussed in section II.B.3 above, this is also consistent with the evidence currently available for this age group of young children, which does not include additional C-R slopes for incremental neurocognitive decrement with blood Pb levels at or below this range. In considering whether this set of functions continues to be well supported by the evidence, as assessed in the ISA (ISA, section 4.3.2), the PA notes the somewhat wide range in slopes encompassed by these study groups, while also noting the stability of the median. For example, omission of any of the four slopes considered in the last review does not appreciably change the median (e.g., the median would change from -1.75 IQ points per μg/dL blood Pb to -1.71 or -1.79). Thus, while differing judgments might be made with regard to inclusion of each of the four study groups, these estimates are generally supported by the current review of the evidence

in the ISA. Further, the stability of the median to modifications to this limited dataset lead the PA to conclude that the currently available evidence continues to support consideration of -1.75 IQ points per µg/dL blood Pb as a well-founded and stable estimate for purposes of describing the neurocognitive impact quantitatively on this age group of U.S. children.

In summary, in considering the evidence and information available in this review pertaining to the level of the current Pb standard, the PA notes that the evidence available in this review, as summarized in the ISA, continues to support the air-related IQ loss evidence-based framework, with the inputs that were used in the last review. These include estimates of air-to-blood ratios ranging from 1:5 to 1:10, with a generally central estimate of 1:7. Additionally, the C-R functions most relevant to blood Pb levels in U.S. children today continue to be provided by the set of four analyses considered in the last review for which the median estimate is -1.75 IQ points per µg/dL Pb in young children's blood. Thus, the PA observed that the evidence available in this review has changed little if at all with regard to the aspects given weight in the conclusion on level for the new standard in the last review and would not appear to call into question any of the basic elements of the standard. In so doing, the PA additionally recognizes that the overall decision on adequacy of the current standard is a public health policy judgment by the Administrator.

2. Exposure/Risk-based Considerations in the Policy Assessment

In consideration of the issue of adequacy of public health protection provided by the current standard, the PA also considered the quantitative exposure/risk assessment completed in the last review, augmented as described in section II.C above. The PA recognizes substantial uncertainty inherent in the REA estimates of air-related risk associated with localized conditions just meeting the current standard, which we have characterized as approximate and falling within

rough bounds.[66] This approximate estimate of risk for children living in such areas is generally overlapping with and consistent with the evidence-based air-related IQ loss estimates described in section II.A.1 above. The PA discussion with regard to interpretation of the exposure/risk information for air quality conditions associated with just meeting the current standard is organized around two questions, as summarized here (PA, section 4.2.2).

In considering the level of confidence associated with estimates of air-related risk generated for simulations just meeting the current Pb standard, the PA recognizes, as an initial matter, the significant limitations and complexity associated with the risk and exposure assessments for Pb that are far beyond those associated with similar assessments typically performed for other criteria pollutants. In completing the assessment, we were constrained by significant limitations with regard to data and tools particular to the problem at hand. Further, the multimedia and persistent nature of Pb and the role of multiple exposure pathways contribute significant additional complexity to the assessment as compared to other assessments that focus only on the inhalation pathway. As a result, the estimates of air-related exposure and risk are approximate, presented as upper and lower bounds within which we consider air-related risk likely to fall. The description of overall confidence in this characterization of air-related risk is based on consideration of the overall design of the analysis (summarized in section II.D), the degree to which key sources of variability are reflected in the design of the analysis (summarized in section II.D.3), and our characterization of key sources of uncertainty (summarized in section II.D.3).

With regard to key sources of uncertainty, the PA notes particularly those affecting the

[66] We note that the value of the upper bound is influenced by risk associated with exposure pathways that were not varied with alternative standard levels, a modeling limitation with the potential to contribute to overestimation of the upper bound with air quality scenarios involving air Pb levels below current conditions for the study area (see sections 3.4.4 and 3.4.7 above).

precision of the air-related risk estimates. Associated sources of uncertainty include the inability to simulate changes in air-related Pb as a function of changes in ambient air Pb in exposure pathways other than those involving inhalation of ambient air and ingestion of indoor dust. This contributes to the positive bias of the upper bound for the air-related risk estimates. The PA additionally recognizes the significant uncertainty associated with estimating upper percentiles of the distribution of *air-related* blood Pb concentration estimates (and associated IQ loss estimates) due to limitations in available information. Lastly, the PA recognizes the uncertainty associated with application of the C-R function at the lower blood Pb levels in the distribution; this relates to the limited representation of blood Pb levels of this magnitude in the dataset from which the C-R function is derived (PA, section 4.2.2).

In the quantitative risk information available in this review, we have air-related risk estimates for simulations just meeting the current standard from one of the location-specific urban case studies (Chicago) and from the generalized (local) urban case study. With regard to the latter, the PA notes its simplified design that does not include multiple exposure zones; thus reducing the dimensions simulated. The PA concludes a reasonable degree of confidence in aspects of the generalized (local) urban case study for the specific situation we consider it to represent (i.e., a temporal pattern of air Pb concentrations that just meets the level of the standard), and when the associated estimates are characterized as approximate, within upper and lower bounds (as described above), while also recognizing considerable associated uncertainty.

In considering the extent to which the estimated air-related risks remaining upon just meeting the current Pb standard are important from a public health perspective, the PA considers the nature and magnitude of such estimated risks (and attendant uncertainties), including such impacts on the affected population, and additionally considers the size of the affected population.

In considering the quantitative risk estimates for decrements in IQ, we recognize that although some neurocognitive effects may be transient, some effects may persist into adulthood, affecting success later in life (ISA, sections 1.9.5 and 4.3.14). The PA additionally recognizes the potential population impacts of small changes in population mean values of metrics such as IQ, presuming a uniform manifestation of Pb-related decrement across the range of population IQ (ISA, section 1.9.1; PA, section 3.3).

As summarized in sections II.D above, limitations in modeling tools and data affected our ability to develop precise risk estimates for air-related Pb exposure pathways and contributed uncertainties to the risk estimates. The results are approximate estimates which we describe through the use of rough upper and lower bounds within which we estimate air-related risk to fall. We have recognized a number of uncertainties in the underlying risk estimates from the 2007 REA and in the interpolation approach employed in the new analyses for this review. We have characterized the magnitude of air-related risk associated with the current standard with a focus on median estimates, for which we have appreciably greater confidence than estimates for outer ends of risk distribution (see section 3.4.7 of the PA) and on risks derived using the C-R function in which we have greatest confidence (see sections 3.4.3.3.1 and 3.4.7 of the PA). These risk estimates include estimates from the last review for one of the location-specific urban study area populations as well as estimates newly derived in this review based on interpolation from 2007 REA results for the generalized (local) urban case study, which is recognized to reflect a generalized high end of air-related exposure for localized populations. Taken together, these results for just meeting the current standard include a high-end localized risk estimate for air-related Pb of a magnitude falling within general rough bounds of 1 and 3 points IQ loss, with attendant uncertainties, and with appreciably lower risks with increasing distance from the

highest exposure locations.

In considering the importance of such risk from a public health perspective, the PA also considers the size of at-risk populations represented by the REA case studies. As summarized in section II.D.1 above (and described more fully in the PA, section 3.4), the generalized (local) urban case study is considered to represent a localized urban population exposed near the level of the standard, such as a very small, compact neighborhood near a source contributing to air Pb concentrations just meeting the standard. This case study provides representation in the risk assessment for such small populations at the upper end of the gradient in ambient air concentrations expected to occur near sources; thus estimates for this case study reflect exposures nearest the standard being evaluated. While we do not have precise estimates of the number of young children living in such areas of the U.S. today, we have information that informs our understanding of their magnitude. For example, as summarized in section II.B.5 above, the PA estimates some 2,700 children, aged 5 years and younger, to be living in localized areas with elevated air Pb concentrations that are above or near the current standard. Based on the 2010 census estimates of approximately 24.3 million children in the U.S. aged 5 years or younger, this indicates the size of the population of young children of this age living in areas in close proximity to areas where air Pb concentrations may be above or near the current standard to be generally on the order of a hundredth of a percent of the full population of correspondingly aged children.[67, 68] While these estimates pertain to the age group of children aged 5 years and

[67] The areas included in this estimate where the standard is currently exceeded are treated, for present purposes, as areas with air Pb concentrations just meeting the current standard and are included for purposes of this analysis (PA, pp. 3-36 to 3-38). This is in light of the requirement for areas not in attainment with the standard to attain the standard as expeditiously as practicable, but no later than 5 years after designation.

younger, the PA additionally notes that a focus on an alternative age range (e.g., through age 7), while increasing the number for children living in such locations, would not be expected to appreciably change the percentage of the full U.S. age group that the subset represents.

3. CASAC Advice

In the current review of the primary standard for Pb, the CASAC has provided advice and recommendations in their review of drafts of the ISA, of the REA Planning Document, and of the draft PA. We have additionally received comments from the public on drafts of these documents.[69]

In their comments on the draft PA, the CASAC concurred with staff's overall preliminary conclusions that it is appropriate to consider retaining the current primary standard without revision, stating that "the current scientific literature does not support a revision to the Primary Lead (Pb) National Ambient Air Quality Standard (NAAQS)" (Frey, 2013b). They further noted that "[a]lthough the current review incorporates a substantial body of new scientific literature, the new literature does not justify a revision to the standards because it does not significantly reduce substantial data gaps and uncertainties (e.g., air-blood Pb relationship at low levels; sources contributing to current population blood Pb levels, especially in children; the relationship between Pb and childhood neurocognitive function at current population exposure levels; the relationship between ambient air Pb and outdoor dust and surface soil Pb concentrations)." In recognition of these limitations in the available information, the CASAC provided

[68] A second PA analysis, performed in recognition of the potential for the first analysis to under-represent sites with elevated Pb concentrations, but with its own attendant uncertainties, indicates the potential for the population group in such areas to be only slightly larger, in terms of hundredths of a percent of the full population of children in this age group (PA, pp. 3-36 to 3-38, 4-25, 4-32).

[69] As noted in section II.E.3 above, written comments submitted to the agency, as well as transcripts and minutes of the public meetings held in conjunction with CASAC's reviews of documents for the review will be available in the docket for this rulemaking.

recommendations on research to address these data gaps and uncertainties so as to inform future Pb NAAQS reviews (Frey, 2013b).

The CASAC comments indicated agreements with key aspects of staff's consideration of the exposure/risk information and currently available evidence in this review (Frey, 2013b, Consensus Response to Charge Questions, p. 7).

> *The use of exposure/risk information from the previous Pb NAAQS review appears appropriate given the absence of significant new information that could fundamentally change the interpretation of the exposure/risk information. This interpretation is reasonable given that information supporting the current standard is largely unchanged since the current standard was issued.*
>
> *The CASAC agrees that the adverse impact of low levels of Pb exposure on neurocognitive function and development in children remains the most sensitive health endpoint, and that a primary Pb NAAQS designed to protect against that effect will offer satisfactory protection against the many other health impacts associated with Pb exposure.*
>
> *The CASAC concurs with the draft PA that the scientific findings pertaining to air-to-blood Pb ratios and the C-R relationships between blood Pb and childhood IQ decrements that formed the basis of the current Pb NAAQS remain valid and are consistent with current data.*

The CASAC concurred with the appropriateness of the application of the evidence-based framework from the last Pb NAAQS review. With regard to the key inputs to that framework, CASAC concluded that "[t]he new literature published since the previous review provides further support for the health effect conclusions presented in that review" and that the studies newly available in this review "do not fundamentally alter the uncertainties for air-to-blood ratios or C-R functions for IQ decrements in young children" (Frey, 2013b, Consensus Response to Charge Questions, p. 6).

The comments from CASAC also took note of the uncertainties that remain in this review, which contribute to the uncertainties associated with drawing conclusions regarding air-related exposures and associated health risk at or below the level of the current standard, stating their agreement with "the EPA conclusion that 'there is appreciable uncertainty associated with

drawing conclusions regarding whether there would be reductions in blood Pb levels from alternative lower levels as compared to the level of the current standard'" (Frey, 2013b, Consensus Response to Charge Questions, p. 6).

Of the limited public comments received on this review to date that have addressed adequacy of the current primary Pb standard, all but one state support for retaining the current standard without revision, citing uncertainties in the available evidence and risk information. The other commenter expressed the view that the standard should be revised to be more restrictive given the evidence of Pb effects in populations with mean blood Pb levels below 10 μg/dL.

4. Administrator's Proposed Conclusions on the Adequacy of the Current Primary Standard

Based on the large body of evidence concerning the health effects and potential public health impacts of exposure to Pb emitted into ambient air, and taking into consideration the attendant uncertainties and limitations of the evidence, the Administrator proposes to conclude that the current primary standard provides the requisite protection of public health, with an adequate margin of safety and should be retained.

In considering the adequacy of the current standard, the Administrator has carefully considered the assessment of the available evidence and conclusions contained in the ISA; the technical information, including exposure/risk information, staff conclusions, and associated rationale, presented in the PA; the advice and recommendations from CASAC; and public comments to date in this review. In the discussion below, the Administrator gives weight to the PA conclusions, with which CASAC has concurred, and takes note of key aspects of the rationale presented for those conclusions which contribute to her proposed decision.

As an initial matter, the Administrator takes note of the PA discussion with regard to the complexity involved in considering the adequacy of protection in the case of the primary Pb standard, which differs substantially from that involved in consideration of the primary NAAQS

for other pollutants, for which the limited focus on the inhalation pathway is a relatively simpler context. Additionally, while an important component of the evidence base for most other NAAQS pollutants is the availability of studies that have investigated an association between current concentrations of the pollutant in ambient air and the occurrence of health effects plausibly related to ambient air exposure to that pollutant, the evidence base that supports conclusions in this review of the Pb NAAQS includes most prominently epidemiological studies focused on associations of blood Pb levels in U.S. populations with health effects plausibly related to Pb exposures. Support for conclusions regarding the plausibility for ambient air Pb to play a role in such findings derives, in part, from studies linking Pb in ambient air with the occurrence of health effects. However, such studies (dating from the past or from other countries) involve ambient air Pb concentrations many times greater than those that would meet the current standard. Thus, in considering the adequacy of the current Pb standard, rather than considering studies that have directly investigated current concentrations of Pb in ambient air (including in locations where the current standard is met) and the occurrence of health effects, we primarily consider the evidence for, and risk estimated from, models, based upon key relationships, such as those among ambient air Pb, Pb exposure, blood Pb and health effects. This evidence, with its associated limitations and uncertainties, contributes to the EPA's conclusions regarding a relationship between ambient air Pb conditions under the current standard and health effects.

With regard to the current evidence, the Administrator first takes note of the well-established body of evidence on the health effects of Pb, augmented in some aspects since the last review, which continues to support identification of neurocognitive effects in young children as the most sensitive endpoint associated with Pb exposure. The evidence, as summarized in the

PA and discussed in detail in the ISA, continues to indicate that a standard that provides protection from neurocognitive effects in young children additionally provides protection for other health effects of Pb, such as those reported in adult populations. The Administrator takes note of the PA finding that application of the evidence-based, air-related IQ loss framework, developed in the last review, continues to provide a useful approach for considering and integrating the evidence on relationships between Pb in ambient air and Pb in children's blood and risks of neurocognitive effects (for which IQ loss is used as an indicator). She additionally takes note of the PA finding (described in section II.E.1 above) that the currently available evidence base, while somewhat expanded since the last review, is not appreciably expanded or supportive of appreciably different conclusions with regard to air-to-blood ratios or C-R functions for neurocognitive decrements in young children. She concurs with the PA findings, summarized in section II.E.1 above, that application of this framework, in light of the current evidence and exposure/risk information, continues to support a standard as protective as the current standard.

In considering the nature and magnitude of the array of uncertainties that are inherent in the scientific evidence and analyses, the Administrator recognizes that our understanding of the relationships between the presence of a pollutant in ambient air and associated health effects is based on a broad body of information encompassing not only more established aspects of the evidence, but also aspects in which there may be substantial uncertainty. In the case of the Pb NAAQS review, she takes note of the recognition in the PA of increased uncertainty in characterizing the relationship of effects on IQ with blood Pb levels below those represented in the evidence base and in projecting the magnitude of blood Pb response to ambient air Pb concentrations at and below the level of the current standard. The PA recognizes this increased

uncertainty, particularly in light of the multiple factors that play a role in such a projection (e.g., meteorology, atmospheric dispersion and deposition, human physiology and behavior), each of which carry attendant uncertainties. The Administrator recognizes that collectively, these aspects of the evidence and associated uncertainties contribute to a recognition that for Pb, as for other pollutants, the available health effects evidence generally reflects a continuum, consisting of 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.

In making a judgment on the point at which health effects associated with Pb become important from a public health perspective, the Administrator has considered the public health significance of a decrement of a very small number of IQ points in the at-risk population of young children, in light of associated uncertainties. She notes that her judgment on this matter relates to her consideration of the IQ loss estimates yielded by the air-related IQ loss evidence-based framework for specific combinations of standard level, air-to-blood ratio and C-R function. In considering the public health significance of IQ loss estimates in young children, the Administrator gives weight to the comments of CASAC and some public commenters in the last review which recognized a population mean IQ loss of 1 to 2 points to be of public health significance and recommended that a very high percentage of the population be protected from such a magnitude of IQ loss (73 FR 67000, November 12, 2008). In so doing, the Administrator additionally notes that the EPA is aware of no new information or new commonly accepted guidelines or criteria within the public health community for interpreting public health significance of neurocognitive effects in the context of a decision on adequacy of the current Pb standard (PA, pp. 4-33 to 4-34).

With the objective identified by CASAC in the 2008 review in mind, the Administrator

considers the role of the air-related IQ loss evidence-based framework in informing consideration of standards that might be concluded to provide such a level of protection. In so doing, she first recognizes, like the Administrator at the time of the last review, that the IQ loss estimates produced with the evidence-based framework do not correspond to a specific quantitative public health policy goal for air-related IQ loss that would be acceptable or unacceptable for the entire population of children in the U.S. Rather, the conceptual context for the evidence-based framework is that it provides estimates for the mean air-related IQ loss of a subset of the population of U.S. children (i.e., the subset living in close proximity to air Pb sources that contributed to elevated air Pb concentrations that equal the current level of the standard). This is the subset expected to experience air-related Pb exposures at the high end of the national distribution of such exposures. The associated mean IQ loss estimate is the average for this highly exposed subset and is not the average air-related IQ loss projected for the entire U.S. population of children. Further, the Administrator recognizes uncertainties associated with those estimates, and notes the PA conclusion that the uncertainties increase with estimates associated with successively lower standard levels. The Administrator additionally takes note of the PA estimates for the size of such a population, drawn from information on numbers of young children (aged 5 years or younger) living near monitors registering ambient Pb concentrations above or within 10 percent of the NAAQS, which indicate it to be on the order of one hundredth of one percent of the U.S. population of children of this age, with an upper bound of approximately four hundredths of one percent, drawn from similar demographic information based on proximity to large Pb sources, as identified using the NEI (PA, pp. 3-36 to 3-38). In summary, the current evidence, as considered within the conceptual and quantitative context of the evidence-based framework, and current air monitoring information indicates that the current

standard would be expected to satisfy the public health policy goal recommended by CASAC in the last Pb NAAQS review, and CASAC did not provide a different goal in the present review. Thus, the evidence indicates that the current standard provides protection for young children from neurocognitive impacts, including IQ loss, consistent with advice from CASAC regarding IQ loss of public health significance.

In drawing conclusions from application of the evidence-based framework with regard to adequacy of the current standard, the Administrator further recognizes the degree to which IQ loss estimates drawn from the air-related IQ loss evidence-based framework reflect mean blood Pb levels that are below those represented in the currently available evidence for young children. For example, in the case of the current standard level of 0.15 $\mu g/m^3$, multiplication by the air-to-blood ratio of 1:7, the value that was the focus of the last review and which the evidence continues to support in this review, yields a mean air-related blood Pb level of 1.05 μg/dL. This blood Pb level is half the level of the lowest blood Pb subgroup of pre-school children in which neurocognitive effects have been observed (PA, Table 3-2; Miranda et al., 2009) and well below the means of subgroups for which continuous C-R functions have been estimated (Table 1 above). The Administrator views such an extension below the lowest studied levels to be reasonable given the lack of identified blood Pb level threshold in the current evidence base for neurocognitive effects and the need for the NAAQS to provide a margin of safety. She takes note, however, of the PA finding that the framework IQ loss estimates for standard levels lower than the current standard level represent still greater extrapolations from the current evidence base with corresponding increased uncertainty (PA, section 3.2, pp. 4-32 to 4-33).

In considering application of the evidence-based framework in this review with regard to the extent there is support within the evidence for a standard with greater protection, the

Administrator additionally takes note of the uncertainties that remain in our understanding of important aspects of ambient air Pb exposure and associated health effects, as discussed in the PA (PA, Chapter 3) and summarized in sections II.B and II.C above. With regard to the air-to-blood ratios that reflect the relationship between concentrations of Pb in ambient air and air-related Pb in children's blood, she particularly notes the limitations and uncertainties identified in the ISA and PA with regard to the available studies and the gaps and uncertainties in the evidence base. These include gaps and uncertainties with regard to studies that have investigated such quantitative relationships under conditions pertaining to the current standard (e.g., in localized areas near air Pb sources where the standard is just met in the U.S. today), as well as with regard to evidence to inform our understanding of the quantitative aspects of relationships between ambient air Pb and outdoor soil/dust Pb and indoor dust Pb. These critical exposure pathways are also represented in the evidence-based air-related IQ loss framework within the estimates of air-to-blood ratios. In light of these uncertainties and limitations in the evidence base, the Administrator gives weight to the PA conclusion of greater uncertainty with regard to relationships between concentrations of Pb in ambient air and air-related Pb in children's blood, and with regard to estimates of the slope of the C-R function of neurocognitive impacts (IQ loss) for application of the framework to levels below the current standard, given the weaker linkage with existing evidence as discussed in the PA (PA, sections 3.1, 3.2 and 4.2.1).

With respect to exposure/risk-based considerations, as in the last review, the Administrator notes the complexity of the REA modeling analyses and the associated limitations and uncertainties. Based on consideration of the risk-related information for conditions just meeting the current standard, the Administrator takes note of the attendant uncertainties, discussed in detail in the PA (PA, sections 3.4 and 4.2.2), while finding that the quantitative risk

estimates, with a focus on those for the generalized (local) urban case study, are "roughly consistent with and generally supportive" of estimates from the evidence-based air-related IQ loss framework. She further takes note of the PA finding of increasing uncertainty for air quality scenarios involving air Pb concentrations increasingly below the current conditions for each case study, due in part to modeling limitations that derive from uncertainty regarding relationships between ambient air Pb and outdoor soil/dust Pb and indoor dust Pb (PA, sections 3.4.3.1 and 3.4.7).

Based on the above considerations and with consideration of advice from CASAC, the Administrator reaches the conclusion that the current body of evidence, in combination with the exposure/risk information, supports a primary standard as protective as the current standard. Based on consideration of the evidence and exposure/risk information available in this review with its attendant uncertainties and limitations and information that might inform public health policy judgments, as well as advice from CASAC, including their concurrence with the PA conclusions that revision of the primary Pb standard is not warranted at this time, the Administrator further concludes that it is appropriate to consider retaining the current standard without revision.

The Administrator bases these proposed conclusions on consideration of the health effects evidence, including consideration of this evidence in the context of the evidence-based, air-related IQ loss framework, and with support from the exposure/risk information, recognizing the uncertainties attendant with both. In so doing, she takes note of the PA description of the complexities and limitations in the evidence base associated with reaching conclusions regarding the magnitude of risk associated with the current standard, as well as the increasing uncertainty of risk estimates for lower air Pb concentrations. Inherent in the Administrator's conclusions are

public health policy judgments on the public health implications of the blood Pb levels and risk estimated for air-related Pb under the current standard, including the public health significance of the Pb effects being considered, as well as aspects of the use of the evidence-based framework that may be considered to contribute to the margin of safety. These public health policy judgments include judgments related to the appropriate degree of public health protection that should be afforded to protect against risk of neurocognitive effects in at-risk populations, such as IQ loss in young children, as well as with regard to the appropriate weight to be given to differing aspects of the evidence and exposure/risk information, and how to consider their associated uncertainties. Based on these considerations and the judgments identified here, the Administrator concludes that the current standard provides the requisite protection of public health with an adequate margin of safety, including protection of at-risk populations, such as young children living near Pb emissions sources where ambient concentrations just meet the standard.

In reaching this conclusion with regard to the adequacy of public health protection afforded by the existing primary standard, the Administrator recognizes that in establishing primary standards under the Act that are requisite to protect public health with an adequate margin of safety, she is seeking 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, even if the risk is not precisely identified as to nature or degree. The CAA requirement that primary standards provide an adequate margin of safety was intended to address uncertainties associated with inconclusive scientific and technical information available at the time of standard setting, as described in section I.A above. This requirement was also intended to provide a reasonable

degree of protection from hazards that research has not yet identified.

In this context, the Administrator's proposed conclusion that the current standard provides the requisite protection and that a more restrictive standard would not be requisite additionally recognizes that the uncertainties and limitations associated with the many aspects of the estimated relationships between air Pb concentrations and blood Pb levels and associated health effects are amplified with consideration of increasingly lower air concentrations. In so doing, she takes note of the PA conclusion, with which CASAC has agreed, that based on the current evidence, there is appreciable uncertainty associated with drawing conclusions regarding whether there would be reductions in blood Pb levels and risk to public health from alternative lower levels of the standard as compared to the level of the current standard (PA, pp. 4-35 to 4-36; Frey, 2013b, p. 6). The Administrator judges this uncertainty to be too great for the current evidence and exposure/risk information to provide a basis for revising the current standard. Thus, based on the public health policy judgments described above, including the weight given to uncertainties in the evidence, the Administrator proposes to conclude that the current standard should be retained, without revision. The Administrator solicits comment on this conclusion.

III. Rationale for Proposed Decision on the Secondary Standard

This section presents information relevant to the rationale for the Administrator's proposed decision to retain the existing secondary Pb standard, which as discussed more fully below, is based on a thorough review in the ISA of the latest scientific information, generally published through September 2011,[70] on ecological or welfare effects associated with Pb and pertaining to the presence of Pb in the ambient air. This proposal also takes into account: (1) the

[70] In addition to the review's opening "call for information" (75 FR 8934), "literature searches were conducted routinely to identify studies published since the last review, focusing on studies published from 2006 (close of the previous scientific assessment) through September 2011" and references "that were considered for inclusion or actually cited in this ISA can be found at http://hero.epa.gov/lead" (ISA, p. 1-2).

PA's staff assessments of the most policy-relevant information in the ISA and staff analyses of potential ecological exposures and risk, upon which staff conclusions regarding appropriate considerations in this review are based; (2) CASAC advice and recommendations, as reflected in discussions of drafts of the ISA and PA at public meetings, in separate written comments, and in CASAC's letters to the Administrator; and (3) public comments received during the development of these documents, either in connection with CASAC meetings or separately.

Section III.A provides background on the general approach for review of the secondary NAAQS for Pb, including a summary of the approach used in the last review (section III.A.1) and the general approach for the current review (section III.A.2). Section III.B summarizes the body of evidence on ecological or welfare effects associated with Pb exposures, focusing on consideration of key policy-relevant questions, and section III.C summarizes the exposure/risk information in this review. Section III.D presents the Administrator's proposed conclusions on adequacy of the current standard, drawing on both evidence-based and exposure/risk-based considerations (sections III.D.1), and advice from CASAC (section III.D.2).

A. General Approach

The past and current approaches described below are all based most fundamentally on using the EPA's assessment of the current scientific evidence and previous quantitative analyses to inform the Administrator's judgment with regard to the secondary standard for Pb. In drawing conclusions for the Administrator's consideration with regard to the secondary standard, we note that the final decision on the adequacy of the current secondary Pb standard is largely a public welfare policy judgment to be made by the Administrator. The Administrator's final decision must 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. This approach is consistent with the requirements of the NAAQS provisions of the Act. These provisions require the Administrator to establish a secondary standard that, in the judgment of the Administrator, 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." In so doing, the Administrator seeks to establish standards that are neither more nor less stringent than necessary for this purpose.

1. Approach in the Last Review

In the last review, completed in 2008, the current secondary standard for Pb was set equal to the primary standard (73 FR 66964, November 12, 2008). As summarized in sections I.C and II.A.1 above, the primary standard was substantially revised in the last review. The 2008 decision considered the body of evidence as assessed in the 2006 CD (USEPA, 2006a) as well as the 2007 Staff Paper assessment of the policy-relevant information contained in the 2006 CD and the screening-level ecological risk assessment (2006 REA; USEPA, 2007b), the advice and recommendations of CASAC (Henderson 2007a, 2007b, 2008a, 2008b), and public comment.

In the previous review, the Staff Paper concluded, based on laboratory studies and current media concentrations in a wide range of locations, that it seemed likely that adverse effects were occurring from ambient air-related Pb, particularly near point sources, under the then-current standard (73 FR 67010, November 12, 2008). Given the limited data on Pb effects in ecosystems, and associated uncertainties, such as those with regard to factors such as the presence of multiple metals and historic environmental burdens, it was at the time, as it is now, necessary to look at evidence of Pb effects on organisms and extrapolate to ecosystem effects. Taking into account the available evidence and current media concentrations in a wide range of locations, the Administrator concluded that there was potential for adverse effects occurring under the then-current standard; however there were insufficient data to provide a quantitative basis for setting a

secondary standard different from the primary (73 FR 67011, November 12, 2008). Therefore, citing a general lack of data that would indicate the appropriate level of Pb in environmental media that may be associated with adverse effects, 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) was likely to have significant beneficial effects on the magnitude of Pb exposures in the environment, the secondary standard was revised to be consistent with the revised primary standard (73 FR 67011, November 12, 2008).

2. Approach for the Current Review

Our approach for reviewing the current secondary standard takes into consideration the approaches used in the last Pb NAAQS review and involves addressing key policy-relevant questions in light of currently available scientific and technical information. In evaluating whether it is appropriate to consider retaining the current secondary Pb standard, or whether consideration of revision is appropriate, we have adopted an approach in this review that builds on the general approach from the last review and reflects the body of evidence and information now available. As summarized above, the Administrator's decisions in the previous review were based on the conclusion that there was the potential for adverse ecological effects under the previous standard.

In our approach here, we focus on consideration of the extent to which a broader body of scientific evidence is now available that would inform decisions on either the potential for adverse effects to ecosystems under the current standard or the ability to set a more ecologically relevant secondary standard than was feasible in the previous review. In considering the scientific and technical information in sections II.B and II.C below, as in the PA, we draw on the ecological effects evidence presented in detail in the ISA and aspects summarized in the PA, along with the information associated with the screening-level risk assessment also in the PA. In

section III.D below, we have taken into account both evidence-based and risk-based considerations framed by a series of policy-relevant questions presented in the PA. These questions generally discuss the extent to which we are able to better characterize effects and the likelihood of adverse effects in the environment under the current standard. Our approach to considering these issues recognizes that the available welfare effects evidence generally reflects laboratory-based evidence of toxicological effects on specific organisms exposed to concentrations of Pb. It is widely recognized, however, that environmental exposures from atmospherically derived Pb are likely to be lower than those commonly assessed in laboratory studies and that studies of exposures similar to those in the environment are often accompanied by significant confounding and modifying factors (e.g., other metals, acidification), increasing our uncertainty about the likelihood and magnitude of organism and ecosystem responses.

B. 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 wellbeing. This discussion presents key aspects of the current evidence of Pb-related welfare effects that are assessed in the ISA and the 2006 CD, drawing from the summary of policy-relevant aspects in the PA (PA, section 5.1).

Lead has been demonstrated to have harmful effects on reproduction and development, growth, and survival in many species as described in the assessment of the evidence available in this review and consistent with the conclusions drawn in the last review (ISA, section 1.7; 2006 CD, sections 7.1.5 and 7.2.5). A number of studies on ecological effects of Pb are newly

available in this review and are critically assessed in the ISA as part of the full body of evidence. The full body of currently available evidence reaffirms conclusions on the array of effects recognized for Pb in the last review (ISA, section 1.7). In so doing, in the context of pollutant exposures considered relevant the ISA determines[71] that causal[72] or likely causal[73] relationships exist in both freshwater and terrestrial ecosystems for Pb with effects on reproduction and development in vertebrates and invertebrates; growth in plants and invertebrates; and survival in vertebrates and invertebrates (ISA, Table 1-3). In drawing judgments regarding causality for the criteria air pollutants, the ISA places emphasis on "evidence of effects at doses (e.g., blood Pb concentration) or exposures (e.g., air concentrations) that are relevant to, or somewhat above, those currently experienced by the population." The ISA notes that the "extent to which studies of higher concentrations are considered varies … but generally includes those with doses or exposures in the range of one to two orders of magnitude above current or ambient conditions." Studies "that use higher doses or exposures may also be considered … [t]hus, a causality determination is based on weight of evidence evaluation for health, ecological or welfare effects, focusing on the evidence from exposures or doses generally ranging from current levels to one or two orders of magnitude above current levels" (ISA, pp. lx to lxi).

Although considerable uncertainties are recognized in generalizing effects observed

[71] Since the last Pb NAAQS review, the ISAs, which have replaced CDs in documenting each review of the scientific evidence (or air quality criteria), employ a systematic framework for weighing the evidence and describing associated conclusions with regard to causality, using established descriptors: "causal" relationship with relevant exposure, "likely" to be a causal relationship, evidence is "suggestive" of a causal relationship, "inadequate" evidence to infer a causal relationship, and "not likely" to be a causal relationship (ISA, Preamble).

[72] In determining that a causal relationship exists for Pb with specific ecological or welfare effects, the EPA has concluded that "[e]vidence is sufficient to conclude that there is a causal relationship with relevant pollutant exposures (i.e., doses or exposures generally within one to two orders of magnitude of current levels)" (ISA, p. lxii).

[73] In determining a likely causal relationship exists for Pb with specific ecological or welfare effects, the EPA has concluded that "[e]vidence is sufficient to conclude that there is a likely causal association with relevant pollutant exposures ... but uncertainties remain" (ISA, p. lxii).

under particular, small-scale conditions, up to the ecosystem level of biological organization, the ISA determines that the cumulative evidence reported for Pb effects at such higher levels of biological organization and for endpoints in single species with direct relevance to population and ecosystem level effects (i.e., development and reproduction, growth, survival) is sufficient to conclude that a causal relationship is likely to exist between Pb exposures and community and ecosystem-level effects in freshwater and terrestrial systems (ISA, section 1.7.3.7).

The ISA also presents evidence for saltwater ecosystems, concluding that current evidence is inadequate to make causality determinations for most population-level responses, as well as community and ecosystem effects, while finding the evidence to be suggestive linking Pb and effects on reproduction and development in marine invertebrates (ISA, Table 1-3, sections 6.3.12 and 6.4.21).

As in prior reviews of the Pb NAAQS, this review is focused on those effects most pertinent to ambient air Pb exposures. Given the reductions in ambient air Pb concentrations over the past decades, these effects are generally those associated with the lowest levels of Pb exposure that have been evaluated. Additionally, we recognize the limitations on our ability to draw conclusions about environmental exposures from ecological studies of organism-level effects, as most studies were conducted in laboratory settings which may not accurately represent field conditions or the multiple variables that govern exposure.

The relationship between ambient air Pb and ecosystem response is important in making the connection between current emissions of Pb and the potential for adverse ecological effects. The limitations in the data available on this subject for the last review were significant. There is no new evidence since the last review that substantially improves our understanding of the relationship between ambient air Pb and measurable ecological effects. As stated in the last

review, the role of ambient air Pb in contributing to ecosystem Pb has been declining over the past several decades. It remains difficult to apportion exposure between air and other sources to inform our understanding of the potential for ecosystem effects that might be associated with air emissions. As noted in the ISA, *"[t]he amount of Pb in ecosystems is a result of a number of inputs and it is not currently possible to determine the contribution of atmospherically-derived Pb from total Pb in terrestrial, freshwater or saltwater systems"* (ISA, section 6.5). Further, considerable uncertainties also remain in drawing conclusions from effects evidence observed under laboratory conditions with regard to effects expected at the ecosystem level in the environment. In many cases it is difficult to characterize the nature and magnitude of effects and to quantify relationships between ambient concentrations of Pb and ecosystem response due to the existence of multiple stressors, variability in field conditions, and differences in Pb bioavailability at that level of organization (ISA, section 6.5). In summary, the ISA concludes that "[r]ecent information available since the 2006 Pb AQCD, includes additional field studies in both terrestrial and aquatic ecosystems, but the connection between air concentration and ecosystem exposure continues to be poorly characterized for Pb and the contribution of atmospheric Pb to specific sites is not clear" (ISA, section 6.5).

It is also important to consider the fate and transport of both current Pb and Pb emitted in the past. It is this past legacy of Pb that was cited as a significant source of uncertainty in the last review. The extensive history of Pb uses in developed countries coupled with atmospheric transport processes has left a legacy of Pb in ecosystems globally (e.g., 2006 CD, sections 2.3.1 and 7.1; 1977 CD, section 6.3.1). Records of U.S. atmospheric emissions of Pb in the twentieth and late nineteenth centuries have been documented in sediment cores (PA, section 2.3; ISA, section 2.6.2; Landers et al., 2010). Once deposited, Pb can be transported by stormwater runoff

or resuspension to catchments and nearby water bodies or stored in soil layers in forested areas, its further movement influenced by soil or sediment composition and chemistry and physical processes. Some new studies are available that provide additional information, briefly summarized below, on Pb cycling, flux and retention within terrestrial and aquatic systems. This new information does not fundamentally change our understanding from the last review of Pb movement through or accumulation in ecosystems over time but rather improves our understanding of some of the underlying processes and mechanisms in soil, water and sediment. There is little new information, however, on fate and transport in ecosystems specifically related to air-derived Pb (ISA, section 2.3). There is limited newly available information with regard to the timing of ecosystem recovery from historic atmospheric deposition of Pb (ISA, sections 2.3.2.4 and 2.3.3.3).

Overall, recent studies in terrestrial ecosystems provide deposition data consistent with deposition fluxes reported in the 2006 CD and demonstrate consistently that atmospheric deposition of Pb has decreased since the phase-out of leaded on-road gasoline (PA, section 2.3.2.2; ISA, section 2.3.3). Follow-up studies in several locations at high elevation sites indicate little change in soil Pb concentrations since the phase-out of leaded onroad gasoline in surface soils, consistent with the high retention reportedly associated with reduced microbial activity at lower temperatures associated with high elevation sites. However, amounts of Pb in the surface soils at some lower altitude sites were reduced over the same time period in the same study (ISA, section 2.3.3). New studies in the ISA also enhance our understanding of Pb sequestration in forest soils by providing additional information on the role of leaf litter as a Pb reservoir in some situations and the effect of litter decomposition on Pb distribution (ISA, section 2.3.3).

Recent research on Pb transport in aquatic systems has provided a large body of

observations confirming that such transport is dominated by colloids rich in iron and organic material (ISA, section 2.3.2). Recent research on Pb flux in sediments provides greater detail on resuspension processes than was available in the 2006 CD, including research on resuspended Pb largely associated with organic material or iron and manganese particles and research on the important role played by anoxic or depleted oxygen environments in Pb cycling in aquatic systems. This newer research is consistent with prior evidence in indicating that appreciable resuspension and release from sediments largely occurs during discrete events related to storms. It has also confirmed that resuspension is an important process that strongly influences the lifetime of Pb in bodies of water. Finally, there have been advances in understanding and modeling of Pb partitioning between organic material and sediment in aquatic environments (ISA, section 2.7.2).

The bioavailability of Pb is also an important component of understanding the effects Pb is likely to have on organisms and ecosystems (ISA, section 6.3.3). It is the amount of Pb that can interact within the organism that leads to toxicity, and there are many factors which govern this interaction (ISA, sections 6.2.1 and 6.3.3). The bioavailability of metals varies widely depending on the physical, chemical, and biological conditions under which an organism is exposed (ISA, section 6.3.3). Studies newly available since the last Pb NAAQS review provide additional insight into factors that influence the bioavailability of Pb to specific organisms (ISA, section 6.3.3). In general, this evidence is supportive of previous conclusions and does not identify significant new variables from those identified previously. Section 6.3.3 of the ISA provides a detailed discussion of bioavailability in terrestrial systems. With regard to aquatic systems, a detailed discussion of bioavailability in freshwater systems is provided in sections 6.4.3 and 6.4.4 of the ISA, and section 6.4.14 of the ISA discusses bioavailability in saltwater

systems.

In terrestrial systems, the amount of bioavailable Pb present determines the impact of soil Pb to a much greater extent than does the total amount present (ISA, section 6.3.11). In such ecosystems, Pb is deposited either directly onto plant surfaces or onto soil where it can bind with organic matter or dissolve in pore water. The Pb dissolved in pore water is particularly bioavailable to organisms in the soil and, therefore, the impact of this Pb on the ecosystem is potentially greater than soil Pb that is not in pore water (ISA, section 6.3.11).

In aquatic systems as in terrestrial systems, the amount of Pb bioavailable to organisms is a better predictor of effect on organisms than the overall amount of Pb in the system. Once atmospherically derived Pb enters surface water bodies through deposition or runoff, its fate and bioavailability are influenced by many water quality characteristics, such as pH, suspended solids levels and organic content (ISA, section 6.4.2). In sediments, bioavailability of Pb to sediment-dwelling organisms may be influenced by the presence of other metals, sulfides, iron oxides and manganese oxides and also by physical disturbance (ISA, section 2.6.2). For many aquatic organisms, Pb dissolved in the water column can be the primary exposure route, while for others sediment ingestion is significant (ISA, section 2.6.2). As recognized in the 2006 CD and further supported in the ISA, there is a body of evidence showing that uptake and elimination of Pb vary widely among aquatic species.

There is a substantial amount of new evidence in this review regarding the ecological effects of Pb on individual terrestrial and aquatic species with less new information available on marine species and ecosystems. On the whole, this evidence supports previous conclusions that Pb has effects on growth, reproduction and survival, and that under some conditions these effects can be adverse to organisms and ecosystems. The ISA provides evidence of effects in additional

species and in a few cases at lower exposures than reported in the previous review, but does not substantially alter our understanding of the ecological endpoints affected by Pb from the previous review. Looking beyond organism-level evidence, the evidence of adversity in natural systems remains sparse due to the difficulty in determining the effects of confounding factors such as co-occurring metals or system characteristics that influence bioavailability of Pb in field studies. The following paragraphs summarize the information presented in this review for terrestrial, aquatic and marine systems.

With regard to terrestrial ecosystems, recent studies cited in this review support previous conclusions about the effects of Pb, namely that increasing soil Pb concentrations in areas of Pb contamination (e.g., mining sites and industrial sites) can cause decreases in microorganism abundance, diversity, and function. Previous reviews have also reported on effects on bird and plant communities (2006 CD, section AX7.1.3). The shifts in bacterial species and fungal diversity have been observed near long-established sources of Pb contamination (ISA, section 6.3.12.7). Most recent evidence for Pb toxicity to terrestrial plants, invertebrates and vertebrates is from single-species assays in laboratory studies which do not capture the complexity of bioavailability and other modifiers of effect in natural systems (ISA, section 6.3.12.7). Further, models that might account for modifiers of bioavailability have proven difficult to develop (ISA, p. 6-16).

Evidence presented in the ISA and prior CDs demonstrates the toxicity of Pb in aquatic ecosystems and the role of many factors, including Pb speciation and various water chemistry properties, in modifying toxicity (ISA, section 1.7.2). Since the 2006 CD, additional evidence for community and ecosystem level effects of Pb is available, primarily in microcosm studies or field studies with other metals present (ISA, section 6.4.11). Such evidence described in previous

CDs includes alteration of predator-prey dynamics, species richness, species composition, and biodiversity. New studies available in this review provide evidence in additional habitats for these community and ecological-scale effects, specifically in aquatic plant communities and sediment-associated communities at both acute and chronic exposures involving concentrations similar to those previously reported (ISA, section 6.4.7). In many cases, it is difficult to characterize the nature and magnitude of effects and to quantify relationships between ambient concentrations of Pb and ecosystem response due to existence of multiple ecosystem-level stressors, variability in field conditions, and differences in Pb bioavailability (ISA, sections 1.7.3.7 and 6.4.7). Additionally, the degree to which air concentrations have contributed to such effects in freshwater ecosystems is largely unknown.

With regard to evidence in marine ecosystems, recently available evidence on the toxicity of Pb to marine algae augments the 2006 CD findings of variation in sensitivity across marine species. Recent studies on Pb exposure include reports of growth inhibition and oxidative stress in a few additional species of marine algae (ISA, section 6.4.15). Recent literature provides little new evidence of endpoints or effects in marine invertebrates beyond those reported in the 2006 CD. For example, some recent studies strengthen the evidence presented in the 2006 CD regarding negative effects of Pb exposure on marine invertebrates (ISA, section 6.4.15.2). Recent studies also identify several species exhibiting particularly low sensitivity to high acute exposures (ISA, section 6.4.15.2). Little new evidence is available of Pb effects on marine fish and mammals for reproductive, growth and survival endpoints that are particularly relevant to the population level of biological organization and higher (ISA, section 6.4.15). New studies on organism-level effects from Pb in saltwater ecosystems (ISA, section 6.4.15) provide little evidence to inform our understanding of linkages among atmospheric concentrations, ambient

exposures in saltwater systems and such effects or to inform our conclusions regarding the likelihood of adverse effects under conditions associated with the current NAAQS for Pb. Nor does the currently available evidence indicate significantly different exposure levels from the previous review at which ecological systems or receptors are expected to experience effects.

During the last review, the 2006 CD assessed the available information on critical loads for Pb (2006 CD, section 7.3). This information included publications on methods and example applications, primarily in Europe, specific to the bedrock geology, soil types, vegetation, and historical deposition trends in each European country (2006 CD, p. E-24), with no analyses available for U.S. locations (2006 CD, sections 7.3.4-7.3.6). As a result, the 2006 CD concluded that "[c]onsiderable research is necessary before critical load estimates can be formulated for ecosystems extant in the United States" (2006 CD, p. E-24).

For this current review, newly available evidence pertaining to critical loads analysis includes limited recent research on consideration of bioavailability in characterizing Pb effect concentrations or indices and on modeling approaches to incorporate chemistry effects on Pb speciation and bioavailability (ISA, sections 6.3.7 and 6.4.8). With consideration of this information and the four critical load analysis studies newly available in this review (none of which are for U.S. ecosystems), the ISA does not modify the conclusions noted above from the 2006 CD (ISA, sections 6.1.3, 6.3.7 and 6.4.8). In summary, the new information in this review does not appreciably change our evidence base or further inform our understanding of critical loads of Pb, including critical loads in sensitive U.S. ecosystems.

There is no new evidence since the last review that substantially improves our understanding of the relationship between ambient air Pb and measurable ecological effects. As stated in the last review, the role of ambient air Pb in contributing to ecosystem Pb has been

declining over the past several decades. It remains difficult to apportion exposure between air and other sources to better inform our understanding of the potential for ecosystem effects that might be associated with air emissions. As noted in the ISA, "[t]he amount of Pb in ecosystems is a result of a number of inputs and it is not currently possible to determine the contribution of atmospherically-derived Pb from total Pb in terrestrial, freshwater or saltwater systems" (ISA, section 6.5). Further, considerable uncertainties also remain in drawing conclusions from evidence of effects observed under laboratory conditions with regard to effects expected at the ecosystem level in the environment. In many cases it is difficult to characterize the nature and magnitude of effects and to quantify relationships between ambient concentrations of Pb and ecosystem response due to the existence of multiple stressors, variability in field conditions, and differences in Pb bioavailability at that level of organization (ISA, section 6.5). In summary, the ISA concludes that "[r]ecent information available since the 2006 Pb AQCD, includes additional field studies in both terrestrial and aquatic ecosystems, but the connection between air concentration and ecosystem exposure continues to be poorly characterized for Pb and the contribution of atmospheric Pb to specific sites is not clear" (ISA, section 6.5).

C. Summary of Risk Assessment Information

The risk assessment information available in this review and summarized here is based on the screening-level risk assessment performed for the last review, described in the 2006 REA, 2007 Staff Paper and 2008 notice of final decision (73 FR 66964, November 12, 2008), as considered in the context of the evidence newly available in this review (PA, section 5.2). As described in the REA Planning Document, careful consideration of the information newly available in this review, with regard to designing and implementing a full REA for this review, led us to conclude that performance of a new REA for this review was not warranted (REA

Planning Document, section 3.3). Based on their consideration of the REA Planning Document analysis, the CASAC Pb Review Panel generally concurred with the conclusion that a new REA was not warranted in this review (Frey, 2011b). Accordingly, the risk/exposure information considered in this review is drawn primarily from the 2006 REA as summarized below (PA, section 5.2 and Appendix 5A; REA Planning Document, section 3.1).

The 2006 screening-level assessment focused on estimating the potential for ecological risks associated with ecosystem exposures to Pb emitted into ambient air (PA, section 5.2; 2006 REA, section 7). A national-scale screen was used to evaluate surface water and sediment monitoring locations across the U.S. for the potential for ecological impacts that might be associated with atmospheric deposition of Pb (2006 REA, section 7.1.2). In addition to the national-scale screen (2006 REA, section 3.6), the assessment involved a case study approach, with case studies for areas surrounding a primary Pb smelter (2006 REA, section 3.1) and a secondary Pb smelter (2006 REA, section 3.2), as well as a location near a non-urban roadway (2006 REA, section 3.4). An additional case study, focused on consideration of atmospherically derived Pb effects on an ecologically vulnerable ecosystem (Hubbard Brook Experimental Forest), was identified (2006 REA, section 3.5). The Hubbard Brook Experimental Forest (HBEF), in the White Mountain National Forest, near North Woodstock, New Hampshire, was selected as a fourth case study because of its location and its long record of available data on concentration trends of Pb in three media (air or deposition from air, soil, and surface water). The HBEF case study was a qualitative analysis focusing on a summary review of the literature, without new quantitative analyses (2006 REA, Appendix E). For the other three case studies, exposure concentrations of Pb in soil, surface water, and/or sediment concentrations were estimated from available monitoring data or modeling analysis and then compared to ecological

screening benchmarks (2006 REA, section 7.1).

In interpreting the results from the 2006 REA, the PA considers newly available evidence that may inform interpretation of risk under the now current standard (PA, section 5.2). Factors that could alter our interpretation of risk would include new evidence of harm at lower concentrations of Pb, new linkages that enable us to draw more explicit conclusions as to the air contribution of environmental exposures, and new methods of interpreting confounding factors that were largely uncontrolled in the previous risk assessment. In general, however, the key uncertainties identified in the last review remain today.

The results for the ecological screening assessment for the three case studies and the national-scale screen for surface water and for sediment in the last review indicated a potential for adverse effects from ambient Pb to multiple ecological receptor groups in terrestrial and aquatic locations. Detailed descriptions of the location-specific case studies and the national screening assessment, key findings of the risk assessment for each, and an interpretation of the results with regard to past air conditions can be found in the 2006 REA. In considering the potential for adverse welfare effects to result from levels of air-related Pb that would meet the current standard, the findings of the 2006 REA, as summarized in the PA, are discussed below.

While the contribution to Pb concentrations from air as compared to nonair sources is not quantified, air emissions from the primary Pb smelter case study facility were substantial (2006 REA, Appendix B). In addition, this facility, which closed in 2013, had been emitting Pb for many decades, including some seven decades prior to establishment of any Pb NAAQS, such that it is likely air concentrations associated with the facility were substantial relative to the 1978 NAAQS, which it exceeded at the time of the last review. At the time of the last review and also since the adoption of the current standard, concentrations monitored near this facility have

exceeded the level of the applicable NAAQS (2007 Staff Paper, Appendix 2B-1; PA, Appendices 2D and 5A). Accordingly, this case study is not informative for considering the likelihood of adverse welfare effects related to Pb from air sources under air quality conditions associated with meeting the current Pb standard.

The secondary Pb smelter case study location continues to emit Pb, and the county where this facility is located does not meet the current Pb standard (PA, Appendices 2D and 5A). Given the exceedances of the current standard, which likely extend back over 4 to 5 decades, this case study also is not informative for considering the likelihood of adverse welfare effects related to Pb from air sources under air quality conditions associated with meeting the current Pb standard.

The locations for the near-roadway non-urban case study are highly impacted by past deposition of gasoline Pb. It is unknown whether current conditions at these sites exceed the current Pb standard, but, given evidence from the past of Pb concentrations near highways that ranged above the previous (1978) Pb standard (1986 CD, section 7.2.1), conditions at these locations during the time of leaded gasoline very likely exceeded the current standard. Similarly, those conditions likely resulted in Pb deposition associated with leaded gasoline that exceeds that being deposited under air quality conditions that would meet the current Pb standard. Given this legacy, consideration of the potential for environmental risks from levels of air–related Pb associated with meeting the current Pb standard in these locations is highly uncertain.

The extent to which past air emissions of Pb have contributed to surface water or sediment Pb concentrations at the locations identified in the national scale surface water and sediment screen is unclear. For some of the surface water locations, nonair sources likely contributed significantly to the surface water Pb concentrations. For other locations, a lack of nearby nonair sources indicated a potential role for air sources to contribute to observed surface

water Pb concentrations. Additionally, these concentrations may have been influenced by Pb in resuspended sediments and may reflect contribution of Pb from erosion of soils with Pb derived from historic as well as current air emissions.

The most useful case study to the current review is that of the Vulnerable Ecosystem Case Study located in the HBEF. This case study was focused on consideration of information which included a long record (from 1976 through 2000) of available data on concentration trends of Pb in three media (air or deposition from air, soil, and surface water). While no quantitative analyses were performed, a summary review of literature published on HBEF was developed. This review indicated: (1) atmospheric Pb inputs do not directly affect stream Pb levels at HBEF because deposited Pb is almost entirely retained in the soil profile; and (2) soil horizon analysis results showed Pb to have become more concentrated at lower soil depths over time, with the soil serving as a Pb sink, appreciably reducing Pb in pore water as it moves through the soil layers to streams (dissolved Pb concentrations were reduced from 5 μg/L to about 5 ng/L from surface soil to streams). As a result, the HBEF studies concluded that the contribution of dissolved Pb from soils to streams was insignificant (2006 REA, Appendix E). Further, atmospheric input of Pb, based on bulk precipitation data, was estimated to decline substantially from the mid-1970s to 1989; forest floor soil Pb concentrations between 1976 and 2000 were also estimated to decline appreciably (2006 REA, sections E.1 and E.2). In considering HBEF and other terrestrial sites with Pb burdens derived primarily from long-range atmospheric transport, the 2006 CD found that "[d]espite years of elevated atmospheric Pb inputs and elevated concentrations in soils, there is little evidence that sites affected primarily by long-range Pb transport have experienced significant effects on ecosystem structure or function" (2006 CD, p. AX7-98). The explanation suggested by the 2006 CD for this finding is "[l]ow concentrations of Pb in soil solutions, the

result of strong complexation of Pb by soil organic matter" (2006 CD, p. AZX7-98). While more recent soil or stream data on Pb concentrations are not available, we find it unlikely, given the general evidence for air Pb emissions and concentration declines over the past several decades (e.g., PA, Figures 2-1, 2-7 and 2-8), that conditions would have worsened from those on which these conclusions were drawn (e.g., soil data through 2000). Therefore, this information suggests that the now-lower ambient air concentrations associated with meeting the current standard would not be expected to directly impact stream Pb levels.

With regard to new evidence of Pb effects at lower concentrations, it is necessary to consider that the evidence of adversity due specifically to Pb in natural systems is limited, in no small part because of the difficulty in determining the effects of confounding factors such as multiple metals and modifying factors influencing bioavailability in field studies. Modeling of Pb-related exposure and risk to ecological receptors is subject to a wide array of sources of both variability and uncertainty. Variability is associated with geographic location, habitat types, physical and chemical characteristics of soils and water that influence Pb bioavailability and terrestrial and aquatic community composition. Lead uptake rates by invertebrates, fish, and plants may vary by species and season. For wildlife, variability also is associated with food ingestion rates by species and season, prey selection, and locations of home ranges for foraging relative to the Pb contamination levels (USEPA, 2005b).

There are significant difficulties in quantifying the role of air emissions under the current standard, which is significantly lower than the previous standard. As recognized in the PA, Pb deposited before the standard was enacted remains in soils and sediments, complicating interpretations regarding the impact of the current standard; historic Pb emitted from leaded gasoline usage continues to move slowly through systems along with more recently deposited Pb

and Pb derived from nonair sources (PA, section 1.3.2). The results from the location-specific case studies and the surface and sediment screen performed in the last review are difficult to interpret in light of the current standard and are largely not useful in informing judgments of the potential for adverse effects at levels of deposition meeting the current standard.

D. Conclusions on Adequacy of the Current Secondary Standard

1. Evidence- and Risk-based Considerations in the Policy Assessment

The current evidence, as discussed more fully in the PA, continues to support the conclusions from the previous review regarding key aspects of the ecological effects evidence for Pb and the effects of exposure associated with levels of Pb occurring in ecological media in the U.S. The EPA's conclusions in this regard are based on consideration of the assessment of the currently available evidence in the ISA, particularly with regard to key aspects summarized in the PA.

In considering the welfare effects evidence with respect to the adequacy of the current standard, the PA considers the array of evidence newly assessed in the ISA with regard to the degree to which this evidence supports conclusions about the effects of Pb in the environment that were drawn in the last review and the extent to which it reduces previously recognized areas of uncertainty. Further, the PA considers the current evidence and associated conclusions about the potential for effects to occur as a result of the much lower ambient Pb concentrations allowed by the current secondary standard (set in 2008) than those allowed by the prior standard, which was the focus of the last review. These considerations, as discussed below, inform the Administrator's conclusions regarding the extent to which the evidence supports or calls into question the adequacy of protection afforded by the current standard.

The range of effects that Pb can exert on terrestrial and aquatic organisms indicated by information available in the current review is summarized in the ISA (ISA, sections 1.7, 6.3 and

6.4) and largely mirrors the findings of the previous review (PA, section 5.1). The integrated synthesis contained in the ISA conveys how effects of Pb can vary with species and life stage, duration of exposure, form of Pb, and media characteristics such as soil and water chemistry. A wide range of organism effects are recognized, including effects on growth, development (particularly of the nervous system) and reproductive success (ISA, sections 6.3 and 6.4). Lead is recognized to distribute from the air into multiple environmental media, as summarized in section I.D above, contributing to multiple exposure pathways for ecological receptors. As discussed in section 5.1 of the PA, many factors affect the bioavailability of Pb to receptors in terrestrial and aquatic ecosystems, contributing to differences between laboratory-assessed toxicity and Pb toxicity in these ecosystems, and challenging our consideration of environmental impacts of Pb emitted to ambient air.

In studies in a variety of ecosystems, adverse ecosystem-level effects (including decreases in species diversity, loss of vegetation, changes to community composition, primarily in soil microbes and plants, decreased growth of vegetation, and increased number of invasive species) have been demonstrated near smelters, mines and other industries that have released substantial amounts of Pb, among other materials, to the environment (ISA, sections 6.3.12 and 6.4.12). As noted in the PA, however, our ability to characterize the role of air emissions of Pb in contributing to these effects is complicated because of coincident releases to other media and of other pollutants. Co-released pollutants include a variety of other heavy metals, in addition to sulfur dioxide, which may cause toxic effects in themselves and may interact with Pb in the environment, contributing uncertainty to characterization of the role of Pb from ambient air with regard to the reported effects (PA, section 5.1). These uncertainties limit our ability to draw conclusions regarding the extent to which Pb-related effects may be associated with ambient air

conditions that would meet the current standard.

The role of historically emitted Pb poses additional complications in addressing this question, as discussed in the PA (PA, section 1.3.2). The vast majority of Pb in the U.S. environment today, particularly in terrestrial ecosystems, was deposited in the past during the use of Pb additives in gasoline (2006 CD, pp. 2-82, AX7-36 to AX7-38, AX7-98; Johnson et al., 2004), although contributions from industrial activities, including metals industries, have also been documented (ISA, section 2.2.2.3, Jackson et al., 2004). The gasoline-derived Pb was emitted in very large quantities (2006 CD, p. AX7-98 and ISA, Figure 2-8) and predominantly in small sized particles which were widely dispersed and transported across large distances, within and beyond the U.S. (ISA, section 2.2). As recognized in the PA, historical records provided by sediment cores in various environments document the substantially reduced Pb deposition (associated with reduced Pb emissions) in many locations (PA, sections 2.3.1 and 2.3.3.2; ISA, section 2.2.1). As Pb is persistent in the environment, these substantial past environmental releases are expected to generally dominate current nonair media concentrations.

There is very limited evidence to relate specific ecosystem effects with current ambient air concentrations of Pb through deposition to terrestrial and aquatic ecosystems and subsequent movement of deposited Pb through the environment (e.g., soil, sediment, water, organisms). The potential for ecosystem effects of Pb from atmospheric sources under conditions meeting the current standard is difficult to assess due to limitations on the availability of information to fully characterize the distribution of Pb from the atmosphere into ecosystems over the long term, as well as limitations on information on the bioavailability of atmospherically deposited Pb (as affected by the specific characteristics of the receiving ecosystem). Therefore, while information available since the 2006 CD includes additional terrestrial and aquatic field studies, “the

connection between air concentration and ecosystem exposure and associated potential for welfare effects continues to be poorly characterized for Pb" (ISA, section 6.5). Such a connection is even harder to characterize with respect to the current standard than it was in the last review with respect to the previous, much higher, standard.

The current evidence also continues to support conclusions from the last review with regard to interpreting the risk and exposure results. These conclusions are based on consideration of the screening-level ecological risk assessment results from the last review as described in the 2006 REA and summarized in the notice of final rulemaking (73 FR 67009, November 12, 2008) and in light of the currently available evidence in the ISA (PA, section 5.2). As noted in section III.C above, the results from three of the four case studies and from the national screens are largely not useful in informing judgments of the potential for adverse effects at levels of deposition associated with conditions that meet the current standard. The Vulnerable Ecosystem Case Study at the HBEF is more illustrative with regard to the current review and, accordingly, is given primary consideration. The EPA concluded that atmospheric Pb inputs of the past did not directly affect stream Pb levels at HBEF because deposited Pb is almost entirely retained in the soil profile and that there was "little evidence that sites affected primarily by long-range Pb transport [such as this one] have experienced significant effects on ecosystem structure or function" (2006 CD, p. AX-98). We further note here that, as conditions are unlikely to have worsened since those on which those conclusions were based, we find it likely that current ambient air concentrations do not directly impact stream Pb levels under air quality conditions associated with meeting the now-current standard.

The available risk and exposure information continues to be sufficient to conclude that the 1978 standard was not providing adequate protection to ecosystems and, when considered

with regard to air-related ecosystem exposures likely to occur with air Pb levels that just meet the now-current standard, additionally does not provide evidence of adverse effects under the current standard.

2. CASAC Advice

In the current review of the secondary standard for Pb, the CASAC has provided advice and recommendations in their review of drafts of the ISA, of the REA Planning Document, and of the draft PA. We have additionally received comments from the public on drafts of these documents.[74]

In their advice and comments conveyed in the context of their review of the draft PA, the CASAC agreed with staff's preliminary conclusions that the available information since the last review is not sufficient to warrant revision to the secondary standard (Frey, 2013b). On this subject, the CASAC letter said that "[o]verall, the CASAC concurs with the EPA that the current scientific literature does not support a revision to the Primary Lead (Pb) National Ambient Air quality Standard (NAAQS) nor the Secondary Pb NAAQS" (Frey, 2013b, p. 1). The CASAC also recognized the many uncertainties and data gaps in the new scientific literature and recommended that research be performed in the future to address these limitations (Frey, 2013b, p. 2).

> *Given the existing scientific data, the CASAC concurs with retaining the current secondary standard without revision. However, the CASAC also notes that important research gaps remain. For example questions remain regarding the relevance of the primary standard's indicator, level, averaging time, and form for the secondary standard. Other areas for additional research to address data gaps and uncertainty include developing a critical loads approach for U.S. conditions and a multi-media approach to account for legacy Pb and contributions from different sources. Addressing these gaps may require reconsideration of the secondary standard in future assessments.*

[74] All written comments submitted to the agency will be available in the docket for this rulemaking, as will be transcripts and minutes of the public meetings held in conjunction with CASAC's review of drafts of the PA, the REA Planning Document and the ISA.

The very few public comments received on this review to date that have addressed adequacy of the current secondary Pb standard indicate support for retaining the current standard without revision, generally grouping the secondary standard with their similar view on the primary standard.

3. Administrator's Proposed Conclusions on the Adequacy of the Current Standard

Based on the evidence and risk assessment information that is available in this review concerning the ecological effects and potential public welfare impacts of Pb emitted into ambient air, the Administrator proposes to conclude that the current secondary standard provides the requisite protection of public welfare from adverse effects and should be retained.

In considering the adequacy of the current standard, the Administrator has considered the assessment of the available evidence and conclusions contained in the ISA; the staff assessment of and conclusions regarding the policy-relevant technical information, including screening-level risk information, presented in the PA; the advice and recommendations from CASAC; and public comments to date in this review. In the discussion below, the Administrator gives weight to the PA conclusions, with which CASAC has concurred, and takes note of key aspects of the rationale presented for those conclusions which contribute to her proposed decision.

The Administrator notes the conclusion in the PA that the body of evidence on the ecological effects of Pb, expanded in some aspects since the last review, continues to support identification of ecological effects in organisms relating to growth, reproduction, and survival as the most relevant endpoints associated with Pb exposure. In consideration of the appreciable influence of site-specific environmental characteristics on the bioavailability and toxicity of environmental Pb in our assessment here, the PA noted the lack of studies conducted under conditions closely reflecting the natural environment. The currently available evidence, while

somewhat expanded since the last review, does not include evidence of significant effects at lower concentrations or evidence of higher level ecosystem effects beyond those reported in the last review. There continue to be significant difficulties in interpreting effects evidence from laboratory studies to the natural environment and linking those effects to ambient air Pb concentrations. Further, the PA notes that the EPA is aware of no new critical loads information that would inform our interpretation of the public welfare significance of the effects of Pb in various U.S. ecosystems (PA, section 5.1). In summary, while new research has added to the understanding of Pb biogeochemistry and expanded the list of organisms for which Pb effects have been described, the PA notes there remains a significant lack of knowledge about the potential for adverse effects on public welfare from ambient air Pb in the environment and the exposures that occur from such air-derived Pb, particularly under conditions meeting the current standard (PA, section 6.2.1). Thus, the scientific evidence presented in detail in the ISA, inclusive of that newly available in this review, is not substantively changed, most particularly with regard to the adequacy of the now current standard, from the information that was available in and supported the decision for revision in the last review (PA, section 6.2.1).

With respect to exposure/risk-based considerations, the PA recognizes the complexity of interpreting the previous risk assessment with regard to the ecological risk of ambient air Pb associated with conditions meeting the current standard and the associated limitations and uncertainties of such assessments. For example, the location-specific case studies as well as the national screen conducted in the last review reflect both current air Pb deposition as well as past air and nonair source contributions (PA, section 6.3). The Administrator takes note of the PA conclusion that the previous assessment is consistent with and generally supportive of the evidence-based conclusions about Pb in the environment, yet the limitations on our ability to

apportion Pb between past and present air contributions and between air and nonair sources remain significant.

In the Administrator's consideration of the information available in this review of the Pb secondary standard, she gives weight to the PA conclusion that the currently available evidence and exposure/risk information do not call into question the adequacy of the current standard to provide the requisite protection for public welfare (PA, section 6.3). In so doing, she also notes the advice from CASAC in this review, including that "[g]iven the existing scientific data, the CASAC concurs with retaining the current secondary standard without revision." In light of these and the above considerations, the Administrator finds that the currently available information does not call into question the adequacy of the current standard to provide the requisite protection for public welfare and, accordingly, reaches the conclusion that it is appropriate to retain the current secondary standard without revision. The Administrator solicits comment on this conclusion.

IV. Statutory and Executive Order Reviews

Additional information about these statutes and Executive Orders can be found at http://www2.epa.gov/laws-regulations/laws-and-executive-orders.

A. Executive Order 12866: Regulatory Planning and Review and Executive Order 13563: Improving Regulation and Regulatory Review

This action is not a significant regulatory action and was, therefore, not submitted to the Office of Management and Budget for review.

B. Paperwork Reduction Act

This action does not impose an information collection burden under the Paperwork Reduction Act. There are no information collection requirements directly associated with

revisions to a NAAQS under section 109 of the CAA and this action does not propose any revisions to the NAAQS.

C. Regulatory Flexibility Act

I certify that this action will not have a significant economic impact on a substantial number of small entities under the Regulatory Flexibility Act. This action will not impose any requirements on small entities. Rather, this action proposes to retain, without revision, existing national standards for allowable concentrations of lead in ambient air as required by section 109 of the CAA. *See also American Trucking Associations v. EPA*. 175 F.3d at 1044-45 (NAAQS do not have significant impacts upon small entities because NAAQS themselves impose no regulations upon small entities).

D. Unfunded Mandates Reform Act

This action does not contain any unfunded mandate as described in the Unfunded Mandates Reform Act, 2 U.S.C. 1531-1538 and does not significantly or uniquely affect small governments. This action imposes no enforceable duty on any state, local or tribal governments or the private sector.

E. Executive Order 13132: Federalism

This action does not have federalism implications. It will not have substantial direct effects on the states, on the relationship between the national government and the states, or on the distribution of power and responsibilities among the various levels of government.

F. Executive Order 13175: Consultation and Coordination with Indian Tribal Governments

This action does not have tribal implications, as specified in Executive Order 13175. This action does not change existing regulations. It does not have a substantial direct effect on one or more Indian Tribes, since Tribes are not obligated to adopt or implement any NAAQS. The Tribal Authority Rule gives Tribes the opportunity to develop and implement CAA programs

such as the Pb NAAQS, but it leaves to the discretion of the Tribe whether to develop these programs and which programs, or appropriate elements of a program, they will adopt. Thus, Executive Order 13175 does not apply to this action.

G. Executive Order 13045: Protection of Children from Environmental Health and Safety Risks

This action is not subject to Executive Order 13045 because it is not economically significant as defined in Executive Order 12866. The health effects evidence and risk assessment information for this action, which focuses on children in addressing the at-risk population, is summarized in sections II.B, II.C and II.D, and described in the ISA and PA, copies of which are in the public docket for this action.

H. Executive Order 13211: Actions that Significantly Affect Energy Supply, Distribution or Use

This action is not subject to Executive Order 13211, because it is not a significant regulatory action under Executive Order 12866.

I. National Technology Transfer and Advancement Act

This rulemaking does not involve technical standards.

J. Executive Order 12898: Federal Actions to Address Environmental Justice in Minority Populations and Low-Income Populations

The EPA believes that this action will not have disproportionately high and adverse human health or environmental effects on minority, low-income or indigenous populations. The action proposed in this notice is to retain without revision the existing NAAQS for Pb based on the Administrator's conclusion that the existing standards protect public health, including the health of sensitive groups, with an adequate margin of safety. As discussed earlier in this preamble (see section II), the EPA expressly considered the available information regarding health effects among at-risk populations in reaching the proposed decision that the existing standards are requisite.

K. Determination Under Section 307(d)

Section 307(d)(1)(V) of the CAA provides that the provisions of section 307(d) apply to "such other actions as the Administrator may determine." Pursuant to section 307(d)(1)(V), the Administrator determines that this action is subject to the provisions of section 307(d).

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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.

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Gina McCarthy,
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