**6560-50-P**

**ENVIRONMENTAL PROTECTION AGENCY**

**40 CFR Part 50**

**[EPA-HQ-OAR-2010-0108; FRL-9952-87-OAR]**

**RIN 2060-AQ44**

# Review of the National Ambient Air Quality Standards for Lead

**AGENCY:** Environmental Protection Agency (EPA).

**ACTION:** Final 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 retaining the current standards, without revision.

**DATES:** This final rule is effective on **[INSERT DATE 30 DAYS AFTER DATE OF PUBLICATION IN THE FEDERAL REGISTER].**

**ADDRESSES:** The EPA has established a docket for this action under Docket ID No. EPA-HQ-OAR-2010-0108. Incorporated into this docket is a separate docket established for the Integrated Science Assessment for this review (Docket ID No. EPA-HQ-ORD-2011-0051). All documents in these dockets are listed on the www.regulations.gov Web site. 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 will be publicly available only in hard copy form. It 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.

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*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) Web site at http://www.epa.gov/ttn/naaqs/standards/pb/s_pb_index.html. These documents include the *Integrated Review Plan for 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.

**SUPPLEMENTARY INFORMATION:**

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**Executive Summary**

This document describes the completion of our current review of the NAAQS for Pb. This review of the standards and the air quality criteria (the scientific information upon which the standards are based) is required by the Clean Air Act on a periodic basis. In conducting this review, the EPA has carefully evaluated the currently available scientific literature on the health and welfare effects of Pb, focusing particularly on the information newly available since the conclusion of the last review in 2008. Between 2008 and 2014, the EPA prepared draft and final versions of the Integrated Science Assessment and the Policy Assessment, multiple drafts of which were subject to public review and comment and were reviewed by the Clean Air Scientific Advisory Committee, an independent scientific advisory committee established pursuant to the Clean Air Act and charged with providing advice to the Administrator. The EPA issued a proposed decision on the standards on January 5, 2015 (80 FR 278), and provided a 3-month period for submission of comments from the public. After consideration of public comments on the proposed decision and advice from the Clean Air Scientific Advisory Committee, the EPA has developed this document, which is the final step in the review process.

The prior review of the NAAQS for Pb was completed in 2008. As a result of that review, we significantly revised both the primary and secondary standards, including a lowering of the standard levels by an order of magnitude. The 2008 change to the primary standard was focused on providing the requisite protection for children and other at-risk populations against an

array of adverse health effects, most notably including neurological effects in children, including neurocognitive effects (e.g., IQ loss) and neurobehavioral effects. Although Pb has long been recognized to exert an array of adverse health effects, over the three decades from the time the standard was initially set in 1978 through its revision with the NAAQS review completed in 2008, the evidence base expanded considerably in a number of areas, including with regard to effects on neurocognitive function in young children at increasingly lower blood Pb levels. These effects formed the principal basis for the 2008 revisions to the primary standard.

The health effects evidence newly available in this review of the 2008 standard, as critically assessed in the ISA in conjunction with the full body of evidence, reaffirms conclusions on the broad array of effects recognized for Pb in the last review. Further, the currently available evidence is generally consistent with the evidence available in the last review, particularly with regard to key aspects of the evidence on which the current standard (set in 2008) is based. These key aspects include those regarding the relationships between air Pb concentrations and the associated Pb in the blood of young children as well as between total blood Pb levels and effects on children's IQ.

Based on consideration of the currently available health effects evidence in the context of this framework, and with support from the exposure/risk information, recognizing the uncertainties attendant in both, as well as the increasing uncertainty of risk estimates for lower air Pb concentrations, the Administrator concludes that the current primary standard provides the requisite protection of public health with an adequate margin of safety, including protection of at-risk populations. With regard to the secondary standard, the EPA has considered the currently available welfare effects evidence and screening-level risk information, including the general consistency of the current evidence with that available in the last review and the substantial

limitations in the current evidence that complicate conclusions regarding the potential for Pb emissions under the current, much lower standard to contribute to welfare effects. Based on these considerations, the Administrator concludes that the current secondary standard is requisite to protect public welfare from known or anticipated adverse effects. Thus, based on the EPA's review of the air quality criteria and the NAAQS for Pb, the EPA is retaining the current standards, without revision.

## 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."

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. 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.

[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).

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,[2] 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 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

[2] As used here and similarly throughout this document, 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.A.2.d below, the identification of sensitive groups (called at-risk groups or at-risk populations) involves consideration of susceptibility and vulnerability.

function has been performed by the Clean Air Scientific Advisory Committee (CASAC).[3]

*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 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 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 (completed in 2008), including completion of several regulations that 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

[3] 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.

nationwide that use furnaces to recover Pb from Pb-bearing scrap, mainly from automobile batteries (13 existing facilities). This action was estimated to result in a Pb emissions reduction of 13.6 tons per year (tpy) across the category (a 68 percent 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.

Pursuant to section 1412 of the Safe Drinking Water Act (SDWA), EPA sets public health goals and enforceable standards for drinking water quality. The Lead and Copper Rule (LCR) is a treatment technique rule. The LCR requires public water systems to treat the water to reduce corrosion of Pb and copper from premise plumbing and drinking water distribution

system components. When corrosion control treatment isn't enough, water systems must educate the public about Pb in drinking water and replace lead service lines, which are the pipes that connect buildings to the drinking water mains (40 CFR 141.80-141.91). The importance of corrosion control treatment was illustrated by the recent events in Flint, MI, when Pb levels in drinking water increased after the water system did not maintain corrosion control treatment when the system changed its water supply. Section 1417 of the SDWA additionally prohibits the use of any pipe, any pipe or plumbing fitting or fixture, any solder, or any flux in the installation or repair of any public water system or any plumbing in a residential or non-residential facility providing water for human consumption, that is not lead free as defined by the Act.[4]

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,

[4] Effective in January 2014, the amount of Pb permitted in pipes, fittings, and fixtures was lowered (see "Section 1417 of the Safe Drinking Water Act: Prohibition on Use of Lead Pipes, Solder, and Flux" at http://www.epa.gov/dwstandardsregulations/section-1417-safe-drinking-water-act-prohibition-use-lead-pipes-solder-and).

certification, and work practice requirements for persons engaged in home renovation, repair and 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.[5] 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 "National Waste Minimization Program" (https://archive.epa.gov/epawaste/hazard/wastemin/web/html/tools.html), "Sustainable Management of Electronics" (https://www.epa.gov/smm-electronics), and the "Sustainable Materials Management (SMM) Electronics Challenge" (https://www.epa.gov/smm-electronics/sustainable-materials-management-smm-electronics-challenge).

The EPA's research program identifies, encourages and conducts research needed to develop methods and tools to characterize and help reduce risks related to Pb exposure. An example of one such effort is the EPA's Integrated Exposure Uptake Biokinetic Model for Lead in Children (IEUBK model), which 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 pharmacokinetic

[5] See, e.g., "Implementation of the Mercury-Containing and Rechargeable Battery Management Act" available from https://www.epa.gov/hw and facts and figures on recycling and disposal in the U.S. at https://www.epa.gov/smm/advancing-sustainable-materials-management-facts-and-figures.

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)[6] 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, statistics for the distribution of blood Pb levels in non-Hispanic black and lower socioeconomic groups of young children, which are generally higher than those for that population as a whole, have also declined, as have the differences in these statistics between non-Hispanic black and other groups, as well as between lower and higher socioeconomic groups (USEPA, 2013a, sections 3.4.1, 5.2.3 and 5.2.4; Jones et al., 2009).

[6] Since the completion of the ISA, more recent NHANES data indicate the geometric mean blood Pb concentration for children in the U.S. population, aged one to five, to have declined to 0.97 μg/dL in the 2011-2012 survey (CDC, 2015).

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 (USEPA, 2011c). As of January, leaded gasoline for on-road use is known to be available (along with unleaded gasoline) in three countries.[8]

The EPA is a contributor to the Global Alliance to Eliminate Lead Paint, a voluntary public-private partnership jointly led by the World Health Organization and the United Nations Environment Programme (UNEP) to prevent children's exposure to Pb from paints containing Pb and to minimize occupational exposures to Pb paint. The objective of this alliance is to promote 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

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

[8] UNEP. "Leaded Petrol Phase-out: Global Status as at January 2016" map downloaded from http://www.unep.org/transport/new/pcfv/

equivalent representatives of Mexico and Canada comprise the CEC's senior governing body (CEC Council).[9]

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

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,

[9] 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.

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 air quality criteria and standards for Pb was initiated in November 2004 (69 FR 64926, November 9, 2004); the agency's plans for preparation of the Air Quality Criteria Document (AQCD) and conduct of the NAAQS review were presented in documents completed in 2005 and early 2006 (USEPA, 2005a; USEPA 2006b).[10] 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,

[10] 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).

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 the 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).[11] The 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

[11] 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).

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. The 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, November 12, 2008).

The November 2008 preamble to the final rule described the EPA's decision to revise the primary and secondary standards for Pb, as discussed more fully in sections II.A.1 and III.A below. In consideration of the much-expanded health effects evidence on neurocognitive effects of Pb in children, the EPA substantially revised the primary standard level from 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 preambles 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 (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)[12] 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

[12] As of this review, the document developed in NAAQS reviews in which the air quality criteria are assessed, previously the AQCD, is the ISA, and the document describing the OAQPS staff evaluation, previously the Staff Paper, is the PA. These documents are described in the IRP.

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 the 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 its 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. We have considered the 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, as described in the Policy Assessment (PA) and proposal.

A draft of the 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). The proposed decision (henceforth "proposal") on this review of the NAAQS for Pb was signed on December 19, 2014, and published in the *Federal Register* on January 5, 2015. Written comments were received from twelve commenters during the public comment period on the proposal. Significant issues raised in the public comments and the EPA's responses to those comments are discussed in the preamble of this final action.

As in prior NAAQS reviews, the EPA is basing its decision in this review on studies and related information included in the ISA and PA[13], which have undergone CASAC and public

[13] As a new REA was not warranted in this review, the exposure and risk information from the last review (2007 REA; 2006 REA) is summarized in the PA in the context of the currently available health and welfare effects evidence.

review. The studies assessed in the ISA[14] and PA, and the integration of the scientific evidence presented in them, have undergone extensive critical review by the EPA, the CASAC, and the public. The rigor of that review makes these studies, and their integrative assessment, the most reliable source of scientific information on which to base decisions on the NAAQS, decisions that all parties recognize as of great import. Decisions on the NAAQS can have profound impacts on public health and welfare, and NAAQS decisions should be based on studies that have been rigorously assessed in an integrative manner not only by the EPA but also by the statutorily mandated independent scientific advisory committee, as well as the public review that accompanies this process. Some commenters have referred to and discussed individual scientific studies on the health effects of Pb that were not included in the ISA ("'new' studies"). In considering and responding to comments for which such "new" studies were cited in support, the EPA has provisionally considered the cited studies in the context of the findings of the ISA. The EPA's provisional consideration of these studies did not and could not provide the kind of in-depth critical review described above.

The decision to rely on studies and related information included in the ISA, REAs and PA, which have undergone CASAC and public review, is consistent with the EPA's practice in prior NAAQS reviews and its interpretation of the requirements of the CAA. Since the 1970 amendments, the EPA has taken the view that NAAQS decisions are to be based on scientific studies and related information that have been assessed as a part of the pertinent air quality

[14] Studies were identified for the Pb ISA based on the review's opening "call for information" (75 FR 8934), as well as literature searches 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" (ISA, p. 1-2). In a subsequent step, "[s]tudies that have undergone scientific peer review and have been published or accepted for publication and reports that have undergone review are considered for inclusion in the ISA" and "[a]nalyses conducted by EPA using publicly available data are also considered for inclusion in the ISA" (ISA, p. xlv). 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).

criteria, and the EPA has consistently followed this approach. This longstanding interpretation was strengthened by new legislative requirements enacted in 1977, which added section 109(d)(2) of the Act concerning CASAC review of air quality criteria. See 71 FR 61144, 61148 (October 17, 2006, final decision on review of NAAQS for particulate matter) for a detailed discussion of this issue and the EPA's past practice.

As discussed in the EPA's 1993 decision not to revise the NAAQS for ozone, ''new'' studies may sometimes be of such significance that it is appropriate to delay a decision on revision of a NAAQS and to supplement the pertinent air quality criteria so the studies can be taken into account (58 FR at 13013–13014, March 9, 1993). In the present case, the EPA's provisional consideration of ''new'' studies concludes that, taken in context, the ''new'' information and findings do not materially change any of the broad scientific conclusions regarding the health and welfare effects and exposure pathways of Pb in ambient air made in the air quality criteria. For this reason, reopening the air quality criteria review would not be warranted.

Accordingly, the EPA is basing the final decisions in this review on the studies and related information included in the Pb air quality criteria that have undergone CASAC and public review. The EPA will consider the "new" studies for purposes of decision making in the next periodic review of the NAAQS for Pb, which the EPA expects to begin soon after the conclusion of this review and which will provide the opportunity to fully assess these studies through a more rigorous review process involving the EPA, CASAC, and the public.

*D. Multimedia, Multipathway Aspects of Lead*

Since Pb is distributed from air to other media and is persistent, our review of the NAAQS for Pb considers the protection provided against 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 is 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 is 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 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 are 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.[15] 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*

[15] The time it takes for exposures to be reduced in response to reductions in air Pb concentrations varies with the various inhalation and ingestion exposure pathways. 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 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, compared with smaller particles that remain in the 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 aerodynamic diameter less than or equal to 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 the 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 NAAQS surveillance network regulations (40 CFR part 58, appendix D, paragraph 4.5) require source-oriented monitoring sites, and also the collection of one year of Pb-TSP measurements at 15 specific airports. The indicator for the current Pb NAAQS is Pb-

TSP, although in some situations,[16] Pb-$PM_{10}$ concentrations may be used in judging nonattainment. Currently, more than 200 Pb-TSP monitors are in operation; these are a mixture of source- and non-source-oriented monitors (PA, p. 2-14).

Since the phase-out of Pb in on-road gasoline, Pb is widely recognized as a near-source air pollutant, the ambient air concentrations of which generally fall off quickly with distance from sources. Variability in ambient 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 placement of monitor sites near sources of air Pb emissions that 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 that emits 0.50 or more tons of Pb per year and from each airport that emits 1.0 or more tons of Pb per year.[17] The EPA Regional Administrators may require additional monitoring beyond the minimum requirements where the likelihood of Pb air quality violations is significant or where the emissions density, topography, or population locations are complex and varied. Such locations may include those near additional industrial Pb sources, recently closed industrial sources and other sources of re-entrained Pb dust, as well as airports where piston-engine aircraft emit Pb associated with combustion of

[16] 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.

[17] 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)).

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[18] in order to gather additional information on ambient air Pb concentrations near airports, including specifically 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 under section 231(a)(2)(A) of the CAA of whether Pb emissions from piston-engine aircraft cause or contribute to air pollution which may reasonably be anticipated to endanger public health or welfare (see for example, EPA's Advance Notice of Proposed Rulemaking on Lead Emissions From Piston-Engine Aircraft Using Leaded Aviation Gasoline, 75 FR 22439, April 28, 2010). The EPA is conducting this investigation separate from the Pb NAAQS review. As a whole, the various data gathering efforts and analyses are expected to improve our understanding of Pb concentrations in ambient air near airports and conditions influencing these concentrations.

Monitoring agencies may also conduct non-source-oriented Pb monitoring at the NCore monitoring sites.[19] In 2015, all NCore sites with a population of 500,000 or more (as defined by

[18] 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 or characteristics were selected as they were expected, "collectively, to identify airports with the highest potential to have ambient air Pb concentrations approaching or exceeding the Pb NAAQS" (75 FR 81132, December 27, 2010).

[19] 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.

the U.S. Census Bureau)[20] were measuring Pb concentrations, with a 2014 analysis indicating generally similar numbers of sites measuring Pb in TSP and Pb in $PM_{10}$ (Cavender, 2014). These numbers may change in the future as the requirement for Pb monitoring at these sites was recently eliminated in consideration of current information indicating concentrations at these sites to be well below the Pb NAAQS and of the existence of other monitoring networks that provide information on Pb concentrations at similar types of sites (81 FR 17248, March 28, 2016). The data available for the NCore sites indicate maximum 3-month average concentrations (of Pb-$PM_{10}$ or Pb-TSP) well below the level of the Pb NAAQS, with the large majority of these sites indicating maximum 3-month average concentrations at or below 0.01 $\mu g/m^3$ (Cavender, 2014). Other monitoring networks that provide data on Pb in $PM_{10}$ or $PM_{2.5}$ at non-source-oriented urban, and some rural, sites 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 (also known as the IMPROVE network).

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).

*F. Summary of Proposed Decisions*

For reasons discussed in the proposal and summarized in sections II.B.1 and III.B.1

[20] Metropolitan area population size information is available at the Census Bureau Web site (http://www.census.gov/population/www/metroareas/metroarea.htm).

below, the Administrator proposed to retain the current primary and secondary standards for Pb, without revision.

*G. Organization and Approach to Final Decisions*

This action presents the Administrator's final decisions in the current review of the primary and secondary Pb standards. The final decisions addressing standards for Pb are based on a thorough review in the ISA of scientific information on known and potential human health and welfare effects associated with exposure to Pb associated with levels typically found in the ambient air. These final decisions also take into account the following: (1) staff assessments in the PA of the most policy-relevant information in the ISA as well as quantitative health and welfare exposure and risk information; (2) CASAC advice and recommendations, as reflected in its letters to the Administrator and its discussions of drafts of the ISA and PA at public meetings; (3) public comments received during the development of these documents, both in connection with CASAC meetings and separately; and (4) public comments received on the proposal.

The primary standard is addressed in section II and the secondary standard is addressed in section III. Section IV addresses applicable statutory and executive order reviews.

## II. Rationale for Decision on the Primary Standard

This section presents the rationale for the Administrator's decision to retain the existing primary Pb standard. This rationale is based on a thorough review in the ISA of the latest scientific information, generally published through September 2011, on human health effects associated with Pb and pertaining to the presence of Pb in the ambient air. This decision 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 the CASAC's letters to the Administrator; (3) public comments received during the development of these documents, either in connection with CASAC meetings or separately, and (4) public comments received on the proposal.

Section II.A provides background on the general approach for review of the primary standard for Pb and brief summaries of key aspects of the currently available health effects and exposure/risk information. Section II.B presents the Administrator's conclusions on adequacy of the current standard, drawing on consideration of this information, advice from the CASAC, and comments from the public. Section II.C summarizes the Administrator's decision on the primary standard.

*A. Introduction*

As in prior reviews, the general approach to reviewing the current primary standard is 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. 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. The 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. 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.

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 in section II.A.1 below, 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.A.2 through II.A.4 below provide an overview of the current health effects and quantitative exposure and risk information with a focus on the specific policy-relevant questions identified for these categories of information in the PA (PA, chapter 3).

1. Background on the Current Standard

The current primary standard was established in the last review, which was completed in 2008 (73 FR 66964, November 12, 2008), and is set at a level that is one-tenth the level of the prior standard. 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. While recognizing that Pb has been demonstrated to exert "a broad array of deleterious effects on multiple organ systems," the 2008 review focused on the effects most pertinent to recent ambient air exposures, which are those associated with relatively lower exposures and associated blood Pb levels (73 FR 66975,

November 12, 2008). Given the general scientific 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, 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). Particular focus was given to the public health implications of effects of air-related Pb on cognitive function (e.g., IQ).

The conclusions reached by the Administrator in the 2008 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). He gave particular attention to 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).

Based on consideration of the health effects evidence, supported by the quantitative risk analyses, 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 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 sensitive or 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 identifying the appropriate revised standard, revisions to each of the four basic elements of the NAAQS (indicator, averaging time, form and level) were considered.

With regard to indicator, the Administrator decided to retain Pb-TSP as the indicator. The EPA recognized 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, and that ultra-coarse Pb particles may have a greater presence in areas near sources where Pb concentrations are highest, contributing uncertainty with regard to whether a Pb-$PM_{10}$-based standard would also effectively control

ultra-coarse Pb particles (73 FR 66991, November 12, 2008). Accordingly, Pb-TSP was retained as the indicator in order to provide sufficient public health protection from the broad range of particle sizes of ambient air Pb, including ultra-coarse particles, given the recognition that Pb in all particle sizes contributes to Pb in blood and associated health effects (73 FR 66991, November 12, 2008).[21]

With regard to averaging time and form for the revised standard, after giving consideration 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, two changes were made. These were to a rolling 3-month average, thus giving equal weight to all 3-month periods, and to the method for deriving the 3-month average to provide equal weighting to each month. Both of these changes afford greater weight to each individual month than did the calendar quarter form of the 1978 standard, thus tending to control both the likelihood that any month will exceed the level of the standard and the magnitude of any such exceedance. The Administrator decided on these changes in recognition of the complexity inherent in this aspect of the standard which is greater for Pb 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 in an integrated manner 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, recognizing that some factors might imply support for a period as short as a month for averaging time, and others

[21] However, in order to take advantage of the increased precision of Pb- $PM_{10}$ 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-$PM_{10}$), a role was provided for Pb- $PM_{10}$ measurements in the monitoring required for a Pb-TSP standard (73 FR 66991, November 12, 2008) at sites not influenced by sources of ultra-coarse Pb, and where Pb concentrations are well below the standard (73 FR 66991, November 12, 2008).

supporting use of a longer time, with all having associated uncertainty. Based on such 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 (73 FR 66996, November 12, 2008).

In reaching the decision on level for the revised standard, that, in combination with the specified choice of indicator, averaging time, and form, the Administrator judged requisite to protect public health, including the health of sensitive groups, with an adequate margin of safety, he considered the evidence using a very specifically defined framework, referred to as an air-related IQ loss evidence-based framework (73 FR 67004, November 12, 2008). 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 with air-related exposures that are in the high end of the national distribution of such 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. Consideration of this framework additionally recognizes that 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 air-related IQ loss evidence-based framework are air-to-blood ratios[22] and C-R functions for the relationship between blood Pb concentration and IQ response in young children (73 FR 67004, November 12, 2008). In applying and drawing conclusions from the framework, the Administrator additionally took into consideration the uncertainties inherent in these two 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 µg/dL blood Pb per $\mu g/m^3$ air concentration), and slope for the C-R function in terms of points of IQ decrement per µg/dL blood Pb. In light of the uncertainties and limitations associated with the evidence on these relationships, and other considerations, application of the air-related IQ loss evidence-based framework was recognized to provide "no evidence- or risk-based bright line that indicates a single appropriate level" for the standard (73 FR 67005-67006, November 12, 2008). Rather, the framework was seen as a useful guide, in the context of the specified averaging time and form, for consideration of health risks from exposure to levels of Pb in the ambient air to inform 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).

Use of the air-related IQ loss evidence-based framework to inform selection of the standard level involved consideration of the evidence for the two primary input parameters

[22] 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).

mentioned above. With regard to air-to-blood ratio estimates, the evidence in the 2008 review indicated a broad range of estimates, each with limitations and associated uncertainties. Based on this evidence, the Administrator concluded that 1:5 to 1:10 represented a reasonable range to consider and focused on 1:7 as a generally central value (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 Pb levels that most closely reflected the then-current population of young children in the U.S.,[23] recognizing the EPA's identification of four such analyses and giving weight to the central estimate or median of the resultant linear 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

[23] 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). Using the air-to-blood ratio 1:7, the estimated air-related blood Pb level associated with the final standard level is approximately 1 µg/dL. In the 2008 decision, the EPA noted that even if it assumed, as an extreme hypothetical example, that the mean for the general population of U.S. children included zero contribution from air-related sources and added that to the estimate of air-related Pb, the result would still be below the lowest mean blood Pb level among the set of C-R analyses (73 FR 67002).

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 air-related IQ loss evidence-based 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 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 national ambient air quality standard, 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 air-related IQ loss evidence-based 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). Further description of the EPA's consideration of this issue is provided in the preamble to the final decision rule (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 $PM_{10}$ 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. Overview of Health Effects Evidence

In this section, we provide an overview of the information presented in section II.B of the proposal on policy-relevant aspects of the health effects evidence available for consideration in this review. Section II.B of the proposal provides a detailed summary of key information contained in the ISA and in the PA on health and public health effects of Pb, focusing particularly on the information most relevant to consideration of effects associated with the presence of Pb in ambient air (80 FR 290-297, January 5, 2015). The subsections below briefly outline this information in the five topic areas addressed in section II.B of the proposal.

a. 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).[24] Consistent with those conclusions, in the context of pollutant exposures considered relevant to the Pb NAAQS review,[25] the ISA determines that causal relationships[26] 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 certain types of effects on the nervous system in adults, and on immune system function, as well as with cancer,[27] as likely to

[24] 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).

[25] 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).

[26] 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).

[27] 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).

be causal[28] (ISA, Table 1-2, sections 1.6.4 and 1.6.7).

Among the nervous system effects of 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). While some studies are newly available of other effects in children with somewhat lower blood Pb levels than previously available for these effects, nervous system effects continue to receive prominence in the current review, as in previous reviews, with particular emphasis on those affecting cognitive function and behavior in children (ISA, section 4.3), with conclusions that are consistent with findings of the last review. For example, 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.

[28] 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).

> 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).

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. For example, the evidence newly available for Pb relationships with cardiovascular effects in adults includes some studies with somewhat lower blood Pb levels than in the last review. However, 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).[29] Evidence available in future reviews may better inform this issue. Recognizing this, the extensive assessment of the full body of evidence available in this review contributed to the following major conclusions drawn by the ISA regarding health effects of Pb in adults (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

[29] 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).

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.

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. Similarly, given the strength of the evidence, including the greater confidence in conclusions regarding the exposures contributing to the observed effects, we focus in this review, as in the last, on neurocognitive effects in young children.

b. 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 (as discussed further in section II.B.3 of the proposal). 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.

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).[30] 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, 5.2.1). 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).[31] 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 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

[30] 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).

[31] 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.

findings for early (e.g., at age 2 years) or concurrent blood Pb levels (ISA, section 4.3.11).[32] 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.

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 exposure circumstances contributing to blood Pb levels measured in epidemiological studies. For example, the evidence does not indicate the exposure magnitude and timing that are eliciting such effects. Across the full evidence base, the effects for which our understanding of relevant exposure circumstances is greatest are neurocognitive effects in young

[32] 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.).

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.

c. Nervous System Effects in Children

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).[33]

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

[33] 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).

et al., 2006; Bellinger, 2008; Canfield, 2008; Tellez-Rojo, 2008; Kirrane and Patel, 2014).[34] Newly available in this review are two studies reporting association of blood Pb levels prior to 3 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).

Newly available in this review are also several studies in older children on neurocognitive effects and other nervous system effects. As described in section II.B.3 of the proposal, however, these studies are focused on population groups of ages for which the available information indicates exposure levels were higher earlier in childhood. Thus, in light of this information, although the blood Pb levels in the studies 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. Rather, this information makes these studies relatively uninformative with regard to evidence of effects associated with lower exposure levels than provided by evidence previously available.

Recognizing the complexity associated with interpretation of studies involving older

[34] 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.

cohorts,[35] 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.[36] 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 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[37]) for young child population subgroups (age 5 years or younger) with individual blood Pb

[35] 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.

[36] 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).

[37] The Bayley Scales of Infant Development, Mental Development Index (BSID MDI) 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).

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, p. 4-274).

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 recognized in section II.A.2.b 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 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 levels 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 levels were higher in the lower peak blood Pb level subsets than the higher groups (ISA, section 4.3.12; Lanphear et al., 2005).[38] 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 of the proposal (drawn from Table 3 of the 2008 preamble to the final rule [73 FR 67003, November 12, 2008], and Kirrane

[38] As described in the PA and noted in the proposal, 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).

and Patel, 2014).[39] These analyses were important inputs for the air-related IQ loss evidence-based 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. Specifically, the framework focused on the median of the four average linear slope estimates from the studies recognized in Table 3 of the 2008 decision (73 FR 67003, November 12, 2008). As shown in Table 1 of the proposal, the median is unchanged by consideration of the information newly available in this review.[40]

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 of the proposal. In summary, the newly available evidence does not

[39] One of these four subgroup analyses is 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.

[40] 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 recalculation described above has no impact on conclusions drawn from the framework.

substantively alter our understanding of the C-R relationship (including quantitative aspects) for neurocognitive impact, such as IQ, with blood Pb in young children.

d. At-Risk Populations

In this section, as elsewhere, we use the term "at-risk populations"[41] 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 referred to as sensitive groups (as in section I.A above). In identifying factors that increase risk of Pb-related health effects, we have 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).

In considering factors that increase risk by contributing to increased exposure or to increased blood Pb levels over those otherwise associated with a given Pb exposure, 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 of the proposal and briefly noted in section II.A.2.c above. An important implication of this finding is

[41] 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.

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 incremental 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.

The information newly available in this review has not appreciably 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 discussed in section II.B.2 of the proposal and briefly noted in section II.A.2.b 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). Thus, 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.

An additional physiological risk factor that contributes to increased blood Pb levels is nutritional status, which can play a role in Pb absorption from the gastrointestinal tract, with iron-, calcium- and zinc-deficient diets contributing to increased Pb absorption and associated blood Pb levels (ISA, sections 3.2.1.2, 5.1, 5.3.10 and 5.4). Risk factors based on increased exposure include spending time in proximity to sources of Pb to ambient air or other

environmental media, such as 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 in some study groups, 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).[42] Although the differences in blood Pb levels, nationally, 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).[43] The evidence is also suggestive of increased risk associated with several other factors: older

[42] The approach used by the EPA in evaluating the evidence regarding factors that may influence the risk of Pb-related health effects is described in chapter 5 of the ISA.

[43] Although the evidence for SES continues to indicate increased blood Pb levels in lower income children, 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.

adulthood,[44] pre-existing disease (e.g., hypertension), variants for certain genes and increased stress (ISA, section 5.3.4).

In summary, we recognize the sensitivity of the prenatal period and several stages 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.

e. 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,

[44] 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).

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). In considering population risk, the distribution of effects across members of the population is important. 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). 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).[45] 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.

As indicated 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 (e.g., concentrations near or above the current standard). Analyses in the PA indicate this group to be a very small subset of all young children in the U.S. Together the analyses 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). It is these children that were the Administrator's focus in revising the primary standard in 2008.

3. Overview of Information on Blood Lead Relationships with Air Lead

This section provides a brief overview of the information summarized in section II.C of the proposal on key aspects of the information available in this review on blood Pb as a biomarker and on relationships of blood Pb with air Pb (80 FR 298-300, January 5, 2015). Blood

[45] 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).

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. 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. 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[46] "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[47] 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 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

[46] 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 $\mu g/m^3$). 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). Slopes are presented as simply the value of x.

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

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 to 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).

4. Overview of Risk and Exposure Assessment Information

This section provides a brief overview of key aspects of the risk and exposure assessment information available in this review, which is based primarily on the exposure and risk assessment developed in the last review of the Pb NAAQS.[48] This overview is drawn from the summary presented in the proposal (80 FR 300-305, January 5, 2015). 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 its 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).[49] Accordingly, the exposure/risk 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 in section II.D of the proposal and in section 3.4 and Appendix 3A of the PA.

The focus for the risk assessment and associated estimates is on Pb derived from sources emitting Pb to ambient air. In order to characterize exposure and risk from these pathways,

[48] The information in this review is based on the assessment from the last review, 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; proposal, section II.D).

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

however, the assessment also recognized the role of Pb exposure pathways unrelated to Pb in ambient air (2007 REA, section 2.1). 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, such as inhalation of ambient air, ingestion of indoor dust, outdoor soil/dust and diet or drinking water (as recognized in section I.D above). 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 older water distribution pipes and Pb in materials used in food processing).

Limitations in our data and modeling tools handicapped our ability to address the various complexities associated with exposure to ambient air Pb and to fully separate the nonair contributions to Pb exposure from estimates of air-related Pb exposure and risk. 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, characterized by bounds within which air-related Pb risk is estimated 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 (PA, section 3.4).

Key aspects of the 2007 REA, such as the exposure populations, exposure or dose metric, health effects endpoint and risk metric 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.[50] 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 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). The generalized (local)

[50] 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).

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.

Air-related risk estimates for the two case studies are accompanied by a number of uncertainties (summarized in section II.D.3 of the proposal and described in detail in section 3.4 of the PA). 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, as recognized at the time of the last review.[51] The multimedia and persistent nature of Pb, the role of multiple exposure pathways, and the contributions of nonair sources of Pb to human exposure

[51] As summarized in section II.D.3 of the proposal, 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), and the newly available information in this review did not substantially reduce any of the primary sources of uncertainty identified to have the greatest impact on risk estimates (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. Nor is 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).

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). Of particular note among the assessment limitations are limitations in the assessment design, data and modeling tools that handicapped us from sharply separating Pb linked to ambient air from Pb that is not air related. The resultant, approximate, air-related risk bounds, however, encompass estimates drawn from the air-related IQ loss evidence-based framework, providing a rough consistency and general support, as was the case in the last review (73 FR 67004, November 12, 2008).

*B. Conclusions on the Primary Standard*

In drawing conclusions on the adequacy of the current primary Pb standard, in view of the advances in scientific knowledge and additional information now available, the Administrator considers the evidence base, information and policy judgments that were the foundation of the last review and reflects upon the body of evidence and information newly available in this review. The Administrator has taken into account both evidence-based and exposure- and risk-based considerations, advice from CASAC and public comment. 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- and 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 of the proposal) and consideration of those results in the PA.

As described in section II.A.2 of the proposal, consideration of the evidence and exposure/risk information in the PA and by the Administrator is framed by consideration of a

series of key policy-relevant questions. Section II.B.1 below summarizes the rationale for the Administrator's proposed decision, drawing from section II.E.4 of the proposal. A fuller presentation of PA considerations and conclusions, and advice from the CASAC, which were taken into account by the Administrator, is provided in sections II.E.1 through II.E.3 of the proposal. Advice received from CASAC in this review is briefly summarized in section II.B.2 below, and public comments on the proposed decision are addressed in section II.B.3. The Administrator's conclusions in this review regarding the adequacy of the current primary standard are described in section II.B.4.

1. Basis for the Proposed Decision

At the time of the proposal, the Administrator carefully considered the assessment of the current evidence and conclusions reached in the ISA; the currently available exposure/risk information, including associated limitations and uncertainties; considerations and staff conclusions and associated rationales presented in the PA; the advice and recommendations from CASAC; and public comments that had been offered up to that point. In reaching her proposed conclusion on the primary standard, the Administrator first took note of the PA discussion with regard to the complexity and associated uncertainties 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 standard in other NAAQS reviews. For the pollutants in the other reviews, the focus is on inhalation as the single route of exposures, which provides a relatively simpler context than the multiple exposure pathways that are relevant to Pb. Additionally, an important component of the evidence base for most other NAAQS pollutants is the availability of studies that have investigated an association between concentrations of the pollutant in ambient air and the occurrence of health effects plausibly related to ambient air exposure to that pollutant.

Such studies of associations with air concentrations do not figure prominently in the review of the NAAQS for Pb. Rather, the evidence base in this review includes most prominently epidemiological studies focused on associations of blood Pb levels in U.S. populations with health effects plausibly related to Pb exposures occurring by multiple pathways. 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.

In considering the nature and magnitude of the array of uncertainties that are inherent in the scientific evidence and analyses, the Administrator recognized that the current 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 her considerations for the proposal, she took into account both the well-established body of evidence on the health effects of Pb, which continues to support identification of neurocognitive effects in young children as the most sensitive endpoint associated with Pb exposure, and of the

recognition in the PA, with which the CASAC concurred, of increased uncertainty in characterizing the relationship of effects on IQ with blood Pb levels below those represented in the evidence base and also in projecting the magnitude of blood Pb response to ambient air Pb concentrations at and below the level of the current standard. In this light, she based her proposed decision on her consideration of the current evidence within the conceptual and quantitative context of the air-related IQ evidence-based loss framework; the available information and advice from CASAC regarding the public health significance of neurocognitive effects; and the limitations and uncertainties inherent in the evidence and its consideration within this framework. The Administrator additionally recognized support from the exposure/risk information, with its attendant uncertainties.

In her consideration of the air-related IQ loss evidence-based framework, the Administrator took note of the PA finding, with which the CASAC concurred, that application of the air-related IQ loss evidence-based 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 took note of the PA finding (described in section II.E.1 of the proposal, and with which the CASAC concurred) that the currently available evidence base, while somewhat expanded since the last review, is not supportive of appreciably different conclusions with regard to air-to-blood ratios or C-R functions for neurocognitive decrements in young children.

In the Administrator's consideration of the level of public health protection provided by the current standard, she gave weight to CASAC advice in the last review (and similar views expressed in the last review by public health experts, such as the American Academy of

Pediatrics), 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, she additionally noted 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, and CASAC provided no alternate advice in this area in the current review (PA, pp. 4-33 to 4-34). Accordingly, with the objective identified in the CASAC advice from the 2008 review in mind, the Administrator considered the role of the air-related IQ loss evidence-based framework in reviewing the level of protection provided by the current standard. In so doing, the Administrator recognized distinctions between estimates produced by the framework, for which the conceptual context is a subset of U.S. children, and specific quantitative public health policy goals for air-related IQ loss for the entire U.S. population of children. She additionally took note of the PA conclusion on the size of the population subset that might pertain to the situation represented by the framework (areas with elevated air Pb concentrations equal to the standard level), as well as uncertainties associated with the framework estimates, particularly at successively lower standard levels. In summary, the Administrator concluded in the proposal that the current evidence, as considered within the conceptual and quantitative context of the evidence-based framework, and current air monitoring information indicate 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.

The Administrator based her proposed conclusions on consideration of the health effects evidence, including consideration of this evidence in the context of the air-related IQ loss

evidence-based framework, and with support from the exposure/risk information, recognizing the uncertainties attendant with both. In so doing, she took 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 proposed 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 the exposure/risk information, and how to consider their associated uncertainties. Based on these considerations and the judgments summarized here, the Administrator proposed to conclude 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.

The Administrator's proposed conclusion that the current standard provides the requisite protection and that a more restrictive standard would not be requisite additionally recognized that the uncertainties and limitations associated with many aspects of the estimated relationship between air Pb concentrations and blood Pb levels and associated health effects are amplified with consideration of increasingly lower air concentrations. In reaching her proposed conclusion,

she took 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 judged 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 proposed to conclude that the current standard should be retained, without revision.

2. CASAC Advice in this Review

In 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, p. 1). The CASAC 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" (Frey, 2013b, p. 1).

The CASAC comments additionally indicated agreement 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, the 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 the 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 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).

3. Comments on the Proposed Decision

The majority of public comments on the proposal supported the Administrator's proposed decision to retain the current primary standard, without revision. This group includes the National Association of Clean Air Agencies (NACAA), both of the state agencies that submitted comments and nearly all of the industry organizations that submitted comments. All of these commenters generally noted their agreement with the rationale provided in the proposal and noted the CASAC's concurrence with the EPA conclusion that the current evidence does not

support revision to the standard. Most also cited the EPA and CASAC statements that information newly available in this review has not substantially altered our previous understanding of at-risk populations, C-R relationships or effects from exposures lower than what was previously examined and does not call into question the adequacy of the current standard. Some commenters stated that multimedia or multipathway aspects of Pb make the review of the primary standard for Pb subject to greater uncertainty than reviews of primary NAAQS for other pollutants and/or noted greater uncertainty with consideration of lower blood Pb and standard levels. Some also noted that EPA's task in setting NAAQS is not to reduce risk to zero but to identify a standard that is neither more nor less stringent than necessary. The EPA generally agrees with these commenters and with the CASAC regarding the adequacy of the current primary standard and the lack of support for revision of the standard.

Four submissions recommending revision of the standard were received; all four advocated a tightening of the standard. These commenters include two individuals, a secondary Pb smelting company, and the Children's Health Protection Advisory Committee to the EPA (CHPAC).[52] In support of their view that the standard should be revised, all four commenters

[52] As described in its charter, the CHPAC is a policy-oriented committee providing policy advice to EPA related to the development of regulations, guidance and policies to address children's environmental health, consistent with provisions of the Federal Advisory Committee Act (http://www.epa.gov/faca/childrens-health-protection-advisory-committee-charter-september-11-2015). The role and scope of activities for the CHPAC differs from those of the CASAC, which is the independent scientific review committee fulfilling the function described in the CAA of reviewing the air quality criteria and the NAAQS for protection of public health and welfare and making recommendations to the Administrator concerning revisions as may be appropriate (as described in section 109(d)(2) of the Act and summarized in section I.A above).

generally stated that there is no safe level of Pb exposure.[53] The CHPAC submission, to which the smelting company submission repeatedly cited, asserted that a lower standard is needed to protect children from impacts related to neurodevelopmental and low birthweight effects, stating that studies it cited that have been published since the cut-off for the ISA indicate effects on children's IQ at "appreciably lower" Pb exposures than those recognized in the last review and raise concerns regarding cumulative effects of multiple chemical exposures. These commenters additionally cited the PA's presentation of the 2007 REA results that included lower risk estimates for alternative more stringent standards, stating that minority and low-income groups are more greatly impacted by Pb, and that for these reasons the standard should be lowered. The CHPAC submission also suggests consideration of some transient sources to provide support for a more stringent standard. Among the reasons given for their recommendations to substantially lower the standard level, the individual commenters variously stated that not revising or lowering the standard will allow increases in air Pb in locations near some sources of Pb emissions, such as airports, and that the persistence of Pb indicated the need for a more stringent standard.

The four commenters that supported revision of the standard suggested a wide array of alternatives. The CHPAC repeated the view it expressed in the 2008 review that the standard should be revised to the most stringent alternative analyzed in the 2007 REA (a potential standard with an averaging time of one month and a level of 0.02 $\mu g/m^3$). One individual commenter expressed a preference for a standard level of 0.0005 $\mu g/m^3$. Another individual

[53] In expressing this view, some commenters cited statements by various government agencies regarding their interpretation of children's blood Pb levels with regard to risk management decisions based on consideration of the available information in those risk management contexts (e.g., CDC, 2005; Cal EPA, 2007; NYDHMH, 2010). The scientific information on health effects of Pb considered by these agencies was also available and, to the extent relevant to consideration of the adequacy of the NAAQS, was assessed in the current and, in some cases, also the prior review. As discussed below, the conclusion that a threshold level for neurocognitive effects has not been identified was a consideration of the EPA in the last review, and the current one.

commenter urged revision to the lowest feasible standard, and the smelting company recommended that EPA adopt an approach similar to a local air quality management district's emissions standards regulation[54] that requires air monitoring at large Pb acid battery recycling metal melting facilities to meet, by a future date, a 30-day average Pb concentration of 0.1 $\mu g/m^3$, which the company indicated its technology can address.

We agree with commenters that a threshold level for neurocognitive effects has not been identified in the current evidence, as stated in section II.A.2.c above, and described in more detail in the ISA. We additionally note that the lack of an established threshold of effects is not uncommon among the criteria pollutant evidence bases. For example, in past reviews of the primary standards for ozone and particulate matter, the EPA has recognized that the available epidemiological evidence neither supports nor refutes the existence of thresholds at the population level, while noting uncertainties and limitations in studies that make discerning thresholds in populations difficult (e.g., 73 FR 16444, March 27, 2008; 71 FR 61158, October 17, 2006). The lack of a discernible threshold of exposure associated with health effects does not of itself provide support for revision of an existing standard or for revision to the most stringent standard one might identify. As recognized in section I.A above, the CAA does not require the Administrator to establish a primary national ambient air quality standard at a zero-risk level or at background concentrations (*Lead Industries v. EPA*, 647 F.2d at 1156 n.51; *Mississippi v. EPA*, 744 F. 3d at 1351), but rather at a level that reduces risk sufficiently so as to protect public health with an adequate margin of safety, and the selection of any particular approach for providing an adequate margin of safety is a policy choice left specifically to the Administrator's judgment (*Lead Industries Association v. EPA*, 647 F.2d at 1161-62; *Mississippi*, 744 F. 3d at

[54] This commenter referred to a March 2015 amendment of a California South Coast Air Quality Management District rule on emission standards for lead and other toxic air contaminants from large lead-acid battery recycling facilities in that state air quality district.

1353). The CAA requirement in establishing a standard is that it be set at a level of air quality that is requisite, meaning "sufficient, but not more than necessary" (*Whitman v. American Trucking Ass'ns*, 531 U.S. 457, 473 [2001]).

In the setting of the current standard in 2008, a key consideration of the Administrator was the recognition of the lack of a discernible threshold level in the evidence with respect to neurocognitive effects associated with Pb exposure. This recognition, which differed from the scientific consensus at the time the previous standard was set in 1978, led the Administrator in 2008 to depart from the threshold-based approach used in setting the 1978 standard and to focus on consideration of air-related Pb in the context of the air-related IQ loss evidence-based framework (described in section II.A.1 above). In the current review of the 2008 standard, while recognizing the continued lack of a discernible threshold of exposure associated with neurocognitive effects, the CASAC commented regarding effects at very low Pb levels when expressing its view that the scientific evidence does not support revision to the Pb NAAQS. It stated that "[a]lthough there is evidence that even very low Pb levels are related to measurable reductions in IQ in children, the extent to which the blood Pb levels observed in children are linked to ambient air Pb levels below the current standard (as opposed to other sources of Pb in the environment) has not been established" (Frey, 2013b, Consensus Response to Charge Questions, pp. 7-8).[55]

The four submissions recommending a revised standard variously cite a number of studies as providing support for their view. Some of these studies have been reviewed in the ISA, some were published too late to be included in the ISA, and a few others were of a type that are

[55] The CASAC recognized the multimedia and legacy aspects of Pb that, unlike the case for other criteria air pollutants, complicate consideration of the risks of Pb concentrations in ambient air (Frey, 2013b, p. 1).

not generally included in the ISA (e.g., review articles).[56] As discussed in section I.C above, we have provisionally considered studies that were not in the ISA or in previous AQCDs ("new" studies)[57] which some of these commenters cite in statements about evidence of effects at low exposures and in the presence of other pollutants. We conclude that these studies are consistent with the scientific conclusions reached in the ISA, including those related to blood Pb levels in studies from which effects on IQ have been reported and related to co-exposure with other metals. Taken in context, the information from these studies and these findings do not materially change any of the broad scientific conclusions of the ISA regarding the health effects and exposure pathways of Pb in ambient air on which the Administrator based her proposed conclusions as well as her final conclusions in this review, as described in section II.B.4 below. We additionally note that with regard to the inputs for the air-related IQ loss evidence-based framework, a key aspect of the Administrator's rationale for her proposed decision to retain the current primary standard (as described in section II.E.4 of the proposal), none of the cited studies indicate a steeper blood Pb-IQ slope or greater air-to-blood ratio than those assessed in the ISA and considered in the PA and the proposal.

We respectfully disagree with the comment from CHPAC that studies available since the cut-off date for the ISA contradict the PA conclusions regarding blood Pb levels in children and

[56] Some studies cited by commenters are review articles or government reviews (e.g., Henn et al., 2014; Grandjean and Landrigan, 2014; Jakubowski, 2011; NTP, 2011), which are not generally cited in the ISA because the ISA considers the original studies underlying a review article, rather than a review's interpretation of the studies. Further, in the case of government reviews, such reports generally review the literature for specific purposes of those government agencies (which differ from the focus for the ISA). Many of the scientific studies reviewed in these reports (as well as the other reviews), however, were considered relevant to review of the lead air quality criteria (based on the description of study selection for inclusion in the preamble to the ISA), and thus were assessed in this review.

[57] These studies are listed in a memorandum to the rulemaking docket (Kirrane, 2016).

effects on cognitive function measures, such as IQ.[58] Of the studies cited in the comment that were published subsequent to the date for publication in the ISA, one is an analysis that relies on data from studies that were published prior to 2008 and assessed in the last review (Budtz-Jorgensen et al., 2013). These data were the subject of the pooled analysis by Lanphear et al (2005) which we assessed in both the last and the current review. As such, this commenter-cited publication does not present a new study of children with lower blood Pb levels; rather, it reanalyzes existing data using a different approach for a different purpose.[59] The other two of the commenter-cited publications are review articles that do not present information on specific blood Pb levels associated with IQ effects. Thus, we do not find these publications to be contrary to the discussion and associated conclusions in the PA or to indicate the current standard to be

[58] The PA recognized the complexity associated with considering the evidence regarding exposure levels associated with health effects, and in particular effects on cognitive function measures, including IQ, which the evidence base indicates to be the most sensitive endpoint. The PA observed that the evidence available in this review is generally consistent with that available in the last review with regard to blood Pb levels in young children at which such effects have been reported. Noting that blood Pb levels are a reflection of exposure history, particularly in early childhood, the PA concludes by extension that the currently available evidence does not indicate Pb effects at exposure levels appreciably lower than recognized in the last review. In so doing, the PA continued to focus in this review (as in the last review) on the evidence of effects in young children for which our understanding of exposure history is less uncertain (PA, pp. 3-21 to 3-26).

[59] This analysis uses the data from the same studies analyzed by Lanphear et al (2005) to extrapolate below the blood Pb concentrations measured in the studies and estimate a 95 percent lower confidence bound on the estimated blood Pb concentration associated with a 1 point decrement in IQ (Budtz-Jorgensen et al., 2013). Unlike the prior study by Lanphear et al (2005) and similar epidemiological analyses of IQ and blood Pb, which are intended to produce a quantitative description of the change in IQ associated with blood Pb concentrations in the studied children, this analysis is focused on estimating a lower bound confidence limit on the incremental concentration in blood Pb, as compared to zero, associated with a single point IQ decrement. Even if we were to interpret the results of the Budtz-Jorgensen et al (2013) analysis as providing another estimate of C-R function for IQ decrement based on the pooled dataset from Lanphear et al (2005), we note that that dataset is already represented among the four low blood Pb analyses on which we focused in identifying a slope estimate for use with the air-related IQ loss evidence-based framework, and as noted in section II.B.3 of the proposal, revision or replacement of the estimate for the pooled dataset has no impact on conclusions drawn from the framework (80 FR 29295, January 5, 2015).

inadequate.

We further disagree with the suggestion in the CHPAC submission that the evidence related to co-exposures to other pollutants, such as metals, provides a basis for concluding that the current standard is not requisite. The ISA assessment of the strength of the evidence for co-exposures to other pollutants, such as other metals, to contribute to increased risk of a Pb-related health effects concluded the evidence to be suggestive, "but overall the evidence was limited" (ISA, sections 1.9.6 and 5.4). With regard to the articles cited by the CHPAC that have been published subsequent to the ISA, the general conclusions of these review articles (Henn et al., 2014; Grandjean and Landrigan, 2014) are consistent with conclusions of the ISA. As stated in the ISA, "interactions between Pb and co-exposure with other metals were evaluated in recent epidemiologic and toxicological studies of health effects" and "[h]igh levels of other metals, such as Cd and Mn, were observed to result in greater effects for the associations between Pb and various health endpoints but evidence was limited due to the small number of studies" (ISA, p. 5-43). We note that even in raising co-exposure as a concern, the comments recognize that the potential for such impacts is not well understood. Further, the comments do not explain how the limited information regarding this factor supports their conclusion that the current standard does not provide the requisite protection or leads to the specific revisions the comments suggest, and we find no such support in the current evidence.

We additionally disagree with the comment that the currently available evidence indicates that the current standard is not protective of effects such as low birth weight. For example, the CHPAC cites epidemiological studies reporting associations of maternal or cord blood Pb concentrations with reduced fetal growth (Xie et al., 2013; Nishioka et al., 2014), stating that these studies strengthen the association of decreased birth weight and maternal blood

Pb levels. Although we would agree that these studies present an addition to the evidence base overall, they do not provide a basis for change in the conclusion of the ISA, which states, "Some well-conducted epidemiologic studies report associations of maternal Pb biomarkers or cord blood Pb with preterm birth and low birth weight/fetal growth; however, the epidemiologic evidence is inconsistent overall and findings from experimental animal studies are mixed" (ISA, p. 1-18). In citing these studies, in fact, the CHPAC also stated its view that the findings of these studies are consistent with a larger study that was assessed in the ISA; it did not explain how these studies support its view that the current standard provides inadequate protection from such effects, and we find no such support.

With regard to information related to Pb impacts in minority and low-income populations, which some comments suggested provided a basis for a more stringent standard, we note that we have considered the available information on such impacts, as recognized in section II.A.2.d above and summarized more fully in section II.B.4 of the proposal and in section 3.3 of the PA. As all of these documents have recognized, the ISA identifies non-white populations as at-risk populations, with this conclusion based primarily on findings of higher blood Pb levels in black compared to white populations (ISA, section 5.4).[60] Blood Pb levels have also been found

[60] Recent data suggest that differences in blood Pb levels between young black and white children is decreasing over time (ISA, section 5.2.3, 5.4). Although more recent data are not available by age group, the CDC data through 2011-2012 indicate little or no difference between non-Hispanic blacks, Mexican Americans or all Hispanics and non-Hispanic whites at the central tendencies of the populations and reduced differences at the 95th percentile (CDC, 2015). Findings of some studies indicate that non-white populations may be at greater risk of Pb-related health effects although, as described in the ISA, this could be related to confounding by other factors (ISA, sections 5.3.7 and 5.4).

to be higher in low SES groups as compared to higher SES[61] (ISA, sections 5.3.6, 5.2.4 and 5.4). However, as noted in the ISA, the number of studies examining the relationship of SES with Pb-related health effects is limited, and the results have differed with regard to finding increased risk with higher or lower SES (ISA, Table 5-1, p. 5-42). The comments generally identify impacts in minority and low income groups as a reason EPA should revise the standard, although they provide no explanation for how the currently available information leads to that conclusion or provides a basis for the alternative standards the comments suggest. [62] While our assessment of the health effects evidence in this review concluded there was adequate evidence for race or ethnicity (and suggestive evidence for SES) to contribute to increased risk of Pb-related health effects, we do not find this information to call into question the adequacy of protection provided by the current primary standard. Nor did the CASAC find this to be the case, based on its review of the scientific materials in this review, including three drafts of the ISA in which the evidence for these factors was presented. Further, to the extent such differences may be related to

[61] As with differences among groups of different races and ethnicities, "[t]he gap between SES groups with respect to Pb body burden appears to be diminishing," although blood Pb levels continue to be higher among lower-income children (ISA, p. 1-80, sections 1.9.6, 5.1, 5.2.1.1, 5.2.4 and 5.4), leading the ISA to conclude that the evidence is suggestive of SES as a risk factor for Pb-related health effects (as summarized in section II.A.2.d above).

[62] In making this statement, these commenters cite a 1988 study on blood Pb and early childhood scores on the BSID MDI infant cognitive development test (Bellinger et al., 1988). The study found that 18 and 24 month BSID MDI scores of the "lower" SES children were adversely affected at lower cord blood Pb levels than were scores of the "higher" SES children, finding significantly lower scores of the lower SES children with cord blood Pb levels of 6-7 µg/dL as compared to children of this SES group with cord blood Pb levels less than 3 µg/dL (Bellinger et al., 1988; USEPA, 1990a; USEPA, 2006). As the study cohort was mostly middle to upper-middle class, the "lower" SES group "refers to [families of SES] less than the highest SES levels and is probably in fact [of SES levels] much closer to the median of the U.S. population than the term suggests" (USEPA, 1990a, p. 53). The ISA considered these study findings in the context of considering available evidence on this issue in the current review (ISA, section 5.3.6; Bellinger et al., 1990). The ISA found that the available study results are limited, have differed with regard to finding increased risk with higher or lower SES and that "they do not clearly indicate whether groups with different socioeconomic status differ in Pb-related changes for cognitive function" (ISA, p. 5-34, Table 5-1, p. 5-42).

exposure contributions from air Pb and proximity to air sources,[63] we note that children that are exposed to air-related Pb in areas with elevated air Pb concentrations near or equal to the level of the standard are among those that were the focus of the 2008 decision, as recognized in sections II.A.1 and II.A.2.e above, and are the focus of the decision described in section II.B.4 below to retain the standard set in 2008.[64]

With regard to consideration of the potential for risk reduction from lower air concentrations, the PA stated that "the uncertainties and limitations associated with 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" (PA, p. 4-35). Contrary to the suggestion by the CHPAC and the smelter company, the PA did not conclude that there would be public health benefits from a lower standard and that such benefits were not large enough to warrant revising the standard. Rather, the PA notes that "[a]s recognized at the time of the last review, exposure and risk modeling conducted for [the REA]

[63] As noted in section I.D above and described in more detail in the PA and ISA, sources of Pb to which children are exposed also include consumer goods, dust or chips of peeling Pb-containing paint and ingestion of Pb in drinking water conveyed through Pb pipes, as well as historically deposited Pb in urban soils (ISA, pp. pp. lxxix to lxxx).

[64] Additionally, the focus of the air-related IQ loss evidence-based framework on C-R functions observed for children with low blood Pb levels closer to those observed in U.S. children today reflects evidence-based conclusions from the last review, affirmed in this review, of a steeper slope for the C-R relationship at lower as compared to higher blood Pb levels. As noted in section II.A.2.d above, 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. The 2008 revision of the primary Pb standard focused on the incremental impact of air-related Pb on young children and in so doing, recognized the greater incremental impact for those children with lower absolute blood Pb levels. Accordingly, the decision focused on those C-R studies involving the lowest blood Pb levels (as summarized in II.A.1 above). Although the comment did not indicate how information that some groups may be generally more highly exposed to Pb should be used, we note that for the Administrator to rely on C-R functions from analyses for higher blood Pb study groups (with a less steep slope) would lead to consideration of a higher standard level, and would not provide the desired protection for the sensitive group of children with lower blood Pb levels that are exposed to air-related Pb in areas with air Pb concentrations at the level of the standard (73 FR 67002-07, November 12, 2008; 80 FR 311-313, January 5, 2015).

was complex and subject to significant uncertainties" (PA, p. 3-67) and recognizes "increasing uncertainty of risk estimates" for air Pb concentrations below those associated with the current standard (PA, p. 4-35). The PA further stated that that "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). The CASAC stated that it agreed with this conclusion regarding "[t]he obvious uncertainty" articulated in the PA, additionally stating, as noted above, that "[a]lthough there is evidence that even very low Pb levels are related to measurable reductions in IQ in children, the extent to which the blood Pb levels observed in children are linked to ambient air Pb levels below the current standard (as opposed to other sources of Pb in the environment) has not been established" and, accordingly (as noted below), that the current information does not provide support for lowering the primary standard (Frey, 2013b, Consensus Response to Charge Questions, pp. 6-8). These conclusions from the CASAC and the PA findings were among the considerations that led to the Administrator's proposed decision (summarized in section II.B.1 above) and her final decision in this review, as described in section II.B.4 below, that, based on the current scientific information, including information regarding at-risk populations, as well as uncertainties and limitations associated with the current information, the current primary standard provides the requisite protection of public health with an adequate margin of safety, including the health of at-risk populations.

The comment regarding a potential for increases in air Pb near sources of Pb emissions if the standard is not revised does not explain how such a potential provides support for revising the standard. The comment also suggests that EPA consider two alternative standard levels well below the current standard level while providing no explanation of why a revised standard with

either of the suggested levels would be requisite. With regard to the potential for increases in air Pb near sources of Pb emissions if the standard is not revised, we note that such a concern, to the extent it applies to the current standard, would also pertain to any more stringent Pb standard except in the extreme case in which the standard is set such that there is no location with air quality conditions better than those that just meet the standard. As discussed in sections II.B.1 above and II.B.4 below, the Administrator has considered the current evidence and exposure/risk information with regard to the potential for a revised standard to offer additional protection, found there to be substantial uncertainty associated with such a potential, and concluded that the current standard is requisite. Regarding the possibility that air Pb concentrations could increase in some locations, we additionally note that the Clean Air Act and associated EPA permitting regulations restrict increases in air Pb concentrations (and in other pollutants for which there are NAAQS) in various circumstances, both in areas already meeting the NAAQS as well as those in nonattainment (e.g., New Source Review regulations at 40 CFR part 51, subpart I, applicable in attainment and nonattainment areas; General Conformity regulations at 40 CFR 93.150-165, applicable in nonattainment and maintenance areas; and, the general anti-backsliding requirements under Section 110(l) of the Clean Air Act).

Regarding the view expressed by some commenters that the most restrictive standard

assessed in the 2007 REA should be adopted, [65] or that the standard level should be revised to a concentration described in one comment as the average air Pb concentration in pristine locations, we note the greater uncertainty in risk estimates associated with air quality scenarios for air Pb concentrations increasingly below those of current conditions. Additionally, the PA described the "increasing uncertainty recognized for air quality scenarios involving air Pb concentrations increasingly below the current conditions for each case study, recognizing that such uncertainty is due in part to modeling limitations deriving from uncertainty regarding relationships between ambient air Pb and outdoor soil/dust Pb and indoor dust Pb" (PA, 4-34). Further, the PA concluded, and the CASAC agreed, 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; PA, p.4-35 to 4-36). The CASAC further stated that "there is not justification for modifying the current standard based on these data at this time" (Frey, 2013b, Consensus Response to Charge Questions, p. 8). In reaching her proposed decision to retain the current standard, the Administrator took note of the PA conclusion and associated CASAC agreement and additionally recognized 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

[65] The alternative more stringent primary standard suggested by the CHPAC was the most stringent assessed in the 2007 REA and included both a lower level and a shorter averaging time than those for the current standard. In establishing the current standard in 2008, the EPA considered these suggestions regarding level and averaging time, which were also made by the CHPAC at that time. The EPA's considerations with regard to averaging time in establishing the current standard in 2008 are summarized in section II.E.1 of the proposal and section 4.1.1.2 of the PA. The comments from the CHPAC repeat its recommendation from the last review and do not provide any additional information or explanation in support of its view on a revised averaging time. The EPA response to substantive comments on averaging time in the last review from the CASAC and the public, including the CHPAC, is described in the notice of final decision (73 FR 66991-996, November 12, 2008).

concentrations" (80 FR 313). Finally, in the proposal, as in the final decision described in section II.B.3 below, 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. With regard to comments recommending consideration of technological feasibility in judging the requisiteness of the primary standard, we note, as we have described in section I.A above, the EPA may not consider technological feasibility or attainability in determining what standard is requisite to protect public health with an adequate margin of safety.

Comments on topics less directly related to consideration of the primary standard included recommendations for addressing data gaps and uncertainties to inform future reviews. Additionally, one comment focused on pathways by which Pb may be further distributed in the environment, recommending use of a "more robust [monitoring] network to adequately estimate children's lead exposures from transient and other sources," emphasizing building demolition and Pb wheel weights. This comment also states that the PA overlooks the contribution from these and other sources and therefore may underestimate the number of children exposed to Pb from transient sources. Another comment described leaded aviation gasoline and airports as a source of Pb emissions but did not explain how such information was relevant to the Administrator's proposed decision that the current standard provided the requisite protection and should be retained without revision.

With regard to the need for research, the PA highlighted key uncertainties associated with reviewing and establishing NAAQS for Pb and areas for future health-related research, model development, and data gathering. The topic areas of key uncertainties, research questions and data gaps that were highlighted in the PA with regard to review of the health-based primary standard overlap with many raised by commenters. We encourage research in these areas,

although we note that research planning and priority setting are beyond the scope of this action.

With regard to the monitoring network in place for Pb NAAQS surveillance, the current regulations require air monitors in areas that are expected to or have been shown to experience or contribute to exceedance of the standards. As described in section I.E above, this includes requirements for monitors in areas with non-airport sources emitting 0.5 tpy or where an airport emits 1.0 or more tpy, based on either the most recent National Emissions Inventory or other scientifically justifiable methods and data (40 CFR part 58, appendix D, section 4.5). The establishment of the source-oriented monitoring requirement reflects our conclusion that monitoring should be presumptively required at sites near sources that have estimated Pb emissions in exceedance of a Pb "emissions threshold" (73 FR 67025). This monitoring requirement applies not only to existing industrial sources of Pb, but also to fugitive sources of Pb (e.g., mine tailing piles, closed industrial facilities) and airports where leaded aviation gasoline is used. Additionally, as noted in section I.E above, to account for other sources that may contribute to a maximum Pb concentration in ambient air in excess of the Pb NAAQS, the monitoring regulations also grant the EPA Regional Administrator the authority to require additional monitoring "where the likelihood of Pb air quality violations is significant or where the emissions density, topography, or population locations are complex and varied" (40 CFR part 58, appendix D, section 4.5(c)).

In addition to this monitoring required for Pb NAAQS surveillance, state or local agencies may site additional monitors and there are also particulate matter monitoring networks that collect Pb data in specific particle size fractions in many urban areas (40 CFR part 58, appendix D, section 4.5). Further, as described in section I.E above,[66] monitoring data collected

[66] The various air Pb monitoring networks are summarized in section I.E above and described in more detail in section 2.2.1 of the PA.

at NCore sites in large population areas, in combination with the data for all other non-source-oriented sites, including those in urban areas, indicate air Pb concentrations well below the Pb NAAQS (as summarized in section I.E above). Accordingly, we believe that the current Pb monitoring requirements are consistent with the currently available information regarding sources of Pb to the ambient air and areas with the potential for exceedance of the Pb standards. Further, as described below, the information available regarding the transient sources mentioned by the commenters does not indicate the potential for such transient sources to result in exceedances of the NAAQS.

As to the comment on the significance of building demolition or Pb wheel weights in contributing to environmental Pb exposure pathways, the ISA and PA considered the very limited available data pertaining to these issues. With regard to building demolition, for which the data are in terms of loading of dust containing Pb on alleys and sidewalks immediately following an event, the ISA concludes that the limited data "suggest that building demolition may be a short-term source of Pb in the environment," and that "it is unclear if demolition is related to long-term Pb persistence in the environment" (ISA, p. 2-21).[67] Accordingly, we do not interpret the limited available information, which does not include measurements of air Pb concentrations, to indicate a potential for such occasional activities as demolition of buildings containing leaded paint to result in air Pb concentrations near or in exceedance of the NAAQS. [68] With regard to the comment on lead wheel weights, we note that the commenter states they are unaware of studies that have assessed the impact of Pb wheel weights on childhood blood Pb

[67] Characterization of this activity by the study published subsequent to the ISA that was cited by the CHPAC (Jacobs et al., 2013) is consistent with findings from the limited number of studies included in the ISA (ISA, p. 2-21).

[68] We note that airborne dust release from demolition of large buildings in some areas may be regulated under various state and/or local programs (e.g., demolition activities in some particulate matter non-attainment or maintenance areas may be subject to specific state implementation plan requirements on airborne dust releases).

levels, as are we. The ISA examined the very limited data on potential contribution of Pb wheel weights to Pb near roadways; these data yield widely varying and uncertain estimates of associated Pb releases (ISA, section 2.2.2.6). Contrary to the commenter's assertion that the PA overlooks these potential Pb exposure pathways, the assessment and consideration of policy-relevant information in the PA[69] reflects these ISA findings based on consideration of the current information for these potential transient pathways. Specifically, the current information does not provide support for specific estimates of exposures associated with these pathways. Further, data for monitoring sites near roads find Pb concentrations well below the NAAQS (e.g., ISA, Figure 2-20). Thus, we conclude that the current information does not provide support for changes to the current Pb monitoring regulations with regard to roadways or occasional activities such as building demolition.

4. Administrator's Conclusions

Having carefully considered the public comments, as discussed above, the Administrator believes that the fundamental scientific conclusions on the effects of Pb in ambient air reached in the ISA and PA, and summarized in sections II.B and II.C of the proposal, remain valid. Additionally, the Administrator believes the judgments she reached in the proposal (section II.E.4) with regard to consideration of the evidence and quantitative exposure/risk information remain appropriate. Thus, as described below, the Administrator concludes that the current

[69] Consistent with the strength and specificity of information described in the ISA, the PA recognizes the loss of Pb wheel weights as an additional source of Pb emissions and notes the potential for previously deposited Pb to be resuspended into the air, without providing detailed consideration (PA, sections 2.1.2.2 and 2.1.2.4). Further, the input for air-to-blood ratio in the air-related IQ loss evidence-based framework, which the Administrator has used as a guide in her consideration of the adequacy of the current standard, does not restrict sources of Pb from consideration. Thus, such ratios, which are drawn from empirical studies, would be expected to reflect all sources contributing to children's blood Pb, including the transient sources identified by commenters to the extent they provide contributions (ISA, section 3.5; PA, section 3.1; 80 FR 298-300, January 5, 2015; 73 FR 66973-66975,67004, November 12, 2008).

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 primary Pb standard, the Administrator has carefully considered the current policy-relevant evidence and conclusions contained in the ISA; the evaluation of this evidence and the exposure/risk information, rationale and conclusions presented in the PA; the advice and recommendations from the CASAC; and public comments. In the discussion below, the Administrator gives weight to the PA conclusions, with which the CASAC has concurred, as summarized in section II of the proposal, and takes note of key aspects of the rationale for those conclusions that contribute to her decision in this review.

As an initial matter, the Administrator recognizes 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 health protection provided by the primary standards in other NAAQS reviews. For the pollutants in the other reviews, the more limited focus solely on the inhalation pathways of exposure is a relatively simpler context. Further, as described in the PA and noted in section II.B.1 above, the influence of multimedia and historical exposure on the internal biomarkers in Pb epidemiological studies contrasts with the epidemiological studies considered for other NAAQS pollutants which focus on generally current concentrations of those pollutants in ambient air. While the use of an internal biomarker strengthens conclusions regarding Pb as the causal agent in associations observed in epidemiological studies, the persistence of Pb and the role of multimedia and historical exposures limit the conclusions that can be drawn regarding the particular exposure circumstances eliciting the reported effects. Thus, as we lack studies that can directly assess 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 information base, both with its strong, long-established evidence of the health effects of Pb in young children, and the associated limitations and uncertainties mentioned here, contributes to our conclusions regarding relationships between ambient air Pb conditions under the current standard and health effects.

The Administrator recognizes that in primary NAAQS reviews, 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 this review of the primary standard for Pb, she takes note of the 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. These aspects of the scientific evidence and analyses, and the associated uncertainties, collectively contribute to the Administrator's recognition that for Pb, as for other pollutants, the available health effects evidence and associated information generally reflect 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.

With regard to the current evidence, as summarized in the PA and discussed in detail in

the ISA, the Administrator takes note of the well-established body of evidence on the health effects of Pb, which has been augmented in some aspects since the last review and continues to support identification of neurocognitive effects in young children as the most sensitive endpoint associated with Pb exposure. For example, while the ISA continues to recognize cardiovascular effects in adults, in addition to neurodevelopmental effects in children, as being associated with the lowest blood Pb levels compared to other health effects (ISA, pp. xciii), the ISA also notes uncertainties regarding the timing, frequency, duration and level of Pb exposures contributing to the effects observed in adult epidemiologic studies and indicates that higher exposures in the past (rather than lower current exposures) may contribute to the development of health effects measured later in life (ISA, p. lxxxviii). Given the evidence-based identification of neurocognitive effects in young children as the most sensitive endpoint associated with Pb exposure, the Administrator has accordingly focused on nervous system effects in young children and particularly neurocognitive effects. In so doing, she finds that the evidence, while describing a broad array of health effects associated with Pb, continues to indicate that a standard that provides protection from neurocognitive effects in young children additionally provides protection from other health effects of Pb, such as those reported in adult populations.

The Administrator takes note of the PA finding that application of the air-related IQ loss evidence-based 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 young children's blood and risks of neurocognitive effects (for which IQ loss is used as an indicator). In so doing, as in the 2008 review, she notes that the framework, and the IQ loss estimates yielded by it for specific combinations of standard level, air-to-blood ratio and C-R function, does not provide an evidence- or risk-based bright line that indicates a single

appropriate level for the standard. Further, the Administrator recognizes uncertainties associated with IQ estimates produced by the framework, noting the PA conclusion that the uncertainties increase with estimates associated with successively lower standard levels. She additionally takes note of the PA finding (described in section II.E.1 of the proposal) 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. The Administrator further notes the concurrence from the CASAC on both of these points and the lack of recommendations in public comments for a change to either of these inputs to the evidence-based framework. Thus, she judges the evidence base and related air-related IQ loss framework to be an appropriate tool for informing her decision on the adequacy of the current standard.

In light of the continuum referenced above, the Administrator additionally recognizes in this review, as in the 2008 review, the role of judgment in reaching conclusions regarding Pb health effects that are important from a public health perspective. Most specifically, 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. With regard to making a public health policy judgment as to the appropriate protection against risk of air-related IQ loss and related effects, the Administrator believes, as did the Administrator at the time of the last review, 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. She recognizes, however, that in the case of setting NAAQS, she is required to make a judgment as to what degree of protection is requisite (neither more nor less than necessary) to protect public health with an adequate margin of safety. As described in the

proposal with regard to considering the public health significance of IQ loss estimates in young children, the Administrator gives weight to the comments of the 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 U.S. population be protected from such a magnitude of IQ loss (73 FR 67000, November 12, 2008). She additionally notes that the CASAC did not provide a different goal in the present review. 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), and no new information has been identified by public commenters.

With the objective identified by the CASAC in the 2008 review in mind, the Administrator recognizes, as was recognized at the time of the last review, that her judgment on the degree of protection against IQ impacts that should be afforded by the primary standard is particularly focused on consideration of impacts in the at-risk population and is not addressing a specific quantitative public health policy goal for air-related decrements in IQ that would be acceptable or unacceptable for the entire population of children in the U.S. As in the last review, the at-risk population to which she gives particular attention is the small subset of U.S. children living in close proximity to air Pb sources that contribute to elevated air Pb concentrations that equal the level of the standard). Accordingly, she is considering IQ impacts in this small subset of U.S. children that is expected to experience air-related Pb exposures at the high end of the national distribution of such exposures (as described in section II.E.4 of the proposal and summarized in section II.B.1 above), and not a projection of the average air-related IQ loss for

the entire U.S. population of children. The evidence-based framework estimates, with which there are associated uncertainties and limitations (as described in section II.A.1 above), relate to this small subset of children exposed at the level of the standard. Based on these considerations, the Administrator judges the conceptual evidence-based framework to continue to be appropriate for her consideration of the public health protection afforded by the current standard. Further, she concurs with the PA findings (summarized in section II.E.1 of the proposal and briefly outlined in II.B.1 above) that the current evidence, as considered within the conceptual and quantitative context of the evidence-based framework, and current air monitoring information indicate that the current standard would be expected to satisfy the public health policy goal recommended by the CASAC in the last Pb NAAQS review, from which it did not indicate a departure in the present review.

In the context of the Administrator's use of the framework as a tool to inform her decision on the adequacy of the current standard, the EPA additionally notes that the maximum, not to be exceeded, form of the standard, in conjunction with the rolling 3-month averaging time, is expected to result in the at-risk population of children being exposed below the level of the standard most of the time (73 FR 67005, November 12, 2008). In light of this and the uncertainty in the relationship between time period of ambient level, exposure, and occurrence of a health effect, the air-related IQ loss considered for the current standard in applying the framework should not be interpreted to mean that a specific level of air-related IQ loss will occur in fact in areas where the standard is just met or that such a loss has been determined as acceptable if it were to occur. Instead, judgment regarding such an air-related IQ loss is one of the judgments that need to be made in using the evidence-based framework to provide useful guidance in the context of public health policy judgment on the degree of protection from risk to public health

that is sufficient but not more than necessary, taking into consideration the patterns of air quality that would likely occur upon just meeting the standard and uncertainties in relating those patterns to exposures and effects.

In drawing conclusions regarding adequacy of the current standard based on considering application of the evidence-based framework, 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, as described in section II.B.4 of the proposal. 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 additionally 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). The Administrator also 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). Thus, consistent with the conceptual continuum referenced above, the Administrator recognizes the increasing uncertainty with regard to likelihood of response and magnitude of the estimates at levels extending below the current standard.

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 air-related IQ loss evidence-based 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 evidence- and exposure/risk-based considerations and with consideration of advice from CASAC and public comment, the Administrator concludes that the current standard provides protection for young children from neurocognitive impacts, including IQ loss, that is consistent with advice from CASAC regarding IQ loss of public health significance. 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 its 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 retain the current standard without revision. The Administrator bases these conclusions on consideration of the health effects evidence, including consideration of this evidence in the context of the air-related IQ loss evidence-based 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 (as noted in section II.A.1 above and the 2008 decision preamble to the final rule, 73 FR 67007, November 12, 2008). 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 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, in particular, 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 has considered conclusions drawn in the ISA and PA with regard to interpretation of the information concerning the broader array of health effects of Pb beyond those on the nervous system of young children. Based on the body of evidence in support of identification of neurocognitive effects in young children as the most sensitive endpoint associated with Pb exposure, as noted previously in this section and briefly summarized in section II.A.2 above, she judges that a standard providing protection from such effects additionally provides adequate protection against the risk of other health effects and she further concludes that consideration of the more limited and less certain information concerning Pb exposures associated with such other effects does not lead her to identify a need for any greater protection.

Further, the Administrator's 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 reaching this conclusion, she additionally takes note of the PA conclusion, with which the 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, Consensus Response to Charge Questions, 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 concludes that the current standard should be retained, without revision.

*C. Decision on the Primary Standard*

For the reasons discussed above, and taking into account information and assessments presented in the ISA and PA, the advice from CASAC, and consideration of public comments, the Administrator concludes that the current primary standard for Pb is requisite to protect public health with an adequate margin of safety, including the health of at-risk populations, and is retaining the standard without revision.

**III. Rationale for Decision on the Secondary Standard**

This section presents the rationale for the Administrator's 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, on welfare effects associated with Pb and pertaining to the presence of Pb in the ambient air. This decision also takes into account (1) the 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) the CASAC advice and recommendations, as reflected in discussions of drafts of the ISA and PA at public meetings, in separate written comments, and in the CASAC's letters to the Administrator; (3) public comments received during the development of these documents, either in connection with

CASAC meetings or separately; and (4) public comments on the proposal.

Section III.A provides background on the general approach for the review of the secondary NAAQS for Pb and brief summaries of key aspects of the current body of evidence on welfare effects associated with Pb exposures and the exposure/risk information considered in this review. Section III.B summarizes the basis for the proposed decision and advice from the CASAC, addresses public comments and presents the conclusions the Administrator has drawn from a full consideration of the information. Section III.C summarizes the Administrator's decision on the secondary standard.

*A. Introduction*

As provided in the Act, the secondary standard is to "specify a level of air quality the attainment and maintenance of which 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" (CAA, section 109(b)(2)). The secondary standard is not meant to protect against all known or anticipated Pb-related effects, but rather those that are judged to be adverse to the public welfare, and a bright-line determination of adversity is not required in judging what is requisite (78 FR 3212, January 15, 2013; 80 FR 65376, October 26, 2015). Thus, the level of protection from known or anticipated adverse effects to public welfare that is requisite for the secondary standard is a public welfare policy judgment to be made by the Administrator. In exercising that judgment, the Administrator seeks to establish standards that are neither more nor less stringent than necessary for this purpose. This section presents the rationale for the Administrator's decision to retain the existing secondary NAAQS for Pb, without revision. The Administrator's decision draws upon scientific information and analyses about welfare effects, exposure and risks, as well as judgments about 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.

In the last review, completed in 2008, the current secondary standard for Pb was revised substantially, consistent with the revision to the primary standard (73 FR 66964, November 12, 2008). 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. At that time, 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, the EPA also considered the evidence of Pb effects on organisms with regard to implications for ecosystem effects. Taking into account the available evidence and information on 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 EPA revised the secondary standard substantially, consistent with revisions made to the primary standard (73 FR 67011, November 12, 2008).

Building on the approach and findings in the last review, this current review of the secondary standard considers the currently available scientific and technical information in the context of key policy-relevant questions. This review focuses on the consideration of the extent to which the body of scientific evidence now available calls into question the adequacy of the current standard. In considering the scientific and technical information, 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. Thus, we have taken into account both evidence-based and risk-based considerations pertaining to the series of policy-relevant questions presented in the PA. These questions generally address the extent to which we are able to characterize effects and the likelihood of adverse effects in the environment under the current standard. Our approach to considering this information recognizes that the available welfare effects evidence generally reflects laboratory-based evidence of toxicological effects on specific organisms exposed to concentrations of Pb (ISA, section 6.5). Additionally, it is widely recognized 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 (ISA, Section 6.5).

1. Overview of Welfare Effects Information

Welfare effects 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 wellbeing" (CAA, section 302(h)). In this section, we provide an overview of the key aspects of the current evidence of Pb-related welfare effects that is assessed in the ISA and the 2006 CD, drawing from the summary of policy-relevant aspects in the PA (PA, section 5.1) and section III.B of the proposed rulemaking (80 FR 314-317, January 5, 2015).

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[70] that causal[71] or likely causal[72] relationships exist at the individual and population level 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). With regard to

[70] 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).

[71] 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).

[72] 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).

saltwater ecosystems, the ISA concludes that the current evidence is inadequate to make causality determinations for most effects, while finding the evidence to be suggestive of a linkage between Pb and effects on reproduction and development in marine invertebrates (ISA, Table 1-3, sections 6.3.12 and 6.4.21). 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 under particular, small-scale conditions, up to the ecosystem level of biological organization, the ISA also determines that a causal relationship is also likely at higher levels of biological organization between Pb exposures and community and ecosystem-level effects in freshwater and terrestrial systems (ISA, section 1.7.3.7).

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 (ISA, section 6.4). 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 (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).

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, 6.4.4 and 6.4.14). It is the amount of Pb that can interact within the organism that can lead 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). On the whole, the current evidence, including that newly available in this review, supports previous conclusions regarding environmental conditions affecting bioavailability and the associated potential for adverse effects of Pb on organisms and ecosystems (ISA, section 6.3.3). 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. As summarized in the ISA, "in natural environments, modifying factors affect Pb bioavailability and toxicity and there are considerable uncertainties associated with generalizing effects observed in controlled studies to effects at higher levels of biological organization" and "[f]urthermore, available studies on community and ecosystem-level effects are usually from contaminated areas where Pb concentrations are much higher than typically encountered in the environment" (ISA, p. xcvi).

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 beyond what was understood in the last review. 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).

2. Overview 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). 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). The CASAC Pb Review Panel generally concurred with the conclusion that a new REA was not warranted for the secondary standard in this review (Frey, 2011b). Accordingly, the exposure/risk information considered in this review is drawn primarily from the 2006 REA as summarized in the 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). Both a national-scale screen and a case study approach were used to evaluate the potential for ecological impacts that might be associated with atmospheric deposition of Pb (2006 REA, section 7.1.2). 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 quality conditions are presented in the 2006 REA. This information, which is outlined below, is summarized more fully in section 5.2 of the PA and section III.C of the proposal for this review (80 FR 317-319, January 5, 2015).

In interpreting the results from the 2006 REA, the PA considers the availability of new 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, such new evidence is limited, and the key uncertainties identified in the last review remain today. For example, with regard to new evidence of Pb effects at lower concentrations, it is necessary to consider that the evidence of adversity in natural systems due specifically to Pb 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, as noted in section III.A.1 above. Modeling of Pb-related exposure and risk to ecological receptors is subject to a wide array of sources of both variability and uncertainty resulting in differences in Pb bioavailability as well as exposure (USEPA, 2005b). Additionally, there are also 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 (PA, section 1.3.2). For example, media in ecosystems across the U.S. are still recovering from the past period of greater atmospheric emissions and deposition, as well as from Pb derived from nonair sources (PA, section 1.3.2).

As summarized in the PA and proposal, we have considered what the risk information from the 2006 REA analyses indicates regarding the potential for adverse welfare effects to result from levels of air-related Pb that would meet the now-current standard. The circumstances assessed in all but one of the case study locations, however, likely include a history of ambient air Pb concentrations that exceeded the NAAQS. Consequently, these analyses are not considered informative for predicting effects at the far lower concentrations associated with the current NAAQS. The nationwide surface water screen was likewise not particularly informative because potential confounding by both nonair inputs and resuspension of Pb related to historic sources was not easily accounted for. The remaining case study was a site remote from Pb sources for which atmospheric deposition was expected to be the primary contributor to media Pb concentrations without obvious confounding inputs. This case study, based on a summary review of published findings for the study site, concluded that atmospheric Pb inputs do not directly affect stream Pb levels because deposited Pb is almost entirely retained in the soil profile, with the soil serving as a Pb sink, appreciably reducing pore water Pb concentrations as it moves through the soil layers to streams. As a result, this case study (and the publications on which it was based) concluded that the contribution of dissolved Pb from soils to streams was insignificant (2006 REA, Appendix E). Additionally, we note that the 2006 CD, in considering the findings for this site and other terrestrial sites with Pb burdens derived primarily from long-range atmospheric transport, 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 PA and proposal concluded that 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 (PA, p. 6-10; 80 FR 319, January 5, 2015).

*C. Conclusions on the Secondary Standard*

1. Basis for the Proposed Decision

The basis for the proposed decision, which is described in section III.D of the proposal, is very briefly summarized here. In considering the welfare effects evidence and risk-based information with respect to the adequacy of the current secondary standard, the Administrator considered 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, she considered 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 informed the Administrator's proposed decision to retain the current standard.

With regard to the evidence, the proposal noted 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 there are newly available field studies in this review, "the connection between air concentration and ecosystem exposure and associated potential for welfare effects continues to be poorly characterized for Pb" (ISA, section 6.4). 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.

With regard to the currently available risk and exposure information, which continues to be sufficient to conclude that the 1978 standard was not providing adequate protection to ecosystems, the proposal concluded that, when considered with regard to air-related ecosystem exposures likely to occur with air Pb levels that just meet the now-current standard, this current information also does not provide evidence of adverse effects under the current standard. Accordingly, in consideration of the risk information in combination with the current evidence and the associated data gaps and uncertainties, the Administrator proposed that the current standards be retained, without revision.

2. CASAC Advice in this Review

In its 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 … Secondary Pb NAAQS" (Frey, 2013b, p. 1). It additionally stated that "[g]iven the existing scientific data, the CASAC concurs with retaining the current secondary standard

without revision" (Frey, 2013b, p. 2). The CASAC additionally noted areas for additional research to address data gaps and uncertainties (Frey, 2013b, p. 2).

3. Comments on the Proposed Decision

All of the public comments on the proposed decision to retain the current secondary standard, without revision, indicated support. These commenters include the NACAA, as well as both of the state agencies and nearly all of the industry organizations that submitted comments. Only a small subset of this group provided rationales for their concurrence with EPA's proposed decision. These commenters emphasized limitations and uncertainties in the welfare effects evidence, including particularly those with regard to relationships between ambient air Pb concentrations, levels of deposition, ecosystem exposures, and adverse public welfare effects. One commenter also noted the CASAC's concurrence with the EPA conclusion that the current evidence does not support revision to the standard, and that information newly available in this review does not substantially improve our understanding in the identified areas of uncertainty or that would indicate that the current standard is inadequate. The EPA generally agrees with these commenters and with the CASAC regarding the adequacy of the current secondary standard and the lack of support for revision of the standard.

4. Administrator's Conclusions

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 concludes 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. In reaching her decision, 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 decision.

As she did in reaching her proposed decision, the Administrator notes 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, there is a 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 relating effects evidence from laboratory studies to the natural environment and linking those effects to ambient air Pb concentrations. Further, as the proposal and the PA note, 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, 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 and assessed 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 previously available and supported the decision for revision in the last review (PA, section 6.2.1).

With respect to exposure/risk-based considerations identified in the PA, the Administrator notes 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. The Administrator additionally takes note 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 summary, based on the considerations summarized above, the Administrator judges that the information available in this review of the Pb secondary standard, including the currently available welfare effects evidence and exposure/risk information, does 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." Thus, the Administrator concludes that the current standard is requisite and should be retained.

*C. Decision on the Secondary Standard*

For the reasons discussed above, and taking into account information and assessments

presented in the ISA and PA, the advice from CASAC, and consideration of public comments, the Administrator concludes that the current secondary standard for Pb is requisite to protect public welfare from known or anticipated adverse effects and is retaining the standard without revision.

## 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 (PRA)*

This action does not impose an information collection burden under the PRA. There are no information collection requirements directly associated with revisions to a NAAQS under section 109 of the CAA and this action does not make any revisions to the NAAQS.

### *C. Regulatory Flexibility Act (RFA)*

I certify that this action will not have a significant economic impact on a substantial number of small entities under the RFA. This action will not impose any requirements on small entities. Rather, this action retains, without revision, existing national standards for allowable concentrations of Pb 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 (UMRA)*

This action does not contain any unfunded mandate as described in the UMRA, 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. It does not have a substantial direct effect on one or more Indian tribes. This action does not change existing regulations; it retains the current NAAQS for Pb, without revision. The NAAQS protect public health, including the health of at-risk or sensitive groups, with an adequate margin of safety and protect public welfare from known or anticipated adverse effects. 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. We note, however, that the primary standard retained with this action provides protection for children and other at-risk populations against an array of adverse health effects, most notably including nervous system effects in children. The health effects evidence and risk assessment information for this action, which focuses on children, is summarized in sections II.A.2, II.A.3 and II.A.4, and described in the ISA and PA, copies of which are in the public docket for this action.

*H. Executive Order 13211: Actions Concerning Regulations 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 action 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 does not have disproportionately high and adverse human health or environmental effects on minority populations, low-income populations and/or indigenous peoples as specified in Executive Order 12898 (59 FR 7629, February 16, 1994). The action described in this document is to retain, without revision, the existing NAAQS for Pb.

The NAAQS decisions are based on an explicit and comprehensive assessment of the current scientific evidence and associated exposure/risk analyses. More specifically, the EPA expressly considers the available information regarding health effects among at-risk populations, including that available for low-income populations and minority populations, in decisions on the primary (health-based) NAAQS. Where low-income populations or minority populations are among the at-risk populations, the decision on the standard is based on providing protection for these and other at-risk populations and lifestages. Where such populations are not identified as at-risk populations, NAAQS that are established to provide protection to the at-risk populations would also be expected to provide protection to all other populations, including low-income populations and minority populations.

As discussed in sections II.A.2.d and II.B above, and in sections II.A and II.B of the proposal, the EPA expressly considered the available information regarding health effects among

at-risk populations in reaching the decision that the existing primary (health-based) standard for Pb is requisite. The ISA and PA for this review, which include identification of populations at risk from Pb health effects, are available in the docket, EPA-HQ-OAR-2010-0108. Based on consideration of this information and the full evidence base, quantitative exposure/risk analyses, advice from the CASAC and consideration of public comments, the Administrator concludes that the existing NAAQS for Pb protect public health, including the health of at-risk or sensitive groups, with an adequate margin of safety and protect public welfare from known or anticipated adverse effects (as discussed in sections II.B.4 and III.B.4 above).

*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).

*L. Congressional Review Act*

The EPA will submit a rule report to each House of the Congress and to the Comptroller General of the U.S. This action is not a "major rule" as defined by 5 U.S.C. 804(2).

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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,
Administrator.

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