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U.S. Environmental Protection Agency
Air and Radiation Docket and Information Center
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Re: Comments on EPA's December 2007 Advance Notice of Proposed Rulemaking for National Ambient Air Quality Standards for Lead, 72 Fed. Reg. 71,488 (Dec. 17, 2007) (EPA-HQ-OAR-2006-0735) (Exhibits A-U submitted via regulations.gov at EPA-HQ-OAR-2006-0735, tracking numbers 8039b772 and 8039b76c)

We submit these comments on behalf of the Natural Resources Defense Council; Airaware of Southern Indiana; the American Association on Intellectual and Developmental Disabilities; the American Lung Association; the American Public Health Association; the American Thoracic Society; the Clean Air Council; Clean Air Watch; Environmental Defense; the Galveston Houston Association for Smog Prevention; the Greater Englewood Community and Family Task Force/CSX Citizen Oversight Panel; the Kentucky Resources Council; Learning Disabilities Association of America; the National Center for Vermiculite and Asbestos-Related Cancers at the Karmanos Cancer Institute; Physicians for Social Responsibility; the Respiratory Health Association of Metropolitan Chicago; the Science and Environmental Health Network; the Sierra Club; the Sierra Club Allegheny Group; the Trust for Lead Poisoning Prevention; Utah Moms for Clean Air; Utah Physicians for a Healthy Environment; Valley Watch, Inc.; the Wasatch Clean Air Coalition; and environmental health scientists, and advocates. We strongly disagree with the EPA's suggestion in its December 17, 2007 Advance Notice of Public Rulemaking ("ANPR") that EPA may consider revoking the primary and secondary National Ambient Air Quality Standards ("NAAQS") for lead or

delisting lead as a criteria pollutant. *See, e.g.*, ANPR at 131.¹ The Clean Air Act (“Act”) does not authorize EPA to revoke the NAAQS, and in fact requires EPA to adopt far more stringent controls on ambient lead. To assure an adequate margin of safety for all segments of the U.S. population, EPA must revise the NAAQS to a level or levels that account for the established science on lead’s threats to public health and welfare, which shows health impacts at levels well below the current NAAQS. We previously addressed these issues in our February 5, 2007 comments on EPA’s December 2006 Draft Staff Paper on the lead NAAQS (“Draft Staff Paper”), incorporated herein by reference. *See* EPA-HQ-OAR-2006-0735-0461. The following comments provide additional scientific evidence and analysis in support of a significantly strengthened NAAQS, and address a number of related issues raised in the ANPR, the November 1, 2007 Final Staff Paper for lead (“Staff Paper”), and the final Human Exposure and Health Risk Assessment for lead (“Risk Assessment”).

¹ Except where EPA’s December 17, 2007 Federal Register notice is referenced, citations to the ANPR in this comment letter apply the pagination in the December 4, 2007 advance copy of the ANPR posted on EPA’s website at http://www.epa.gov/ttn/naaqs/standards/pb/data/20071204_pb_anpr.pdf.

I. The Clean Air Act Does Not Authorize EPA to Delist Lead as a Criteria Pollutant or Eliminate the Primary or Secondary Lead NAAQS.²

The ANPR inappropriately requests comment on whether lead may be delisted as a criteria pollutant, whether the lead NAAQS may be revoked, and whether other federal and state lead controls programs “are sufficient to provide appropriate public health protection in lieu of a lead NAAQS.” ANPR at 131. The plain language of the Act does not allow for a revocation of the lead NAAQS and does not leave room for EPA to consider that question. The other lead programs referenced in the ANPR do not address the legislative concerns embodied in the NAAQS requirement as they do not protect public health with an adequate margin of safety as required by the Act. As both the Clean Air Scientific Advisory Committee (“CASAC”) and EPA’s own staff have already concluded, EPA can satisfy the Clean Air Act’s mandate to protect public health with an adequate margin of safety only by retaining and significantly strengthening the NAAQS. Staff Paper at 5-44; ANPR at 114.

A. The Plain Language of the Act Precludes Revocation of the Lead NAAQS or Delisting of Lead as a Criteria Pollutant.

The ANPR improperly suggests that “revocation of the [lead NAAQS]” may be “an appropriate option for [EPA] to consider” in connection with its evaluation of the appropriate NAAQS. ANPR at 111. As NRDC noted in its February 2007 comments on the Draft Staff Paper, any such revocation would contravene the Act’s plain language and the established science on the health effects of lead. EPA cannot legally consider this option.

Section 108(a) of the Clean Air Act (“Act”), 42 U.S.C. § 7401 *et seq.*, requires EPA to establish and maintain a list of all “criteria” air pollutants (1) whose emissions “may reasonably be anticipated to endanger public health or welfare” and (2) whose presence “in the ambient air results from numerous or diverse mobile or stationary sources.” 42 U.S.C. §§ 7408(a)(1)(A)-(B).³ Section

² The primary and secondary national ambient air quality standards for lead have been set at 1.5 micrograms per cubic meter of air (1.5 µg/m³), measured on a maximum quarterly average basis, since their October 1978 promulgation. *See generally National Primary and Secondary Ambient Air Quality Standards for Lead*, 43 Fed. Reg. 46246 (Oct. 5, 1978).

³ Section 108(a) further requires EPA to issue, for each listed pollutant, “air quality criteria” which “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

109 of the Act requires EPA to establish primary and secondary national ambient air quality standards, or NAAQS, for each pollutant listed under Section 108. *See generally* 42 U.S.C. §§ 7409(a)-(b), 7408(b). These provisions, as our federal courts have recognized for decades, oblige EPA to list lead as a criteria pollutant and maintain NAAQS for it. *See generally Natural Resources Defense Council v. Train*, 545 F.2d 320 (2nd Cir. 1976); *see also Lead Industries Ass’n. v. EPA*, 647 F.2d 1130, 1136-37 (D.C. Cir. 1980) (discussing *Train*). The Act does not give EPA any discretion to refuse to list, under Section 108, lead or any other air pollutant that “may reasonably be anticipated to endanger public health and welfare” and whose presence “in the ambient air results from numerous mobile or stationary sources.” *Train*, 545 F.2d at 324-25 (D.C. Cir. 1976) (describing listing as “mandatory” once “the criteria of [Sections 108(a)(1)(A) and (B)] are met.”). Similarly, EPA cannot refuse to promulgate NAAQS for lead and other pollutants with those characteristics. *Id.* at 322-23, 325 (observing that the Section 109 NAAQS provisions are “automatically invoked” by a Section 108 listing); *see also Whitman v. American Trucking Ass’ns*, 531 U.S. 457, 462-63 (2001).

The ANPR’s suggestion that it may no longer be “appropriate” for EPA to regulate lead under Sections 108 and 109, ANPR at 111, contravenes the plain language of the Act. First, as we discuss at Part II below and as EPA’s scientific staff and CASAC recognize, lead remains a significant public health hazard. *See, e.g.*, Staff Paper at 5-12 to 5-13. There is substantial evidence that lead’s presence in the human environment at *any* level constitutes a threat to general public health and a significant threat to sensitive sub-populations and highly exposed communities. *Id.*; *see infra* Part II. As such, it is beyond dispute that lead pollution “may reasonably be anticipated to endanger public health or welfare.” 42 U.S.C. § 7408(a)(1)(A). Second, lead’s presence “in the ambient air” still “results from numerous or diverse mobile or stationary sources.” 42 U.S.C. § 7408(a)(1)(B). Although emissions of lead from passenger vehicles have decreased sharply with the phaseout of leaded gasoline, there remain many other significant stationary and mobile sources of lead emissions. By the time Congress

expected from the presence of such pollutant in the ambient air, in varying quantities.” 42 U.S.C. § 7408(a)(2). These criteria “shall include,” wherever practicable, “information on—”

- (A) those variable factors (including atmospheric conditions) which of themselves or in combination with other factors may alter the effects on public health or welfare of such air pollutant;
- (B) the types of air pollutants which, when present in the atmosphere, may interact with such pollutant to produce an adverse effect on public health or welfare; and
- (C) any known or anticipated adverse effects on welfare.

Id.

adopted its comprehensive 1990 amendments to the Clean Air Act, most of the reductions in airborne lead levels referenced by EPA had already been achieved.⁴ Congress nonetheless chose to both *preserve* the NAAQS mandates at Sections 108 and 109, *and* to list lead as a hazardous air pollutant under the newly promulgated Section 112 of the Act, *see* 42 U.S.C. § 7412(b). EPA's own data shows that there remain over 13,000 commercial, industrial and institutional lead sources in the United States. Staff Paper at 2-12. In addition, EPA's partial inventory of sources emitting over 5 tons of lead per year includes 37 separate categories of mobile and stationary sources. Staff Paper at 2-7; *see also generally id.* at 2-5 to 2-15.

Because lead clearly satisfies the standards for criteria pollutants under Section 108 of the Act, and Section 109 of the Act requires EPA to maintain NAAQS for all criteria pollutants, EPA should not, and legally cannot, include delisting lead as a criteria pollutant or revoking the lead NAAQS as policy "options" in its current review. In fact, as discussed below in Part II, Sections 108 and 109 oblige EPA to respond to the evidence on lead's dangers to public health by promulgating significantly more protective NAAQS.

B. EPA Cannot Rely on Other Programs Addressing Lead to Avoid Its Mandatory Duty to Maintain and Strengthen the Lead NAAQS.

EPA may not rely on the existence of other lead control programs to revoke the lead NAAQS. These other programs do not address the independent, stringent Congressional mandates embodied in Sections 108 and 109. Furthermore, as the discussion of health impacts at Part II illustrates, other lead programs, even when combined with the existing NAAQS, have simply not provided the level of protection from ambient lead contemplated by Section 109 of the Act, which requires EPA to protect the public health and welfare with an adequate margin of safety. The other programs, by themselves, cannot assure the requisite margin of safety. Therefore, neither the programs implemented pursuant to other sections of the Act nor programs under other regulatory schemes can replace the lead NAAQS.

EPA's regulation of lead under other sections of the Clean Air Act, such as the Section 112 Hazardous Air Pollutant ("HAP") and Maximum Achievable

⁴ *See* Agency for Toxic Substance and Disease Registry, U.S. Department of Health and Human Services, Toxicological Profile for Lead 2-3 (August 2007), *available at*: <http://www.atsdr.cdc.gov/toxprofiles/tp13.html> (last visited Jan. 14, 2008) (Attached as Exhibit A).

Control Technology (“MACT”) provisions, neither alters nor satisfies its mandates under Sections 108 and 109. When Congress added Section 112 to the Clean Air Act in 1990, it made clear that “[n]o emission standard or other requirement promulgated under this section shall be interpreted, construed or applied to diminish or replace the requirements of a more stringent emission limitation or other applicable requirement established pursuant to . . . any other authority of this chapter.” 42 U.S.C. § 7412(d)(7). In addition, the courts have repeatedly held that each section of a statute is presumed to have independent meaning and effect. *See, e.g., Mackey v. Lanier Collection Agency & Svc., Inc.*, 486 U.S. 825, 837 (1988) (“[W]e are hesitant to adopt an interpretation of a congressional enactment which renders superfluous another provision of that same law.”); *Davis County Solid Waste Mgmt. v. EPA*, 101 F.3d 1395, 1404 (D.C. Cir. 1996) (noting, in Clean Air Act case, the “well-established maxim of statutory construction that courts should avoid interpretations that render a statutory provision superfluous”).

The ANPR’s implication that EPA may rely on its regulation of lead under Section 112 in lieu of maintaining adequately protective NAAQS, *see* ANPR at 111, 113, is contrary to law. Section 112 does not provide for lead regulations as comprehensive and stringent as those mandated by Sections 108 and 109. As the Staff Paper acknowledges, Section 112 focuses on regulation of emissions from stationary sources, and thus does not provide for regulation of the mobile sources EPA must also consider under the NAAQS provisions. Staff Paper at 5-13; *Compare* 42 U.S.C. § 7412(d)(2) *with* 42 U.S.C. § 7408(a)(1)(B). Moreover, Section 112 provides for consideration of economic costs. 42 U.S.C. § 7412(d)(2) (describing “standards” which “take[] into consideration the cost of achieving” reductions in emissions). EPA may not consider economic costs in setting NAAQS. ANPR at 11; *Whitman*, 531 U.S. at 464-71; *Lead Industries*, 647 F.2d at 1148-50. Thus, and as EPA’s Staff Paper recognizes, Section 112 cannot replace the Section 108 and 109 obligations. *See* Staff Paper at 5-13.

EPA also cannot rely on lead regulatory schemes outside the Clean Air Act to avoid its NAAQS mandates. *See, e.g., Agri Processor Co. v. NLRB*, ___ F.3d ___, 2008 WL 53879 (D.C. Cir. Jan. 4, 2008), at *2 (“where two statutes are capable of co-existence, it is the duty of the courts, absent a clearly expressed congressional intention to the contrary, to regard each as effective.” (citation omitted)); *Headwaters, Inc. v. Talent Irrigation Dist.*, 243 F.3d 526, 530-31 (9th Cir. 2001) (“we must interpret the two statutes ‘to give effect to each if we can do so while preserving their sense and purpose. When two statutes are capable of co-existence, it is the duty of the courts . . . to regard each as effective.’” (citation omitted)). The ANPR points to regulatory programs designed to address disposal of lead-based paint waste; to specify maximum levels of lead permitted in public

water systems under the Safe Drinking Water Act (“SDWA”); to reduce hazards from lead-based paint and residential lead dust under the Toxic Substances Control Act (“TSCA”); and to implement the hazardous and solid waste requirements of the Comprehensive Environmental Response, Compensation and Liability Act (“CERCLA”) and the Resource Recovery and Conservation Act (“RCRA”). ANPR at 14-18. EPA also references nonregulatory and research programs directed at reducing lead exposure. *Id.* at 18-20. None of these programs address the issue addressed by the lead NAAQS – lead air pollution from all mobile and stationary sources, extending beyond paint, water, and hazardous waste sites. As the ANPR acknowledges, other than the MACT provisions, these efforts are focused “primarily” on *nonair* sources of lead pollution. *Id.* at 13, 14. Moreover, some of the programs rely entirely on voluntary compliance measures that the ANPR acknowledges are likely to be sensitive to the presence of underlying regulatory mandates (such as the NAAQS). *See id.* at 16. EPA may not rely on programs that regulate lead in other media to revoke the NAAQS.

Finally, these other programs do not satisfy the Act’s mandate to protect public health with an adequate margin of safety, as discussed in greater detail in Part II. EPA’s scientific staff, CASAC, and the Centers for Disease Control and Prevention (“CDC”) have all concluded that, even with the current NAAQS in place, lead continues to threaten public health and welfare, especially for sensitive subpopulations. Staff Paper at 5-44; ANPR at 114. The CDC estimates that 310,000 U.S. children aged 1-5 still have blood lead levels over 10 µg/dL.⁵ Moreover, CDC has formally recognized that *no level of lead in a child’s blood can be specified as safe.*⁶ CASAC and EPA’s scientific staff have concluded, consistent with CDC’s findings, that the currently available information does not support delisting lead as a criteria pollutant or revoking the current lead NAAQS. Staff Paper at 5-44; ANPR at 114. Specifically, CASAC and EPA’s scientific staff determined that the scientific information accumulated since the promulgation of the current NAAQS demonstrates that existing controls on lead have not decreased blood lead levels below levels of concern sufficient to provide an adequate margin of safety; that lead-emitting activities have not been reduced to a point such that emissions will remain low even without NAAQS controls; and that other regulatory programs, including the Section 112 programs, would not sufficiently address lead’s health threats if lead were delisted as a criteria air

⁵ See CDC, *Blood Lead Levels – 1999-2002* (May 2005), available at <http://www.cdc.gov/MMWR/preview/mmwrhtml/mm5420a5.htm>.

⁶ ANPR at 29; see *Preventing Lead Poisoning in Young Children: A Statement by the Centers for Disease Control and Prevention*, at 1-6 (emphasis added) (U.S. Department of Health and Human Services, Public Health Service, August 2005).

pollutant. Staff Paper at 5-12 to 5-13; ANPR at 114. The Children's Health Protection Advisory Committee ("CHPAC") agrees with these conclusions.⁷

Because the lead NAAQS provision has different requirements and serves different purposes than the other federal and state regulatory programs aimed at lead pollution, and because other programs do not provide the level of protection mandated by the NAAQS provision of the Act, EPA may not rely on these other programs in lieu of a lead NAAQS which ensures adequate protection for public health.

II. Because There Is No Established "Safe" Level of Lead Exposure, In Order to Establish an "Adequate Margin of Safety," EPA Must Adopt Significantly More Stringent NAAQS.

As noted in our previous comments, and discussed further below, there is broad scientific consensus that the current lead NAAQS, now thirty years old, does not sufficiently protect human health and welfare. Therefore, the Act obliges EPA to review and tighten the existing standards. *Whitman*, 531 U.S. 462-63 (citing 42 U.S.C. § 7409(d)(1)) ("Once a NAAQS has been promulgated, [EPA] must review the standard . . . and make 'such revisions as may be appropriate.'").

Under Section 109 of the Act, EPA must set the primary NAAQS at a level which, "based on [the Section 108] criteria and allowing an adequate margin of safety, [is] requisite to protect the public health." 42 U.S.C. § 7409(b)(1). The secondary NAAQS must be set at a level which, "based on [the Section 108] criteria, is requisite to protect the public welfare⁸ from any known or anticipated adverse effects associated with the presence of such air pollutant in the ambient air." 42 U.S.C. § 7409(b)(2). The federal courts have repeatedly recognized that the NAAQS provisions, like the Act as a whole, require EPA to use a "preventative," "precautionary" and "protective" approach in setting air quality

⁷ See Letter from Dr. Melanie A. Marty, Chair, Children's Health Protection Advisory Committee, to Administrator Stephen L. Johnson, Re: OAQPS Final Staff Paper/Policy Assessment of Scientific and Technical Information and Advanced Notice of Proposed Rulemaking for the Lead NAAQS 2. January 9, 2008 ("CHPAC Letter") (Attached as Exhibit B).

⁸ The "public welfare" effects for which NAAQS must account include effects on "soils, water, crops, vegetation, manmade materials, animals, wildlife, weather, visibility, and climate," "property," "transportation," "economic values," and "personal comfort and well-being," "whether caused by transformation, conversion, or combination with other air pollutants." 42 U.S.C. § 7602(h); see also *Lead Industries*, 647 F.2d at 1137 (citing definition of "public welfare").

standards, rather than limit its purview to what it believes are “clearly harmful” effects. *American Lung Ass’n. v. EPA*, 134 F.3d 388, 389 (D.C. Cir. 1999); *American Trucking Ass’ns. v. Whitman*, 283 F.3d 355, 369-70 (D.C. Cir. 2002); *Lead Industries*, 647 F.2d at 1153-55. Specifically, courts have found:

- In setting NAAQS, EPA must “allow an adequate margin of safety”⁹ to protect against not only the established health or welfare effects of exposure, but also those “effects which have not yet been uncovered by research and effects whose medical significance is a matter of disagreement.” *Lead Industries*, 647 F.2d at 1154 (emphasis added); *American Lung*, 134 F.3d at 389 (citing *Lead Industries*); *American Trucking*, 283 F.3d at 369 (citing with approval similar language in EPA final rule for ozone NAAQS).
- In setting NAAQS, EPA must account for not only national or average health and welfare effects, but also more geographically “localized” effects on specific communities, including “site-specific” and/or “infrequent” spikes in ambient air pollution. *American Lung*, 134 F.3d at 392.
- As long as a criteria pollutant still “adversely affects the health of” even a single sensitive sub-population, such as children or asthmatic adults, “EPA must strengthen” the NAAQS to eliminate those adverse effects. *American Lung*, 134 F.3d at 389 (discussing sulfur dioxide NAAQS and citing *Lead Industries*, 647 F.2d at 1152-53).

The scientific knowledge gathered since 1978, when EPA adopted the current NAAQS for lead, indicates that lead poses a far *greater* threat to public health than was commonly understood in the mid-to-late 1970s. For example, in the 1970’s a blood lead level of 30 µg/dL was recognized by CDC as requiring individual intervention. Now the CDC action level is 10 µg/dL and CDC has revised its statement on lead poisoning in children to recognize adverse effects below 10 µg/dL.¹⁰ The science cited by EPA shows that “health effects associated with blood [lead] levels extend below 5 µg/dL, and some studies have observed these effects at the lowest blood levels considered.” ANPR at 33. The lowest

⁹ When it adopted its primary and secondary lead NAAQS for lead in October 1978, EPA recognized that Section 109 obliged it to set NAAQS not “at the level estimated to be at the threshold for adverse health effects,” but rather “at a lower level in order to provide a margin of safety.” 43 Fed. Reg. at 46247.

¹⁰ *Preventing Lead Poisoning in Young Children: A Statement by the Centers for Disease Control and Prevention*. U.S. Department of Health and Human Services, Public Health Service, August 2005, at 1-6.

blood lead levels showing statistically significant associations with adverse effects are generally in the range of 2 $\mu\text{g}/\text{dL}$,¹¹ which translates to a NAAQS below 0.2 $\mu\text{g}/\text{m}^3$. ANPR at 147; Staff Paper at 5-34. Even after accounting for reductions in lead levels since 1978, the current NAAQS does not protect public health with an adequate margin of safety. Therefore, the Act requires EPA to adopt lower, more protective NAAQS. Uncertainties in EPA's calculations of risk and exposure only highlight that the current NAAQS does not provide an adequate margin of safety for public health and that the standard must be tightened. Moreover, the ANPR's failure to provide any justification for policy options revoking the lead NAAQS and de-listing lead as a criteria pollutant or for policy options retaining the current NAAQS renders these options arbitrary.

A. The “Latest Available Science” Indicates That the Present Primary NAAQS of 1.5 $\mu\text{g}/\text{m}^3$ Should Be Reduced to a Level Below 0.2 $\mu\text{g}/\text{m}^3$ That is Sufficient to Protect Public Health With an Adequate Margin of Safety.

A large and consistent body of scientific literature, including both animal toxicology and hundreds of epidemiological studies, establishes that lead is a significant environmental public health hazard that is particularly dangerous for children and other sensitive sub-populations that collectively constitute over ninety million U.S. residents.¹² ANPR at 33; *see, e.g.*, Staff Paper at 3-19 (Table 3-3). The “evidence indicates both the sensitivity of the first 3 years of life, and a sustained sensitivity throughout the lifespan as the human central nervous systems continues to mature and be vulnerable to neurotoxicants.” ANPR at 39. Lead affects the brain by impairing neurological development, blunting IQ, and shortening children's attention spans. *See* ANPR at 34-36. Lead's effects on

¹¹ *See e.g.* Menke A, Muntner P, Batuman V, et al. Blood lead below 0.48 $\mu\text{mol}/\text{L}$ (10 $\mu\text{g}/\text{dL}$) and mortality among US adults. *Circulation* 114:1388-1394, 2006. Nigg JT, Knottnerus GM, Martel MM, et al. Low blood lead levels associated with clinically diagnosed attention-deficit/hyperactivity disorder and mediated by weak cognitive control. *Biol Psych* 2007. doi:10.1016/j.biopsycho.2007.07.013. Braun JM, Kahn RS, Froehlich T, et al. Exposures to environmental toxicants attention deficit hyperactivity disorder in U.S. children. *Environ Health Perspect* 114(12):1904-1909, 2006. Miranda ML, Kim D, Galeano MAO, et al. The relationship between early childhood blood lead levels and performance on end-of-grade tests. *Environ Health Perspect* 115(8):1242-1247, 2007.

¹² The Staff Paper correctly observes that these other sensitive populations include the elderly and individuals suffering from hypertension; from diabetes; from calcium, iron, and other nutritional deficiencies; from chronic renal insufficiency; or from certain genetic polymorphisms. ANPR at 39; Staff Paper at 3-16 to 3-19, 5-3.

adults and on children and other sensitive subpopulations require EPA to set the primary NAAQS below $0.2 \mu\text{g}/\text{m}^3$, at a level sufficient to protect public health with an adequate margin of safety, on no longer than a monthly average basis.

i. Lead's Threats to Adult Health Alone Compel EPA to Set the Primary NAAQS Below $0.2 \mu\text{g}/\text{m}^3$.

Lead threatens the health of adults, particularly elderly or other uniquely sensitive adults. Lead exposures affect the kidneys and cardiovascular system and have been linked to diseases ranging from osteoporosis to cataracts to cognitive decline in the elderly. Toxicological and epidemiological information that has become available since EPA's last review of the lead NAAQS shows that lead could have "deleterious effects" at "the lowest blood levels considered" and that "there is no level of [lead] exposure that can yet be identified, with confidence, as clearly not being associated with some risk of deleterious health effects." ANPR at 33, 146.

Adults have been shown to suffer from adverse effects at blood lead levels ("BLLs") well below $10 \mu\text{g}/\text{dL}$. A major study based on the third National Health and Nutrition Examination Survey followed 13,946 adults over a 12 year period to ascertain all-cause and cause-specific mortality.¹³ The geometric mean BLL in the study participants was $2.58 \mu\text{g}/\text{dL}$. The researchers found statistically significant increases in both cardiovascular mortality (55% increase) and in all-cause mortality (25% increase) when they compared participants in the highest tertile ($\text{BLL} \geq 3.62 \mu\text{g}/\text{dL}$) with those in the lowest tertile ($\text{BLL} < 1.94 \mu\text{g}/\text{dL}$). In addition, BLLs were significantly associated with myocardial infarction and stroke mortality at all levels $\geq 2 \mu\text{g}/\text{dL}$. This study suggests that in order to protect the general population of U.S. adults from adverse effects of lead, EPA must set the NAAQS at a level which assures that the distribution of BLLs in the adult population (including more exposed subpopulations) will be below $2 \mu\text{g}/\text{dL}$.

At blood lead levels of $2 \mu\text{g}/\text{dL}$, using even EPA's 1978 primary NAAQS methodology with current information yields a NAAQS of 0.1 to $0.2 \mu\text{g}/\text{m}^3$. ANPR at 147; Staff Paper at 5-34. As EPA recognized when it adopted its secondary lead NAAQS in 1978, Section 109 of the Act obliges EPA to set NAAQS not at "the level estimated to be at the threshold for adverse health effects," but rather "at a lower level in order to provide a margin of safety." 43 Fed. Reg. at 46,247. Therefore, EPA's Staff Paper recommends a primary NAAQS in the range between 0.02 and $0.2 \mu\text{g}/\text{m}^3$. Staff Paper at 5-40, 5-44 to 5-

¹³ Menke A, Muntner P, Batuman V, et al. Blood lead below $0.48 \mu\text{mol}/\text{L}$ ($10 \mu\text{g}/\text{dL}$) and mortality among U.S. adults. *Circulation* 114:1388-1394, 2006 (Attached as Exhibit C).

45. CASAC has also recommended a primary NAAQS of 0.2 $\mu\text{g}/\text{m}^3$ or less. *Id.* at 5-23. To protect the health of adults with an adequate margin of safety, EPA must follow the unanimous recommendation of its independent scientific advisors and staff and set the primary NAAQS at a level below 0.2 $\mu\text{g}/\text{m}^3$.

ii. Lead's Threats to Children's Health Compel EPA to Set the Primary NAAQS Well Below 0.2 $\mu\text{g}/\text{m}^3$ to Provide an Adequate Margin of Safety to Protect Public Health.

a. Lead Remains a Serious Threat to Children's Health.

Lead continues to pose a serious threat to children's health. Thousands of major scientific studies link lead to threats to the health of children, the sensitive subpopulation identified in the ANPR. As the ANPR notes, the CDC has formally recognized that *no level of lead in a child's blood can be specified as safe.*¹⁴ EPA's own Staff Paper also concludes that "there is now no recognized safe level of [lead] in children's blood, and studies appear to show adverse effects at mean concurrent blood [lead] levels as low as 2 $\mu\text{g}/\text{dL}$." ANPR at 120.

In the time since EPA reviewed the literature for the Staff Paper, even more scientific research has emerged that supports the conclusion that there is no "safe" blood lead level for children. These new studies substantially add to the overwhelming body of scientific evidence clearly showing adverse effects in children at BLLs well below 5 $\mu\text{g}/\text{dL}$.

Nigg et al (2007) studied 150 children ages 8-17 years, including children with two forms of Attention Deficit Hyperactivity Disorder (ADHD) and normal children.¹⁵ The blood lead levels in this study population ranged from 0.4 to 3.4 $\mu\text{g}/\text{dL}$, with a mean of 1.03 $\mu\text{g}/\text{dL}$, mirroring the range in the U.S. population today. Blood lead levels in the children with ADHD-combined type were statistically significantly higher than in control children. Blood lead was associated with both inattention-disorganization and with hyperactivity-impulsivity in these children. Thus, this study reported adverse effects in children at BLLs below 3.4 $\mu\text{g}/\text{dL}$.

¹⁴ ANPR at 29; *see Preventing Lead Poisoning in Young Children: A Statement by the Centers for Disease Control and Prevention*, at 1-6 (emphasis added) (U.S. Department of Health and Human Services, Public Health Service, August 2005).

¹⁵ Nigg JT, Knottnerus GM, Martel MM, et al. Low blood lead levels associated with clinically diagnosed attention-deficit/hyperactivity disorder and mediated by weak cognitive control. *Biol Psych* 2007. doi:10.1016/j.biopsycho.2007.07.013 (Attached as Exhibit D).

The association between ADHD and lead exposure in childhood was further defined by Braun et al (2006), based on data from the National Health and Nutrition Examination Survey (NHANES), a statistically representative subsample of the entire U.S. population in 1999-2002.¹⁶ This study, which included data on 4,704 children ages 4-15, found a statistically significant association between higher blood lead levels and ADHD. Children in the top quintile (BLL of ≥ 2.0 $\mu\text{g}/\text{dL}$) of population blood lead had a 4.1-fold higher likelihood of carrying a diagnosis of ADHD compared to children in the lowest quintile (BLL of ≤ 0.7). On the basis of these findings, the authors projected that environmental lead exposure accounts for 290,000 excess cases of ADHD in U.S. children. This study supports the finding that BLLs as low as 2 $\mu\text{g}/\text{dL}$ are associated with adverse neurobehavioral effects in children.

Two recent studies on children in Mexico found significant cognitive and neurobehavioral deficits even at low blood lead levels. These studies are particularly important because they involve children whose main exposures to lead are primarily from air sources.

Kordas et al (2006) studied 602 first-graders living near a metal foundry.¹⁷ According to the authors: "Lead particles are swept from the depository by winds and settle in dust around schools and houses. Smelting also releases lead particles into the air." This study found that as lead levels went up, performance on cognitive tasks went down. As in several other studies, the relationship was not linear, with greater estimated cognitive deficit for every 1 $\mu\text{g}/\text{dL}$ in the range below 10 $\mu\text{g}/\text{dL}$ than in the range above that level. The authors also found significant combined effects on cognitive performance from blood lead and other factors, such as lower socioeconomic status, mothers who did not complete high school, and less parental involvement. This suggests vulnerable subpopulations requiring special consideration by EPA.

The second study in Mexico was a recent cohort study of 294 1-2 year-old children in Mexico City, focused on the effects of BLLs below 10 $\mu\text{g}/\text{dL}$.¹⁸ The

¹⁶ Braun JM, Kahn RS, Froehlich T, et al. Exposures to environmental toxicants attention deficit hyperactivity disorder in U.S. children. *Environ Health Perspect* 114(12):1904-1909, 2006 (Attached as Exhibit E).

¹⁷ Kordas K, Canfield RL, Lopez P, et al. Deficits in cognitive function and achievement in Mexican first-graders with low blood lead concentrations. *Environ Res* 100:371-386, 2006 (Attached as Exhibit F).

¹⁸ Tellez-Rojo MM, Bellinger DC, Carmen Arroyo-Quiroz H, et al. Longitudinal associations between blood lead concentrations lower than 10 $\mu\text{g}/\text{dL}$ and neurobehavioral

study found that higher blood lead levels at ages 1 and 2 were associated with lower scores on the Mental Development Index and the Psychomotor Development Index at age 2. The slope of the relationship was steeper at BLLs below 5 µg/dL. The authors concluded that: “These findings thus provide additional evidence that 10 µg/dL should not be viewed as a biological threshold for lead neurotoxicity.”

Other new research has shown that blood lead levels as low as 2 µg/dL cause statistically significant discernable impacts on the performance of school-aged children on standardized tests.¹⁹ This study analyzed the educational testing data for over 8,600 fourth grade students in North Carolina in 2000-2004, and linked the test results to blood lead surveillance data for seven counties. The authors found that a blood lead level of 5 µg/dL was associated with a decline in reading and mathematics test scores that is roughly equal to the magnitude of the impact of poverty on educational performance. There was a discernable effect on the test results in this cohort at blood lead levels as low as 2 µg/dL.

A prospective cohort study on 194 children in Rochester, NY focused specifically on the question of whether blood lead levels less than 10 µg/dL can adversely affect cognitive function.²⁰ In this study population, the lifetime average BLL was 7.2 µg/dL, with a range from 1.4-27.1 µg/dL. Lifetime average blood lead concentrations in these children (now 6 years old) were inversely associated with IQ. In comparison to the children with an average BLL < 5 µg/dL, children with average BLLs between 5-9.9 µg/dL scored 4.9 points lower on both full-scale IQ and on performance IQ. The researchers found similar relationships when they examined concurrent lead levels, infancy lead levels, and peak lead levels. A dose-response assessment of the peak blood lead relationship with IQ revealed a non-linear relationship with an inverse association between BLL and full-scale IQ down to 2.1 µg/dL (the lowest peak lead level measured in this study population). The slope of the relationship was steeper at lower blood lead levels, consistent with the findings in numerous other studies.

development in environmentally exposed children in Mexico City. *Pediatrics* 118:e323-e330, 2006 (Attached as Exhibit G).

¹⁹ Miranda ML, Kim D, Galeano MAO, et al. The relationship between early childhood blood lead levels and performance on end-of-grade tests. *Environ Health Perspect* 115(8):1242-1247, 2007 (Attached as Exhibit H).

²⁰ Jusko TA, Henderson CR, Lanphear BP, et al. Blood lead concentrations less than 10 micrograms per deciliter and child intelligence at 6 years of age. *Environ Health Perspect* doi:10.1289/ehp.10424, Online November 20, 2007.

As many of these studies indicate, current lead concentrations are associated with significant IQ losses. The Staff Paper shows that, at current lead levels, in the general urban case studies, the estimate for median total IQ loss is 4.5 points and the 95th percentile estimate is 7.7 points. Staff Paper at 4-34 to 4-35 (Tables 4-3 and 4-4). The Staff Paper estimates that IQ loss associated with policy-relevant lead falls between 1.3 and 3.6 points for the population median and between 2.2 and 6.0 points for the 95th percentile. *Id.* at 5-19.

The commenters urge EPA to set a health-protective NAAQS well below 0.2 $\mu\text{g}/\text{m}^3$ to provide an adequate margin of safety to protect public health. According to the ANPR, “to the extent that incremental exposure reductions achieved through lowering the NAAQS might contribute to incremental reductions in children’s blood [lead] and to associated reductions in health effects, consideration of NAAQS levels below 0.1 $\mu\text{g}/\text{m}^3$ (e.g., the lower levels included in the risk assessment of 0.02 and 0.05 $\mu\text{g}/\text{m}^3$) may be appropriate.” ANPR at 158. According to EPA, this low end of the range would limit “the air-related component of risk to somewhat less than 1 point IQ loss at the population level.” ANPR at 159. As described below, the loss of one IQ point at a population level is judged by the CASAC and by the existing scientific literature to be “highly significant from a public health perspective.” ANPR at 122. Therefore, it is critical that EPA select a level for the primary NAAQS that will prevent a population-level IQ loss of greater than or equal to one point.

b. The Loss of Even One IQ Point Is Highly Significant From a Public Health Perspective.

EPA solicits comment on whether the loss of a single IQ point is an adverse effect that must be protected against. ANPR at 160. The answer is an emphatic yes. There has been extensive research into the effect of the loss of a single IQ point, and this research suggests that the impacts are large and significant. Schwartz et al (1994) estimated the impact of the loss of one IQ point.²¹ In this analysis, IQ scores increased between 0.185 and 0.323 points for each 1 $\mu\text{g}/\text{dL}$ blood lead concentration. It was further estimated that each IQ point raises worker’s productivity by 1.76–2.38%, and that the economic benefit for each year’s cohort of 3.8 million 2-year-old children ranges from \$110 to \$319 billion. The Schwartz study found an economic loss per IQ point of \$5,307 per child, a loss attributable largely to reduced cognitive ability, reduced academic achievement, and ultimately decreased individual earning potential. An updated reanalysis estimated an earnings loss per IQ point of \$6,303 per male and \$10,929

²¹ Schwartz J. Societal benefits of reducing lead exposure. *Environ Res* 66:105-124, 1994.

per female (even in 1987 dollars).²² An updated study based on the more recent distribution of blood lead levels in the general U.S. population in 1997 calculated a total economic loss attributable to lead exposure in the birth cohort of 5-year-olds to be \$43.4 billion per year.²³

These studies are consistent with the Staff Paper and CASAC's determinations that a population level loss of 1-2 IQ points is highly significant from a public health perspective. Staff Paper at 5-39; ANPR at 122.

Not surprisingly, the studies also concluded that the benefit of reducing the average BLL in the population by 1 µg/dL is also highly significant. Schwartz estimated the total benefit of a 1 µg/dL reduction in BLL in one year's cohort of children at \$6.937 billion (in 1987 dollars).²⁴ Salkever, using gender-specific data and updated labor market trends, suggests that the impacts are likely to be greater - - approximately \$8.4 billion per 1 µg/dL change in BLL for each year's cohort of children.²⁵

**c. The Staff Paper's, CASAC's, and CHPAC's
Conclusions and Recommendations Are Consistent
with the Latest Science on Lead's Significant Public
Health Impacts.**

All the studies discussed above support the conclusion of the Staff Paper, CASAC, and CHPAC that lead continues to be a public health hazard. Staff Paper at 5-13; CHPAC Letter. The Staff Paper concludes that lead has "adverse health effects at appreciably lower blood [lead] levels than those demonstrated by the evidence at the time" the current standard was set. Staff Paper at 5-24. In addition, the Staff Paper notes that "the current health effects evidence and findings in our exposure assessment . . . supports the conclusion that airborne [lead] exposure pathways (by inhalation and ingestion) contribute to blood [lead]

²² Salkever DS. Updated estimates of earnings benefits from reduced exposure of children to environmental lead. Environmental Research 70:1-6, 1995 (Attached as Exhibit I).

²³ Landrigan PJ, Schechter CB, Lipton JM, et al. Environmental pollutants and disease in American children: Estimates of morbidity, mortality, and costs for lead poisoning, asthma, cancer, and developmental disabilities. Environ Health Perspect 110(7):721-728, 2002 (Attached as Exhibit J).

²⁴ Schwartz J. Societal benefits of reducing lead exposure. Environ Res 66:105-124, 1994.

²⁵ Salkever DS. Updated estimates of earnings benefits from reduced exposure of children to environmental lead. Environmental Research 70:1-6, 1995.

levels in young children, and that the proportion of the contribution from these pathways to total blood [lead] levels is likely larger than that estimated when the standard was set and may be several times higher.” *Id.* at 5-24. In light of such evidence, the Staff Paper concludes that “the overall body of evidence clearly calls into question the adequacy of the current standard.” *Id.* at 5-44. Similarly, CASAC concludes that lead toxicity “remains a major public health problem” and that the current NAAQS are “totally inadequate” for assuring necessary decreases of lead exposures for sensitive populations. *Id.* at 5-23. As discussed above, blood lead levels of 2 µg/dL show statistically significant associations with adverse health effects. Using the 1978 methodology, this blood lead level translates to an ambient lead level in the range of 0.1-0.2 µg/m³. ANPR at 147; Staff Paper at 5-34. However, to abide by the Act’s mandate, EPA must set the NAAQS at a level below 0.2 µg/m³ that is sufficient to provide an adequate margin of safety to protect public health. CHPAC specifically recommends a NAAQS of 0.02 µg/m³ or lower, well below the current NAAQS. CHPAC Letter at 1. EPA’s own analysis shows that to avoid a 1 IQ point loss at the population level, a highly significant loss from a public health perspective, the NAAQS should be set at the lower end of the range of options considered (0.02-0.05 µg/m³). ANPR at 158-59. Both of these levels are more appropriate levels than a level of 0.2 µg/m³.

d. The CDC Action Level of 10 µg/dL Should Not be Misinterpreted as a “Safe Level,” and the NAAQS Should Be Set at a Level That Provides an Adequate Margin of Safety to Protect Public Health.

It is clear from the studies above that a blood lead level of 10 µg/dL is not a threshold for toxicity. The ANPR notes that “current evidence does not indicate a threshold for the more sensitive health endpoints such as adverse effects on the developing nervous system.” ANPR at 117. The extensive research well-summarized in the Staff Paper, and the additional studies cited above, clearly show that lead has adverse effects at levels significantly below 10 µg/dL, and even well below 5 µg/dL. Although the CDC still uses the 1991 level of 10 µg/dL as an action level, CDC recently issued a document on “Interpreting and Managing Blood Lead Levels <10 µg/dL in Children and Reducing Childhood Exposures to Lead” (November 2, 2007).²⁶ This document clearly qualifies the action level of 10 µg/dL, stating that “CDC also recognized that a BLL of 10 µg/dL did not define a threshold for the harmful effects of lead. Research conducted since 1991

²⁶ Recommendations of CDC’s Advisory Committee on Childhood Lead Poisoning Prevention. Interpreting and Managing Blood Lead Levels <10 µg/dL in Children and Reducing Childhood Exposures to Lead. MMWR 56(RR08):1-14, 2007.

has strengthened the evidence that children's physical and mental development can be affected at BLLs $<10 \mu\text{g/dL}$.”²⁷ The report also reaffirms the 2005 CDC report's finding that “no safe level for blood lead in children has been identified.”²⁸

As discussed above, statistically significant associations with adverse health effects have been shown at blood lead levels of $2 \mu\text{g/dL}$, and at a target blood lead level of $2 \mu\text{g/dL}$, EPA should establish a primary NAAQS below $0.2 \mu\text{g/m}^3$ as recommended by the Staff Paper and CASAC. The current assessment that no blood lead level has been established as safe for children argues for EPA to adopt a far lower standard in order to protect public health with an adequate margin of safety. The Staff Paper, CASAC, and CHPAC recommendations recognize this principle. EPA's Staff Paper recommends a NAAQS in the range between 0.02 and $0.2 \mu\text{g/m}^3$, while noting that at $0.1 \mu\text{g/m}^3$, the upper end of the range of policy-relevant IQ loss estimates extends up to and above 1 point IQ loss. Staff Paper at 5-40, 5-44 to 5-45. To be more precautionary, and to avoid the risk of population level IQ loss in the 1-2 point range, which has significant impacts and has been identified as highly significant from a public health perspective by the Staff Paper and CASAC, the Staff Paper suggests that the NAAQS should be set more appropriately in the 0.02 - $0.05 \mu\text{g/m}^3$ range. *Id.* at 5-39, 5-40 to 5-41. Similarly, CHPAC recommends setting the NAAQS at or below $0.02 \mu\text{g/m}^3$. CHPAC Letter at 1, 3. Both of these levels are more appropriate as a NAAQS than $0.2 \mu\text{g/m}^3$.

EPA's own estimates of IQ loss demonstrate the need to substantially lower the primary NAAQS. For example, in the ANPR, EPA presents an analysis that focuses only on “recent air estimates” and still finds that “estimates of IQ loss (based on the LLL function) associated with policy-relevant [lead] at the 95th percentile of population total IQ loss are greater than 1 point for all current conditions scenarios in all three urban case studies for which the lowest air [lead] concentrations are $0.09 \mu\text{g/m}^3$ maximum quarterly average and $0.17 \mu\text{g/m}^3$ maximum monthly average.” ANPR at 153. This analysis underestimates IQ loss in several regards, including the limitation to “recent air,” which excludes all airborne lead that has settled onto soil; the limitation to “policy-relevant” lead, which excludes all dietary lead that has occurred from airborne emissions; and the focus on the 95th percentile, which excludes the top 5% of the exposed population. Yet the analysis still reveals an IQ loss that is considered by CASAC to be highly significant from a public health perspective at airborne lead levels that are

²⁷ *Ibid* p. 1.

²⁸ *Ibid* p. 1.

significantly below the current standard. This analysis strongly suggests that the primary standard should be set well below $0.17 \mu\text{g}/\text{m}^3$ maximum monthly average.

Within the three location-specific urban case study areas, EPA found that there would be a range of “approximately 100 to 3,000 fewer children having total IQ loss greater than 1.0 for an alternative standard of $0.02 \mu\text{g}/\text{m}^3$, maximum monthly mean. Further, just meeting the lowest alternative standard [of $0.02 \mu\text{g}/\text{m}^3$] in these three study areas is estimated to reduce the number of children having an IQ loss greater than seven points by one to two percent. This corresponds to a range of approximately 350 (for the Cleveland study area) up to 8,000 (for the Chicago study area) fewer children with total [lead]-related IQ loss greater than 7.0.” ANPR at 155-156. This EPA scientific analysis argues strongly for a NAAQS at a level well below $0.2 \mu\text{g}/\text{m}^3$, one that is sufficient to protect public health with an adequate margin of safety.

iii. Lead’s Threats to the Health of Other Sensitive Subpopulations and Urban Communities Also Compel EPA to Set the Primary NAAQS Below $0.2 \mu\text{g}/\text{m}^3$.

As noted above, the Clean Air Act requires EPA to set its NAAQS at a level or levels stringent enough to protect all sensitive sub-populations from the harmful effects of lead exposure with an “adequate margin of safety.” *American Lung*, 134 F.3d at 389-92; *Lead Industries*, 647 F.2d at 1153. The Act also requires EPA, in setting NAAQS, to account not only for the national or average health and welfare effects, but also more geographically “localized” effects on specific communities. *American Lung*, 134 F.3d at 392. These directives require EPA to establish more stringent NAAQS than the current standards.

EPA must ensure that its new NAAQS are not only adequate to protect the average member of the population, but also to guard against adverse effects in vulnerable subpopulations. As the U.S. Court of Appeals for the D.C. Circuit has stated,

In its effort to reduce air pollution, Congress defined public health broadly. NAAQS must protect not only average healthy individuals, but also “sensitive citizens” – children, for example, or people with asthma, emphysema, or other conditions rendering them particularly vulnerable to air pollution. If a pollutant adversely affects the health of these sensitive individuals, EPA must strengthen the entire national standard.²⁹

²⁹ *American Lung Assn. v. EPA*, 134 F.3d 388, 390 (D.C. Cir. 1998) (citations omitted); see also *Lead Industries Assn, Inc. v. EPA*, 647 F.2d 1130, 1153 (D.C. Cir.

Research on the pharmacokinetics of lead has shown that there are significant differences in absorption of ingested lead based on nutritional factors. This scientific information is highly relevant to the lead NAAQS because hand-to-mouth ingestion of airborne lead deposited into soil or dust is acknowledged as the main pathway of exposure for young children. Subpopulations of children with nutritional deficiencies are likely to be significantly more susceptible to lead toxicity, and therefore are entitled to greater protection under the Act. Diets low in calcium are known to significantly increase lead absorption and toxicity.^{30 31} In fact, there is an inverse relationship between brain lead levels and dietary calcium.³² Similarly, dietary iron deficiency has been found in numerous studies to be associated with increased intestinal absorption of lead, and with increased vulnerability to lead toxicity.³³ EPA must therefore incorporate a margin of safety into the lead NAAQS that is adequate to protect not just children, but also children with nutritional deficiencies as well as other sensitive subpopulations.³⁴ The need to protect sensitive subpopulations like children with nutritional deficiencies – especially in light of the preceding discussion concerning the health impacts of lead on children – only underscores EPA’s obligation to establish a primary NAAQS well below 0.2 $\mu\text{g}/\text{m}^3$.

The CDC identified a variety of “populations that are unusually susceptible” in its updated Toxicological Profile for Lead (2007). These include “crawling and house-bound children (< 6 years old), pregnant women (and the fetus), the elderly, smokers, alcoholics, and people with genetic diseases affecting heme synthesis, nutritional deficiencies, and neurological or kidney

1980) (NAAQS must “be set at a level at which there is ‘an absence of adverse effect’ on these sensitive individuals”).

³⁰ Mahaffey KR. Environmental lead toxicity: nutrition as a component of intervention. *Environ Health Persp* 89:75-78, 1990 (Attached as Exhibit K).

³¹ Mahaffey KR. Nutrition and lead: strategies for public health. *Environ Health Perspect* 103(supp 6):191-196, 1995 (Attached as Exhibit L).

³² Goyer RA. Nutrition and metal toxicity. *Am J Clin Nutr* 61(suppl):646S-650S, 1995.

³³ Ros C, Mwanri L. Lead exposure, interactions and toxicity: food for thought. *Asia Pacific J Clin Nutr* 12(4):388-395, 2003 (Attached as Exhibit M).

³⁴ As the Staff Paper observes that these other sensitive populations include the elderly and individuals suffering from hypertension; from diabetes; from calcium, iron, and other nutritional deficiencies; from chronic renal insufficiency; or from certain genetic polymorphisms. ANPR at 39; Staff Paper at 3-16 to 3-19, 5-3.

dysfunction.”³⁵ These populations must all be considered and protected in setting the NAAQS.

EPA must also account for localized lead impacts in setting the NAAQS, specifically the impacts on urban communities. EPA modeled total blood lead increases in the urban case studies associated with increased emissions of lead that would still meet the NAAQS. This roll-up analysis suggested an increase in median blood lead levels of approximately 1 µg/dL in two of the case studies (Chicago and Los Angeles). Applying a 1 µg/dL increase in median BLL to the combined population of these two cities (more than 500,000 children under age 5),³⁶ would have a devastating impact on both the health of these children and the future economy of those regions. For example, the benefit of reducing the average BLL in one year’s cohort of children has been estimated at \$6.937 billion even in 1987 dollars.³⁷ A reanalysis using gender-specific data and updated labor market trends suggests that the impacts are likely to be greater – approximately \$8.4 billion per 1 µg/dL change in BLL.³⁸ Thus, the impacts of the current NAAQS standard on urban communities also compel EPA to consider a NAAQS standard well below 0.2 µg/m³.

iv. Lead’s Disproportionate Threats to Environmental Justice Communities Also Compel EPA to Set the Primary NAAQS at a Level Below 0.2 µg/m³.

The ANPR makes clear that EPA has not yet met its obligation to analyze the environmental justice implications of the various NAAQS options it proposes for the Administrator’s consideration. EPA must do more to respond to the established evidence of lead’s disproportionate impacts on environmental justice communities.

Executive Order 12898, *Federal Action to Address Environmental Justice in Minority Populations and Low-Income Populations*, requires EPA, to “the

³⁵ Agency for Toxic Substance and Disease Registry, U.S. Department of Health and Human Services, *Toxicological Profile for Lead*, at 237 *et seq.*

³⁶ See U.S. Census Bureau, State and County Quickfacts (Illinois), *available at*: <http://quickfacts.census.gov/qfd/states/17/1714000.html> (last revised Jan. 2, 2008); U.S. Census Bureau, State and County Quickfacts (California), *available at*: <http://quickfacts.census.gov/qfd/states/06/0644000.html> (last revised Jan. 2, 2008).

³⁷ Schwartz J. Societal benefits of reducing lead exposure. *Environ Res* 66:105-124, 1994.

³⁸ Salkever DS. Updated estimates of earnings benefits from reduced exposure of children to environmental lead. *Environmental Research* 70:1-6, 1995.

greatest extent practicable and permitted by law ... make achieving environmental justice part of its mission by identifying and addressing, as appropriate, disproportionately high and adverse human health or environmental effects of its programs, policies, and activities on minority populations and low-income populations in the United States.” Exec. Order No. 12,898, § 1-101, 52 Fed. Reg. 7,629 (Feb. 11, 1994). In 2006, twelve years after the Executive Order’s passage, EPA’s Inspector General determined that EPA senior management had not sufficiently directed program and regional offices to conduct environmental justice reviews in accordance with the Executive Order and that the majority of programs and offices had not conducted such reviews.³⁹ Yet, the ANPR continues this pattern of inattention to environmental justice.

Lead’s hazards have long been recognized to fall disproportionately on poor, minority and urban communities most likely to have elevated blood lead levels. In 1999, the General Accounting Office (“GAO”) recognized that low-income children in older housing are at particularly “high risk for lead poisoning.”⁴⁰ GAO also recognized that despite several decades of federal and state lead-control efforts, “the risk for lead exposure remains disproportionately high for some groups, including children who are poor, non-Hispanic black, or Mexican American or are living in large metropolitan areas or in older housing.”⁴¹ The following year, GAO—crediting EPA studies—wrote that “the evidence is unambiguous: children of color have a higher prevalence of elevated blood lead levels than white children do, and children in lower-income communities have a higher prevalence than children in higher income families.”⁴² Recent scientific studies echo these findings, noting that “[m]ost children with elevated blood lead concentrations and lead poisoning have been African-American or Hispanic children from low-income families who live in urban areas.”⁴³ ⁴⁴ As CHPAC

³⁹ U.S. EPA Office of Inspector General, *Evaluation Report: EPA Needs to Conduct Environmental Justice Reviews of Its Programs, Policies, and Activities* (September 18, 2006).

⁴⁰ See GAO, *Lead Poisoning: Federal Health Programs Are Not Effectively Reaching At-Risk Children 2* (January 1999), GAO/HEHS-99-18, available at <http://www.gao.gov/archive/1999/he99018.pdf> (last visited Jan. 10, 2008) (Attached as Exhibit N).

⁴¹ *Id.* at 15.

⁴² GAO, *Toxic Chemicals: Long-Term Coordinated Strategy Needed to Measure Exposures in Humans 17*, GAO/HEHS-00-80 (May 2000), available at <http://www.gao.gov/new.items/he00080.pdf> (last visited Jan. 10, 2008) (Attached as Exhibit O).

⁴³ Kemp FW, Neti PVS, Howell, RW, et al. Elevated Blood Lead Concentrations and Vitamin D Deficiency in Winter and Summer in Young Urban Children. *Environ Health Persp* 115:630-635, 2007 (Attached as Exhibit P).

notes, because “elevated blood lead levels occur disproportionately among black, non-hispanic and Mexican-American children” and “among children living in poverty,” “protecting children from exposure to lead is inherently an environmental justice issue.” CHPAC Letter at 1-2.

EPA’s own data confirms that the health risks and burdens of lead pollution continue to fall disproportionately on poor communities and communities of color. While the 2001-2004 media blood lead level for all children aged 1-5 was 1.5 $\mu\text{g}/\text{dL}$, the median for the subset of the children living below the poverty line was 2.3 $\mu\text{g}/\text{dL}$. ANPR at 31 n.15. The 90th percentile distinction was even greater – 5.4 $\mu\text{g}/\text{dL}$ for poor children aged 1-5 as compared to 4.0 $\mu\text{g}/\text{dL}$ for all children aged 1-5. *Id.* According to the EPA data, black non-hispanic children are even worse off, with a median blood lead level of 2.5 $\mu\text{g}/\text{dL}$. *Id.* Black children from poor families had a median blood lead level of 2.9 $\mu\text{g}/\text{dL}$, significantly above the median for all poor children. *Id.* Even children from families with income more than 200% of the poverty level had blood lead level of 1.9 $\mu\text{g}/\text{dL}$, above the median blood lead levels for all children. *Id.* The numbers for the 90th percentile of black children was similarly higher than for the other populations discussed, with a median blood lead level of 6.4 $\mu\text{g}/\text{dL}$ for all black children in the age range and 7.7 $\mu\text{g}/\text{dL}$ for black children under the poverty line. *Id.* Despite this striking evidence of disproportionate impacts, the ANPR fails to address the environmental justice implications of the various policy options it sets forth. As CHPAC observes, neither the term “environmental justice” nor a reference to the Executive Order appear in the ANPR. CHPAC Letter at 2.

To account for these environmental justice impacts, and protect the many poor, minority, and urban individuals whose blood already contains dangerously high levels of lead, EPA must set the NAAQS at a level well below 0.2 $\mu\text{g}/\text{m}^3$ – at a level sufficient to provide an adequate margin of safety to protect public health. For instance, CHPAC’s recommended level of 0.02 $\mu\text{g}/\text{m}^3$ or below would be more appropriate. *See* CHPAC Letter at 1, 3.

v. EPA’s “Roll-Up” Analyses Confirm that EPA Cannot Protect Public Health With an Adequate Margin of Safety by Simply Maintaining the Current Primary NAAQS.

⁴⁴ One study has even suggested that African-Americans face particularly heightened risks, perhaps because of a higher prevalence of vitamin D deficiency. Kemp FW, Neti PVSV, Howell, RW, et al. Elevated Blood Lead Concentrations and Vitamin D Deficiency in Winter and Summer in Young Urban Children. *Environ Health Persp* 115:630-635, 2007.

EPA has solicited comment on the propriety of its “roll-up” analysis, which attempts to simulate the effects on blood lead of raising ambient lead levels to just meet the current NAAQS. ANPR at 112. We believe it is not only appropriate, but also *necessary* for EPA to conduct this analysis for all areas where either there are limited data or where the current ambient levels are below the standard. If there is nothing to legally prevent an area from increasing emissions up to the level of the current standard, and ambient lead concentrations at that level would not protect the public health with an adequate margin of safety, then the standard is clearly inadequate. In this sense, the roll-up analysis serves as an important “truth test” of the current lead NAAQS.

The roll-up analysis presented at section III.B of the ANPR indicates very elevated BLLs in children at the current NAAQS. These levels are clear evidence that the current primary NAAQS is inadequate to protect public health and cannot simply be maintained. For example, the roll-up scenario suggests that 5-17 percent of the population in the study areas would suffer from an IQ loss greater than seven points *if the current NAAQS of 1.5 $\mu\text{g}/\text{m}^3$ were met*. EPA observes that “this increase corresponds to approximately a doubling in the number of children with this magnitude of [lead]-related IQ loss in the study area most affected.” ANPR at 128. This analysis clearly shows that the current primary lead NAAQS is not adequate to protect health.

In conclusion, we agree with CASAC that the current lead NAAQS “are totally inadequate to assure the necessary decreases of lead exposures in sensitive U.S. populations below those current health hazard markers identified by a wealth of new epidemiological, experimental, and mechanistic studies.” ANPR at 130; Staff Paper at 5-23.

vi. EPA Has Previously Recognized That Eliminating Incremental Lead Exposures is Necessary to Protect Public Health With an Adequate Margin of Safety.

EPA has previously responded to a mandate to protect public health with an adequate margin of safety by setting a goal of zero lead exposure from drinking water. Following the same reasoning, EPA must establish a more stringent primary NAAQS for lead.

EPA has previously dealt with the requirement to protect public health with an adequate margin of safety pursuant to the Safe Drinking Water Act (“SDWA”). The SDWA requires EPA to establish Maximum Contaminant Level Goals (“MCLGs”) for all contaminants, including lead, that EPA determines may have

adverse health effects and present meaningful opportunities for health risk reduction in the context of public water systems. 42 U.S.C. § 300g-1(b)(1)(A); see also *id.* § 300f(5). MCLGs must be set at a level at which, in the Administrator's judgment, "no known or anticipated adverse health effects on the health of persons occur and which allows an adequate margin of safety." 42 U.S.C. § 300g-1(b)(4)(B). In 1991, EPA recognized that there was no level of lead at which it could assure "no anticipated or adverse health effects" while providing "an adequate margin of safety," and accordingly set the MCLG for lead at zero. See EPA, Maximum Contaminant Level Goals and National Primary Drinking Water Regulations for Lead and Copper, 56 Fed. Reg. 26,460, 26,462 (June 7, 1991) (to be codified at 40 C.F.R. pts. 141 and 142). EPA preserved the MCLG, and reiterated its rationale, when it revised other national drinking water regulations for lead in 2007. EPA, National Primary Drinking Water Regulations for Lead and Copper: Short Term Regulatory Revisions and Clarifications, 72 Fed. Reg. 57,782, 57,790 (Oct. 10, 2007) (to be codified at 40 C.F.R. pts 141 and 142).

EPA's rationale in setting the lead MCLG at zero is equally applicable to the lead NAAQS, which similarly must be set at levels which, in the Administrator's judgment, "allowing for an adequate margin of safety, are requisite to protect the public health." 42 U.S.C. 7409(b)(1). In establishing the lead MCLG, EPA observed that the statutory mandate to provide an "adequate margin of safety" required it to consider lead exposure from sources other than the regulated source (drinking water), as well as adverse effects that may be experienced by sensitive subpopulations. 72 Fed. Reg. 57,790; 56 Fed. Reg. 26,469. It then relied on 1) the occurrence of various low-level health effects for which it is difficult to identify clear thresholds levels below which there are no risks of adverse health effects; 2) EPA's policy goal that drinking water should contribute minimal additional lead to existing exposures because a portion of the sensitive subpopulation (children) already exceeds acceptable blood lead levels; and 3) the classification of lead as a probable human carcinogen. *Id.* As discussed in Sections II.A.i-iii, and as reiterated in EPA's 2007 decision to maintain the lead MCLG at zero, these factors continue to be serious public health concerns. In fact, lead threatens human health at far lower blood lead levels than previously understood.

EPA must apply similar reasoning here to provide an adequate margin of safety for public health by setting the NAAQS at levels that virtually eliminate the incremental contributions of ambient lead to existing lead exposures and blood lead levels.

B. EPA Should Measure the NAAQS Over an Averaging Time No Longer Than One Month, with the Maximum Monthly Mean Calculated Over a Period No Longer than One Calendar Year.

EPA should establish an averaging time no longer than one month for the lead NAAQS, as recommended by CASAC, CHPAC, and EPA's staff. Staff Paper at 5-31, 5-29, 5-31; CHPAC Letter at 2. EPA should also ensure that the maximum monthly mean is calculated over a period no longer than a calendar year.

Based on the scientific evidence, we urge EPA to select an averaging time no longer than one month. The ANPR appropriately references the NHANES II study and associated findings that children's blood lead levels respond to monthly variations in lead emissions. ANPR at 138. In addition, during pregnancy and early life, increases of lead exposure lasting weeks or a month may be critical to future development. ANPR at 139. EPA acknowledges that "the health evidence indicates that very short exposures can lead to increases in blood [lead] levels." ANPR at 142.

Furthermore, very short-term emissions (even over days) can result in significant localized soil and dust lead loading. A New York City study measured the deposition of lead onto glass settling plates and found significant concentrations of lead were deposited over the course of one week, with up to 40.8 mg/feet²/week deposited on the interior plates (located near open windows), and up to 62.0 mg/feet²/week on outdoor plates.⁴⁵ The researchers remarked: "It is startling to find that the interior lead dust standards may be exceeded after only 3 weeks of accumulation."⁴⁶ Due to the persistence of lead, even short-term airborne emissions can result in long-term exposure to children via soil or indoor dust. EPA acknowledges that "the time period of response of indoor dust [lead] to airborne [lead] can be on the order of weeks." ANPR at 142.

It is important to note that the Staff Paper demonstrated that four percent of the monitoring sites in the dataset that met the current standard would have exceeded the current standard if the monitoring data were averaged over a month instead of over a quarter. EPA notes that this same analysis indicates that this number "is as high as ten percent for some alternate lower levels," ANPR at 140, suggesting that over periods such as one week, there is significant airborne loading

⁴⁵ Caravanos J, Weiss AL, Jaeger RJ. An exterior and interior leaded dust deposition survey in New York City: results of a 2-year study. *Environ Res.* 100(2):159-64, 2006 (Attached as Exhibit Q).

⁴⁶ *Ibid.* p. 164.

of lead at a significant fraction of the existing monitoring sites. When one considers the fact that most of the major lead emitters in the U.S. do not have monitors located downwind, the implication is that there are significant unmeasured and uncontrolled airborne lead emissions that can and should be controlled in order to protect public health. EPA would be shirking its responsibilities under the Act if it failed to appropriately take steps to reduce the averaging time so as to adequately control short-term releases of lead.

EPA should set the form of the standard at no greater than the maximum monthly mean in one calendar year. As summarized above, even short-term high-level emissions of airborne lead are a significant health problem because the lead is deposited onto soil and dust, and poses an ongoing ingestion risk. Moreover, health evidence indicates that very short exposures can lead to increases in blood lead levels and that adverse effects may occur with exposures during short windows of susceptibility. ANPR at 142. The ambient air concentration of lead allowed by the NAAQS represents the level beyond which exposure to lead does not allow for an adequate margin of safety for public health. *See* 42 U.S.C. 7409(b)(1). Therefore, it is inconsistent with the language of the Act to allow *any exceedances of the standard at all*. Point source emissions are notable for being sporadic or cyclical.⁴⁷ Depending on the process conditions, emissions from a facility such as a smelter can vary widely. Yet the lead that is emitted will fall to earth in the community and impact the health of the residents regardless of whether it is emitted continuously at a low level, or in one large burst. The responsibility therefore falls on EPA to assure that significant amounts of lead are not emitted in short bursts from these point source facilities. Setting the standard at the maximum monthly mean would be the most likely of the options presented in the ANPR to protect against these sporadic emission problems. However, EPA should look beyond the options presented in the ANPR to the shortest averaging time that is technically feasible, including a daily averaging option which would better ensure that large sporadic emissions (and therefore exposures beyond levels that allow for an adequate margin of safety) do not occur. In no event, however, should the NAAQS be measured over an averaging time greater than a month.

The averaging time should apply over a period no greater than a year. It would be reckless and arbitrary to change the form to apply to a three-year period

⁴⁷ For example, a single facility could release one ton per year of lead, but may release most of that amount during a process that occurs only once a week, say on Monday nights. Such a situation could generate over 35 pounds of lead emissions just on Monday night, with essentially no emissions other days. Such variation in emissions may also occur because of accidents or equipment breakdowns.

(as suggested, without any discussion, on p. 143 of the ANPR). A three-year period is not supported by any scientific or analytical evidence, and has no substantive basis. The result of a three-year averaging period would be to unreasonably delay any designations of areas as non-attainment (since such designations could only be made on the basis of three years of data), and to allow sporadic emitters (such as smelters) to continue to blanket communities with lead, contaminating the soil and indoor dust, without violating the NAAQS.

C. The Significant Uncertainties in EPA's Assessments of Lead's Threats to Public Health and Welfare Oblige EPA to Set a More Stringent NAAQS.

EPA's preliminary analyses rely in part on incomplete datasets and methodologies that tend to understate or render more uncertain estimates of the health risks associated with lead. These significant modeling and data issues, coupled with EPA's statutory obligation to assure an adequate margin of safety for all sensitive sub-populations, require EPA to err on the side of a more, not less, stringent standard. *See American Trucking Ass'n v. EPA*, 283 F.3d at 369-70, 378 (discussing ozone NAAQS); *Lead Industries*, 647 F.2d at 1154-55 (discussing lead NAAQS).

a. EPA's Analysis Improperly Minimizes the Significant Contributions of Outdoor Dust and Soil to Blood Lead by Establishing a False Distinction Between "Past Air" and "Present Air," Allocating Outdoor Soil Exposure to "Past Air," and Treating Dietary Soil Exposure as "Background."

EPA solicits comment on "the approach of considering exposures and risks resulting from the ingestion of historically emitted [lead] that may now be present in indoor dust and outdoor soil . . . impacted by ambient air [lead] as being policy-relevant for the purpose of setting a NAAQS." ANPR at 112-113. As EPA acknowledges, "there is uncertainty related to parsing out exposure and risk between background and policy-relevant pathways (and subsequent parsing of recent air and past air) resulting from a number of technical limitations." ANPR at 65. Yet the Agency fails to make an adequate case that such parsing is useful or necessary for the analysis. Lead from all sources, including historic sources, contributes to the present-day problem of airborne lead, soil lead, and dust lead that results in elevated BLLs and adverse health effects. It is not possible to separate these sources effectively, and the source is not germane to the responsibility the Act imposes on EPA to promulgate a standard that will protect the public health with an adequate margin of safety. In addition, as EPA

mentions, “[lead] that is a soil or dust contaminant today may have been airborne yesterday or many years ago.” ANPR at 117. This same lead may again be airborne tomorrow, or many months or years from now. Furthermore, the scientific studies “do not usually distinguish between outdoor soil/dust [lead] resulting from historical emissions and outdoor soil/dust [lead] resulting from recent emissions.” *Id.* Overall, there appears to be no utility to, or legal basis for, the division of lead exposure pathways into “present air”, “past air” and “policy-relevant background.” EPA should not use this artificial and poorly-substantiated division of pathways, and instead should assume that all lead is “policy relevant” for purposes of the analysis.

EPA’s methodology also has the effect of understating the contributions of outdoor soil to children’s blood lead levels and, by extension, understates both total lead exposures and the need for a more stringent NAAQS, because it erroneously lumps all outdoor soil and dust into the “past air” category and thus assumes this dust and soil originates entirely from sources that no longer emit lead. ANPR at 62-64. EPA recognizes that *indoor* dust/soil is an important lead exposure pathway for young children. ANPR at 25. However, outdoor dust/soil is also an important exposure pathway for children.⁴⁸ In fact, Mielke et al have made an excellent argument that lead in soil is at least as important a contributor to children’s blood lead levels as lead in paint.⁴⁹

It is incorrect and inappropriate to assign all soil lead to the “past air” category. EPA has acknowledged, and recent studies confirm, that emissions from point sources of lead remain significant contributors to outdoor soil contamination. *See, e.g.*, Staff Paper at 2-69; *infra* note 49 (citing Mielke et al 1998). EPA has also acknowledged that there are “some 13,067 point sources (industrial, commercial or institutional) in the 2002 NEI, each with one or more processes that emit [lead] to the atmosphere.” Staff Paper at 2-12. These point source lead emissions come from a long list of industrial categories ranging from incinerators, to battery manufacturers, to cement kilns. In fact, there are 37 different source categories in the U.S., each of which emits more than 5 tons per year of lead. Staff Paper at Table 2-2. The commenters reasonably believe that there are millions of people living near active lead sources in the United States. Even areas that are not located near a point source of lead have a significant amount of soil contamination from airborne sources. A 2006 study of outdoor lead deposition

⁴⁸ Lanphear BP, Succop P, Roda S, Henningsen G. The effect of soil abatement on blood lead levels in children living near a former smelting and milling operation. *Public Hlth Reports* 118:83-91, 2003 (Attached as Exhibit R).

⁴⁹ Mielke HW, Reagan PL. Soil is an important pathway of human lead exposure. *Environ Health Perspect* 106(suppl 1):217-229, 1998 (Attached as Exhibit S).

from air onto surfaces in New York City showed that there is significant ongoing deposition occurring today.⁵⁰ The vintage of the original emissions is unknown in this study, but it is clear that the lead must have been airborne recently in order to have been deposited onto these surfaces.

Some commenters may make the argument that lead paint is the major source of present-day soil or dust-borne lead exposure, and that airborne lead is therefore not a significant contributor to lead exposures in the U.S. That argument is contradicted by several important research studies. In urban areas, historical use of lead in gasoline has caused pervasive dust and soil contamination. This dust and soil is an ongoing source of airborne lead, and is also a source of direct exposure to children through hand-to-mouth contact. There is extensive research showing that soil lead is a key contributor to children's blood lead levels.⁵¹ Moreover, studies around lead smelters have shown that lead in soil is more bioavailable than lead in paint by 2-6 fold.⁵² This may owe, in part, to the very fine particle size of airborne lead vs. paint chips and fragments. Several studies have found that the lead in children's blood varies proportionately with lead in soil, but not with age of housing, implying that airborne lead and soil lead are more important than lead paint as exposure sources to children.⁵³ Researchers have also reported that soil lead and house dust lead concentrations are closely correlated, and that house dust is at least 50% soil dust.⁵⁴ Soil lead concentrations of 1000 ppm are commonly found near point sources of airborne lead and in urban areas with a history of heavy vehicle traffic. Mielke et al (1998) have calculated that such concentrations result in loadings of more than 1,000,000 $\mu\text{g}/\text{ft}^2$ in the upper centimeter of the soil and that ingestion of as little as one-sixth of a gram of

⁵⁰ Caravanos J, Weiss AL, Blaise MJ, Jaeger RJ. A survey of spatially distributed exterior dust lead loadings in New York City. *Environ Res.* 100(2):165-72, 2006 (Attached as Exhibit T).

⁵¹ Mielke HW, Reagan PL. Soil is an important pathway of human lead exposure. *Environ Health Perspect* 106(suppl 1):217-229, 1998.

⁵² Roberts TM, Hutchinson TC, Paciga J, et al. Lead contamination around secondary smelters: estimation of dispersal and accumulation by humans. *Science* 186:1120-1123, 1974.

⁵³ Mielke HW, Lead dust contaminated U.S.A cities: comparison of Louisiana and Minnesota. *Appl Geochem* 2(suppl):257-261, 1993.

⁵⁴ Fergusson JE, Forbes EA, Schroeder RJ, Ryan DE. Lead: petrol lead in the environment and its contribution to human blood lead levels. *Sci Total Environ* 50:1-54, 1986.

soil daily at these concentrations could result in a blood lead level of over 30 µg/dL in a child.⁵⁵

We further observe that the dietary contribution of lead involves a soil pathway, since it is lead-contaminated soil that results in uptake into food and resulting dietary exposures.^{56 57} Dietary exposures should therefore be included in the same category as soil lead, rather than in a “background” category. ANPR at 64.

For the reasons above, it is erroneous and inappropriate for EPA to attempt to allocate soil and dust exposures between “past” and “present” air, and to allocate all outdoor soil and dust exposures to “past air” and (in the case of the dietary soil exposure pathway) “background.”

b. EPA’s Analysis Relies on Data from an Incomplete Monitoring Network That Excludes Many of the Largest Lead Point Sources and Thus Understates Lead’s Effects on Urban and Other High-Exposure Communities.

The inadequacy of the existing monitoring network for airborne lead raises significant concerns about EPA’s ability to evaluate the actual extent of the lead’s effects on urban and other high-exposure communities. The number of monitors in the network have decreased from over 900 to about 200 sites, and some states have no monitors “resulting in large portions of the country with no data on current ambient Pb-TSP concentrations.” ANPR at 191. As stated in the ANPR, “many of the largest Pb emitting sources in the country do not have nearby monitors, and there is substantial uncertainty about ambient air [lead] levels resulting from historic [lead] deposits near roadways.” *Id.* Specifically, and despite the fact that a staggering 94% of lead point source emissions are emitted by the largest 10% of sources, there are monitors within a mile of only 2 of the 26 facilities in the 2002 NEI with emissions greater than 5 tons per year. Staff Paper at 2-12, 2-20; ANPR at 43. Also, none of the 189 monitors are located within a mile of airports identified as a location where aircraft that still use leaded aviation

⁵⁵ Mielke HW, Reagan PL. Soil is an important pathway of human lead exposure. *Environ Health Perspect* 106(suppl 1):217-229, 1998.

⁵⁶ Hough RL, Breward N, Young SD, et al. Assessing potential risk of heavy metal exposure from consumption of home-produced vegetables by urban populations. *Environ Health Perspect* 112(2):215-2, 2004. Erratum in: *Environ Health Perspect* 112(5):A270, 2004.

⁵⁷ Finster ME, Gray KA, Binns HJ. Lead levels of edibles grown in contaminated residential soils: a field survey. *Sci Total Environ* 320(2-3):245-57, 2004.

fuel operate. Staff Paper at 2-20. This “significant ‘under-monitoring’” exists despite the fact that air quality analyses show dramatically higher lead concentrations at monitors near sources compared with more remote monitors. ANPR at 42-43; Staff Paper at 2-69. As a result, EPA’s analysis significantly understates the extent of occurrences of relatively higher lead concentrations. Staff Paper at 2-20.

The ANPR acknowledges that “the limited size and spatial coverage of the current [lead] monitoring network constrains our ability to characterize current levels of airborne [lead] in the U.S.” ANPR at 43. Only 1.73 million people are “in proximity” of a lead air monitor included in the analysis. Staff Paper at 2-26. EPA also has not estimated the population levels in the vicinity of many of the largest industrial and commercial point sources. *See* ANPR at 43 (“The significant limitations of monitoring and emissions information constrain our efforts to characterize the size of at-risk populations in areas influenced by policy-relevant sources of ambient [lead].”). In addition, the ANPR notes “various [other] uncertainties and limitations associated with source information” in the inventory.” *Id.* at 43.

EPA’s location-specific case studies are seriously weakened by the lack of an adequate monitoring network and current, comprehensive monitoring data. The urban case studies are based on only a handful of monitors representing large and complex urban areas. For example, the case study for Cleveland is based on data from only six monitors, ANPR at 73, that cover a total area of 78 square miles.⁵⁸ The Chicago case study is based on data from eleven monitors, *id.*, covering approximately 227 square miles.⁵⁹ Thus EPA is modeling with a data density of less than one monitor for every 13 square miles – thereby introducing major uncertainties into the estimates of risk.

The secondary lead smelter case study is also handicapped by the lack of adequate monitoring data. EPA admits that emissions from this facility are not well accounted for, that prior air quality modeling and performance analysis was not done, and that there are “a substantially smaller number of Pb-TSP monitors in the area that could be used to evaluate and provide confidence in model performance.” ANPR at 72.

⁵⁸ U.S. Census Bureau, State and County Quickfacts (Ohio), *available at*: <http://quickfacts.census.gov/qfd/states/39/3916000.html> (last revised Jan. 2, 2008).

⁵⁹ U.S. Census Bureau, State and County Quickfacts (Illinois), *available at*: <http://quickfacts.census.gov/qfd/states/17/1714000.html> (last revised Jan. 2, 2008).

The serious gaps in the monitoring network suggest that EPA should assume that ambient lead levels are likely to be higher – and the areas of high ambient lead levels more extensive – than reflected in the exposure scenarios, and that (as further discussed at Part III, below) EPA should move quickly to repair and improve the monitoring network. It is noteworthy that several of these scenarios generated estimates for the median total blood lead level in children that are very significant. For example, at the current NAAQS, the upper 95th percentile of the population in several cities is predicted to exceed the CDC action level of 10 µg/dL. ANPR at 83. The primary lead smelter case study also shows that the 95th percentile of the population would exceed the CDC action level. This is particularly distressing both because these case studies likely underestimate the exposures to lead in these populations, and because the scientific consensus shows that lead is severely toxic at levels far below the action level.^{60 61}

More broadly, the location-specific urban case studies for current conditions show that at the 95th percentile, IQ loss associated with total lead is between 4.1-11.4 points. ANPR at 125. The true loss is likely to be substantially higher because of the weaknesses in the monitoring data that forms the basis of the case studies and because, as further discussed below, the upper five percent of children are improperly excluded from the analysis. As noted above, we agree with CASAC that “a population loss of 1-2 IQ points is highly significant from a public health perspective,” ANPR at 122, and further observe that such IQ loss has been associated with considerable economic losses from decreased educational achievement and earning potential. The uncertainties associated with the lack of adequate monitoring and the attendant possibility of significantly higher lead exposures and impacts than estimated require EPA to err on the side of a more stringent NAAQS. *See American Trucking Ass’ns. v. EPA*, 283 F.3d at 369-70, 378 (discussing ozone NAAQS).

c. EPA’s Analysis Improperly Focuses on Impacts to the 95th Percentile of the Population, Thus Obscuring Lead’s Threats to the Most Susceptible Individuals and Subpopulations.

⁶⁰ Recommendations of CDC’s Advisory Committee on Childhood Lead Poisoning Prevention. Interpreting and Managing Blood Lead Levels <10 µg/dL in Children and Reducing Childhood Exposures to Lead. MMWR 56(RR08):1-14, 2007.

⁶¹ Letter from Dr. Rogene Henderson, Chair, Clean Air Scientific Advisory Committee, to Administrator Stephen L. Johnson. Re: Clean Air Scientific Advisory Committee’s (CASAC) Review of the 2nd Draft Lead Human Exposure and Health Risk Assessments (September 27, 2007) (“CASAC Letter 2007b”).

EPA improperly presents risks in the scenarios only up to the 95th percentile of the population, and appears to propose to set a standard that is based on the 95th percentile. In other risk assessment and risk management contexts, EPA aims to protect the population up to the 99.9th percentile. For example, in a 2000 guidance document, EPA chose the 99.9th percentile of exposure as the default threshold of regulatory concern for pesticide residues in food.⁶² In justifying this choice, EPA stated:

As lower percentiles are considered, the estimated size of the population potentially exposed to levels greater than the [standard] increases... Moreover, the size of the exposed population potentially exceeding the [standard] at the 99th or 95th percentiles would be 10 and 50 times larger, respectively, than the number at the 99.9th percentile.⁶³

In fact, in establishing the 1978 lead NAAQS, EPA identified “a target of 99.5 percent of this population [young children] to be brought below the maximum safe blood [lead] level.” ANPR at 100. Over the intervening three decades, EPA’s approach seems to have become less sophisticated; the current document states “5 percent of the child study population at each case study location is estimated to have IQ loss above the 95th percentile estimates presented here, however, due to technical limitations of our modeling tools, it is not feasible at this point to reasonably predict the distribution of IQ loss levels for that top 5 percent.” ANPR at 87. This is not an adequate excuse for failing to protect the most susceptible and most exposed children. In Chicago alone, there are over 215,000 children ages five and under.⁶⁴ Five percent of the children at this case study location represents nearly 11,000 children under age five. Nationally, there were 20,417,636 children under the age of five in 2006.⁶⁵ By protecting children up to only the 95th percentile, EPA would be exposing more than a million children under the age of five to the adverse health impacts of lead, including potential IQ loss and attention deficit concerns. EPA has a statutory obligation to protect these children from the hazards of lead. EPA must consider impacts to the 99.5th percentile in setting the NAAQS. In fact, CHPAC recommends that EPA

⁶² U.S. EPA Office of Pesticide Programs, *Choosing a percentile of acute dietary exposure as a threshold of regulatory concern* (March 16, 2000), available at: <http://www.epa.gov/oppfead1/trac/science/trac2b054.pdf> (last visited Jan. 14, 2008).

⁶³ *Id.* at 33.

⁶⁴ See U.S. Census Bureau, State and County Quickfacts, available at: <http://quickfacts.census.gov/qfd/states/17/1714000.html> (last revised Jan. 2, 2008).

⁶⁵ U.S. Census Bureau, Population Estimates Program, available at: http://factfinder.census.gov/servlet/QTable?-ds_name=PEP_2006_EST&-qr_name=PEP_2006_EST_DP1&-geo_id=01000U.S. (last visited Jan. 10, 2008).

move to protect all children, not just children up to the 99.5th percentile. CHPAC Letter at 3. Even at a standard based on the 99.5th percentile five out of every thousand children would be exposed to toxic levels of lead.

In short, EPA's preliminary evaluation of the primary NAAQS necessary to protect public health has been compromised by incomplete data and a series of assumptions that tend to understate risks to the most exposed populations. Because EPA must "err on the side of caution" in setting NAAQS, *American Trucking*, 283 F.3d at 378 (discussing ozone NAAQS), and must assure that the final standard provides an adequate margin of safety for all sensitive sub-populations, these concerns underline the need for an event more stringent NAAQS.

D. EPA Has Not Sufficiently Analyzed the Secondary NAAQS.

The ANPR misrepresents CASAC's advice on the secondary NAAQS standard and fails to carry out CASAC's recommendations. The ANPR claims, "we concur with CASAC's conclusion that the Agency lacks the relevant data to provide a clear, quantitative basis for setting a secondary [lead] NAAQS that differs from the primary in indicator, averaging time, level or form." ANPR at 186. However, CASAC reached no such conclusion. Rather, CASAC stated that "at a minimum," the secondary NAAQS should be at least as low as the "*lowest-recommended* primary NAAQS." ANPR at 183; CASAC Letter 2007b at 7 (emphasis added). CASAC recommended that EPA carry out further research to inform its decision before reaching a conclusion regarding the secondary NAAQS. CASAC Letter 2007b at 7. EPA has not done any such research. *See* ANPR at 186.

EPA should promptly carry out the analysis recommended by CASAC, and absent some basis in such further analysis, should not set the secondary NAAQS at a level any higher than the lowest-recommended primary NAAQS – 0.02 µg/m³. *See* ANPR at 183; Staff Paper at 5-23, 5-40, 5-44 to 5-45.

E. EPA Has Provided No Basis for Departing From CASAC and Staff Paper Recommendations.

The ANPR offers no scientific basis for departing from the conclusions and recommendations of CASAC and the Staff Paper, only vague allusions to "other views" and "differing interpretations" on the propriety of maintaining the lead NAAQS. 72 Fed. Reg. at 71,532-53; 71,539; 71,542. The ANPR repeatedly fails

to provide the scientific or policy basis for the other less stringent options under consideration.⁶⁶

For instance, after summarizing CASAC's views, the Staff Paper recommendations, and the views of public commenters that the NAAQS must be reset to considerably more stringent levels, the ANPR makes note of unidentified "other views" as the basis for solicitation of comment on whether the current NAAQS should be retained or set at a level above that recommended by the Staff Paper and CASAC. ANPR at 161. The ANPR provides no basis for considering this option in light of CASAC's emphatic rejection of this option. *Id.*; see CASAC Review 2007b at 2. Similarly, the ANPR notes that the CDC action level of 10 µg/dL has "frequently been misinterpreted as a definitive toxicological threshold" and recognizes that levels below 10 µg/dL are associated with deleterious health effects. ANPR at 148. However, without explanation, the ANPR then solicits comment on using that action level as the basis for deriving the NAAQS in recognition of unidentified "differing views on this subject." *Id.*

The ANPR's attempt to present the options of revoking the NAAQS and maintaining the NAAQS at close to the present levels as worthy of equal consideration and analysis as the Staff Paper and CASAC recommendations, without independent support or justification, is inappropriate at this stage of the NAAQS review. It suggests a level of debate about whether – and how – to revise the NAAQS that is inconsistent with the scientific consensus on ambient lead's dangers. We urge EPA not to waste any further resources on "options" that the findings of CASAC, EPA's staff and the broader scientific community already conclusively establish as inconsistent with the mandates of Sections 108 and 109. Staff Paper at 5-44; ANPR at 114. EPA must discard these unlawful "options," and set the upper end of the range of policy options presented for the Administrator's consideration at the upper end of the range identified by the Staff Paper and CASAC.

a. The New NAAQS Review Process Lacks Scientific Integrity and Jeopardizes Public Health.

As the discussion above illustrates, the ANPR's consideration of policy options that radically depart from the Staff Paper's recommendations, as well as those of CASAC (and CHPAC), raises serious concerns regarding the scientific

⁶⁶ CASAC itself has raised many of these concerns. See *Citing Lead Standard, CASAC Will Urge New NAAQS Review Process*, Clean Air Report, Dec. 27, 2007 (Attached as Exhibit U).

integrity of the new NAAQS review process.⁶⁷ The hybrid nature of the current lead NAAQS review, which incorporates aspects of both the old and new EPA process for setting air quality standards, provides a disconcerting preview of the future of U.S. air quality regulation under the revised NAAQS review process. The Staff Paper, which would be abolished and replaced by the ANPR in future air quality rulemakings, unequivocally recommends strengthening the primary lead NAAQS from the current $1.5 \mu\text{g}/\text{m}^3$ standard to between 0.02 and $0.2 \mu\text{g}/\text{m}^3$. In comparison, the ANPR offers no clear recommendations for a revised air quality standard for lead. Instead, the ANPR presents a range of policy options, many of which have already been rejected both by the Staff Paper and CASAC as inadequate to protect public health with an adequate margin of safety, and even solicits comments on delisting lead as a criteria pollutant and revoking the lead NAAQS altogether.

For nearly three decades, EPA has relied on its staff scientists to independently translate the science on the health and environmental impacts of air pollution into a set of focused, concrete policy recommendations expressed in a Staff Paper. EPA has also allowed CASAC to review the Staff Paper, and thus to provide independent scientific input on the staff's recommendations, before issuing any rulemaking documents. These independent, thorough evaluations of the science and associated policy concerns are critical in updating the NAAQS to adequately reflect our growing understanding of the harmful effects of air pollution, and have worked remarkably well in improving air quality nationwide. While EPA has argued that replacing the Staff Paper with the ANPR will enable broader participation of the scientific community and the public early in the NAAQS review process, the current "hybrid" lead NAAQS review highlights the inadequacies of EPA's new approach. While the Staff Paper in the current review has connected the science on the adverse effects of lead to clear and transparent recommendations regarding a revised lead NAAQS level, the ANPR has failed to do so. This suggests that in future NAAQS rulemakings in which only an ANPR is issued, informed scientific opinion will no longer play the central role required by the Act. *See* 42 U.S.C. §§ 7408(a)(2), 7409(d)(2). The Act requires EPA to involve CASAC to the "maximum extent practicable within the time provided" in considering and shaping policy options for each NAAQS review. 42 U.S.C. § 7417(c) (discussing CASAC role in issuance of air quality criteria under Section 108(a)(2)); *see* 42 U.S.C. § 7409(d)(2)(C)(iv) (stating that CASAC shall advise EPA on public health, welfare, social, economic, or energy impacts of NAAQS policy options). EPA has previously involved CASAC to a greater degree in formulating the policy options in the last three decades by seeking its input in

⁶⁷ CASAC too raises these concerns. *See Citing Lead Standard, CASAC Will Urge New NAAQS Review Process*, Clean Air Report, Dec. 27, 2007.

developing and finalizing the Staff Paper (the counterpart to the new ANPR). The ANPR process provides for less involvement than before and thus veers from the Act's directive to involve CASAC to the "maximum extent practicable." We urge the Administrator to uphold the scientific integrity of the NAAQS review process by re-instating the issuance of the Staff Paper in all future NAAQS reviews and restoring the critical role of EPA scientific staff and independent science advisors at all stages of the NAAQS rulemaking.

In conclusion, because the current NAAQS has failed to protect the public from the harmful effects of lead exposure, let alone assured an "adequate margin of safety," the Clean Air Act requires EPA to replace those NAAQS with significantly more protective standards that incorporate the "latest scientific knowledge" on lead's threats to public health and welfare. 42 U.S.C. §§ 7408(a)(2), 7409(b); *see also, e.g., Lead Industries*, 647 F.2d at 1154-55 (noting Congress' observation that "more often than not the 'margins of safety' that are incorporated into air quality standards turn out to be very modest or nonexistent as new information reveals adverse health effects at pollution levels once thought to be harmless"). EPA must follow the law and the science as reflected in the recommendations of its staff and independent scientific advisors by adopting considerably more stringent NAAQS. We also note that the Act's mandate to account for scientific uncertainties and developing science, and to protect both the general population and all sensitive sub-populations with an adequate margin of safety, may well require EPA to set standards more stringent than those at the upper end of the ranges recommended by its staff and CASAC.

III. Additional Comments on Technical and Data Issues

The following comments address specific technical and data issues relevant to implementation of the considerably more stringent lead NAAQS the Clean Air Act requires EPA to adopt.

A. Indicator of the Standard

Although we generally agree with CASAC's recommendation that there are some significant technical advantages to moving toward low-volume Pb-PM₁₀ sampling, we recognize that there are also compelling public health arguments not to make that change at this time. *See* ANPR at 133-135. In particular, we are very concerned about point sources that produce significant larger-particle dust emissions. *See id.* at 135. These large particles, from sources such as smelters, are known sources of soil contamination that can pose significant risks to children. *Id.* Moving to Pb-PM₁₀ sampling would tend to underestimate emissions from

these important point sources. Because the current data do not support the derivation of a single scaling factor, changing the indicator would tend to protect some communities (i.e. urban communities with fine PM) more than others (i.e. those near large point source dust emitters). We urge EPA to prioritize research that will allow direct comparison of Pb-TSP and Pb-PM₁₀ in a variety of communities and near a range of lead sources. If one or more scaling factor can subsequently be derived, it would ultimately be beneficial to move toward implementing CASAC's recommendation. Therefore, we support the recommendation of the EPA staff paper to maintain Pb-TSP as the indicator at this time.

B. Averaging Time and Form

As discussed above, at Part II.B, EPA should measure NAAQS over an averaging time no longer than one month, with the maximum monthly mean calculated over a period no longer than one calendar year.

C. Monitoring Network Design

We recommend modifying the network design according to an option that combines elements of the second and fourth options suggested in the ANPR on page 192. We support utilizing the current PM₁₀ network in order to increase monitoring coverage without requiring a large investment in new monitoring stations. This approach is resource-efficient and will assure broad coverage of urban and rural areas across the country, thereby answering many questions about urban ambient dust. However, the approach should be supplemented with additional monitors downwind of major lead emitters. There are approximately 240 lead emitters in the NEI with emissions in excess of 1 ton per year (tpy). Staff Paper at Table 2-4. To the extent that the PM₁₀ network and existing Pb-TSP network do not include stations located near the maximum predicted concentrations for these sources, additional monitoring stations should be added at these locations.

D. Sampling Frequency

EPA also invites comments on sampling frequency within the monitoring network. ANPR at 194-94. We urge EPA to increase the sampling frequency in order to improve the quality of the data collection. Specifically, we recommend complete (daily) sampling and the use of a 24-hour sampling duration. We recommend against compositing of samples. It is important, especially for monitors located near point sources, that the sampling equipment be capable of

detecting short-term “spikes” in emissions, such as those that occur during upset or intermittent conditions. A short-term but large release of lead can result in a significant amount of material being deposited onto soil where it is then available to children and can pose a hazard. For example, a single facility may release one ton per year of lead, but may release most of that amount during a process that occurs only once a week, say on Monday nights. Such a situation could generate over 35 pounds of lead emissions each Monday night – surely a significant quantity to be deposited on nearby soil – yet this would be largely missed (or underestimated) with the current 1-in-6 day monitoring schedule, and would also be underestimated with a 1-in-3 day schedule, or with composite sampling.

E. Monitoring Periods for Non-Attainment and Attainment Determinations

In response to some of EPA’s suggested approaches to data handling, *see* ANPR at 195, we support the proposals to require only one period to demonstrate non-attainment, and three consecutive years of complete data to re-designate an area “attainment.” Incomplete monitoring periods should be used if they demonstrate non-attainment, but not to establish attainment.

F. Remediation of Lead in Soil and Dust

As noted above, lead levels in soil and dust are an important exposure pathway (particularly for children) that EPA must account for in revising the NAAQS. Abatement of soil lead contamination in areas known to be contaminated from airborne sources is technically feasible, and has been proven to reduce childhood blood lead levels.⁶⁸ Regulatory programs that focus on abatement of lead paint alone would not be able to accomplish the reductions in blood lead levels that could be achieved through more comprehensive efforts to reduce airborne and soil lead and to reduce overall exposures through these pathways.⁶⁹

IV. Conclusion

For the reasons above, EPA must abandon any consideration of revoking the NAAQS for lead or of maintaining the current NAAQS, and instead focus its

⁶⁸ Lanphear BP, Succop P, Roda S, Henningsen G. The effect of soil abatement on blood lead levels in children living near a former smelting and milling operation. *Public Hlth Reports* 118:83-91, 2003.

⁶⁹ Mielke HW, Reagan PL. Soil is an important pathway of human lead exposure. *Environ Health Perspect* 106(suppl 1):217-229, 1998.

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efforts on adopting the more protective NAAQS the Clean Air Act mandates. A significant strengthening of the NAAQS is long overdue. We hope EPA will move to establish an aggressive NAAQS, as recommended by CASAC, CHPAC, and EPA staff, and to protect public health with an adequate margin of safety, especially the health of those who are most vulnerable and most exposed to lead's dangers.

Sincerely,



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