



UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON D.C. 20460

OFFICE OF THE ADMINISTRATOR
SCIENCE ADVISORY BOARD

March xx, 2007

EPA-CASAC-07-00x

Honorable Stephen L. Johnson
Administrator
U.S. Environmental Protection Agency
1200 Pennsylvania Avenue, NW
Washington, DC 20460

Subject: Clean Air Scientific Advisory Committee's (CASAC) Review of the 1st Draft
Lead Staff Paper and Draft Lead Exposure and Risk Assessments

Dear Administrator Johnson:

The Clean Air Scientific Advisory Committee (CASAC or Committee), augmented by subject-matter-expert Panelists — collectively referred to as the CASAC Lead Review Panel (Lead Panel) — completed its review of the Agency's 1st Draft Lead Air Quality Criteria Document (AQCD) in September 2006 (EPA-CASAC-06-010). On December 7, 2006, Mr. Marcus Peacock, the EPA Deputy Administrator, issued a memorandum providing his final decisions on revisions to the process by which the National Ambient Air Quality Standards (NAAQS) are reviewed. In this memo, Deputy Administrator Peacock directed that this revised NAAQS review process should begin with the current, ongoing review of the NAAQS for Lead. (See URLs: http://www.epa.gov/ttnnaqs/memo_process_for_reviewing_naaqs.pdf and http://www.epa.gov/ttnnaqs/naqs_process_report_march2006_attachments.pdf).

On February 6–7, 2007, the CASAC's Lead Panel conducted a peer review of EPA's *Draft Review of the National Ambient Air Quality Standards for Lead: Policy Assessment of Scientific and Technical Information* (1st Draft Lead Staff Paper, December 2006) and a related draft technical support document, *Lead Human Exposure and Health Risk Assessments and Ecological Risk Assessment for Selected Areas: Pilot Phase, Draft Technical Report* (Draft Lead Exposure and Risk Assessments, December 2006). The CASAC roster is found in Appendix A of this report, and the Lead Panel roster is attached as Appendix B. The charge questions provided to the Lead Panel by EPA staff are contained in Appendix C to this report, and supplemental tables from Lead Panel members that furnish additional analyses for the primary Lead NAAQS are found in Appendix D. Panelists' individual review comments are provided in Appendix E **[not attached to this V1-1 Working Draft]**.

1 At the February 6–7 public meeting, the CASAC expressed serious concerns both about
2 the EPA documents to be reviewed and the Agency’s proposed rulemaking schedule for the Lead
3 NAAQS, as follows:

- 4 • 1st Draft Lead Staff Paper had no staff-derived options for keeping or altering the current
5 lead standard.
- 6 • The Draft Lead Exposure and Risk Assessments document did not have a full discussion
7 of the risk associated with different options for keeping or altering the lead standard. The
8 CASAC judges that, while the latter document represented a good first effort, it was
9 nowhere near completion.
- 10 • Under the Agency’s new NAAQS review process, EPA’s Staff Paper will no longer be
11 prepared but will be replaced by a Policy Assessment (PA) for Lead, to be issued in the
12 form of an Advance Notice of Proposed rulemaking (ANPR). However, the Agency’s
13 proposed schedule for the Lead NAAQS calls for the completion of the Lead Exposure
14 and Risk Assessments document *after* the PA for Lead is issued via the ANPR. Thus,
15 CASAC would not be given an opportunity to review a more fully-developed, second-
16 draft version of the Risk/Exposure Assessment (RA) prior to the ANPR, so that the PA
17 would not be informed by the science assessments of the CASAC.

18
19 Subsequent to the February 6–7 meeting of the lead Panel, Agency officials, managers
20 and staff held administrative discussions with the members of the CASAC to learn directly from
21 them their specific concerns with the scheduled for the Lead standards and the revised NAAQS
22 review in general. The CASAC looks forward to receiving the EPA’s modified timeline, both
23 for the generic NAAQS review and the Lead NAAQS in particular.

24
25 The CASAC used the scientific information found in the Agency’s Final Lead AQCD,
26 which was also reviewed by the Committee, in its review of EPA’s 1st Draft Lead Staff Paper
27 and the Draft Lead Exposure and Risk Assessments document. The CASAC’s recommendations
28 and the associated scientific basis for these recommendations are presented below. *The Lead*
29 *Panel was in consensus that lead should not be de-listed as a criteria air pollutant, as defined by*
30 *the Clean air Act, for which a NAAQS is established, and that both the primary and secondary*
31 *air standards should be lowered substantially.* It was also suggested that future lead sampling be
32 conducted with low volume PM₁₀ samplers rather than with total suspended particulate (TSP)
33 samplers, and that the averaging time be reduced from seasonal to monthly.

34
35 Reasons for those conclusions are given below.

36 37 **Introduction**

38 Over the past three decades, blood lead (PbB) levels in the U.S. population have
39 plummeted (1). This decline was largely due to the elimination of leaded gasoline (2). In 1976,
40 the Consumer Product Safety Commission restricted the allowable level of lead in residential
41 paint to 0.06 percent (600 ppm) (3). Lead solder used in canned foods was reduced, from over
42 90% in 1978 to less than 5% in 1988 (4). Finally, there was a decline in housing that contained
43 lead-based paint (5). Although it is difficult to quantify the PbB decline attributable to specific

1 sources, the 1978 NAAQS for Lead was undoubtedly one of the major reasons for the rapid and
2 widespread decline in PbB levels in the U.S. population (6).

3
4 Despite the dramatic decline in environmental lead exposure, lead toxicity remains a
5 major public health problem. Environmental lead exposure in children has been associated with
6 an increased risk for reading problems, school failure, Attention Deficit Hyperactivity Disorder
7 (ADHD), delinquency and criminal behavior (6–10). Moreover, there is no evidence of a
8 threshold for the adverse consequences of lead exposure; studies show that the decrements in
9 intellectual function are proportionately greater at PbB levels < 10 µg/dl, the level considered
10 acceptable by the Centers for Disease Control (11–14). Among U.S. children, eight to fifteen
11 years old, those in the highest quintile (> 2 µg/dl) of lead exposure were four times more likely
12 to have doctor-diagnosed ADHD (11).

13
14 Lead's effects extend beyond childhood. In adults, lead exposure is a risk factor for some
15 of the most prevalent diseases or conditions of industrialized society, including cardiovascular
16 disease and renal disease (16–20). There is also compelling evidence that the risk for mortality
17 from stroke and myocardial infarction is increased at PbB levels below 10 µg/dl, which is
18 considerably lower than the levels considered acceptable for adults (19). Finally, although less
19 definitive, there is also evidence that lead exposure during pregnancy is a risk factor for
20 spontaneous abortion or miscarriage at PbB levels < 10 µg/dl (21).

21 22 **Scientific Basis for Continuing or De-listing the Lead NAAQS**

23 The CASAC considered the implications of the state of the science on the protection of
24 public health from exposure to lead in the environment. One of these implications relates to the
25 question of whether the current science continues to support the need for lead to be listed as a
26 criteria air pollutant for which a NAAQS is established, or might warrant the de-listing of lead,
27 as presented as a policy option in the 1st Draft Lead Staff Paper. In addressing this question, the
28 CASAC examined several scientific and public health issues that are considered essential in
29 determining whether or not a pollutant such as lead should be de-listed or maintained as a criteria
30 air pollutant.

- 31
- 32 1. *Do new scientific data accumulated since EPA's promulgation of the current lead*
33 *NAAQS of 1.5 ug/m³ in 1978 suggest that science previously overstated the toxicity of*
34 *lead?* Here, the Committee's answer clearly is No. The data accumulated over the past
35 decade make it apparent that adverse health effects on both humans and other species
36 appear at PbB levels and environmental exposures well below those previously thought to
37 pose risks. Indeed, if anything, these new data suggest that science previously
38 *understated* the toxicity of lead.
39
 - 40 2. *Have past regulatory and other controls on lead emissions reduced the PbB levels in*
41 *human populations so far below levels of concern as to suggest there is now a significant*
42 *margin of safety inherent in those PbB levels?* Again, the answer here is No. The Nation
43 can take great pride in the extent to which exposures to lead have been reduced, leading
44 to laudable decreases in PbB levels to an average approaching 2 µg/dl. However, there
45 remains a significant segment of the population with blood-lead levels above 5 µg/dl —
46 and some even above 10 µg/dl — and scientific evidence supports the contention that

1 these PbB levels do not provide a margin of safety. In fact, this evidence suggests these
2 blood lead levels below 5 µg/dl are associated with unacceptable adverse effects.
3

- 4 3. *Have the activities that produced emissions of lead in the past changed their practices to*
5 *such an extent that society can have confidence that emissions will remain low even in*
6 *the absence of NAAQS controls?* Here, the CASAC concludes that the answer is again
7 No. While there have been major advances in reducing emissions from leaded gasoline,
8 industrial and other activities, even the current emissions levels in some operations
9 produce unacceptable environmental exposures once the concentrations of lead in
10 environmental media equilibrate. The CASAC concludes that past success in reducing
11 PbB levels in the population are due in large part to NAAQS controls, and that in the
12 absence of such controls there is a significant possibility that blood-lead levels would
13 begin to rise again.
14
- 15 4. *Are airborne levels of lead sufficiently low throughout the United States that future*
16 *regulation of lead can be effectively accomplished by regulation of products, soil and/or*
17 *water?* CASAC concludes that the answer to this question is No. While airborne lead
18 levels have been reduced throughout much of the United States, airborne lead remains a
19 primary vehicle for movement of lead between different environmental compartments.
20 While control of airborne lead is not sufficient by itself to control exposure to lead, it is
21 an essential component of a successful control strategy. Maintaining an appropriate
22 NAAQS for lead is considered by CASAC to be an essential component of a national
23 program to reduce the ongoing adverse effects of lead in children, adults and in the
24 ecosystem.
25
- 26 5. *If lead were de-listed as a criteria air pollutant, would it be appropriately regulated*
27 *under the Agency's Hazardous Air Pollutants (HAP) program?* The answer is again No.
28 The HAP program regulates according to use of maximum achievable control technology
29 (MACT) and is appropriate for point sources. However, the most widespread source of
30 airborne lead throughout the nation is the historically-deposited lead along roadways.
31 Thus, this source of airborne lead could not be regulated under the HAP program.
32

33 *As a result of our answers to these scientific and public health issues, the CASAC*
34 *concludes that the existing state of science is consistent with continuing to list ambient lead as a*
35 *criteria air pollutant for which a fully-protective NAAQS is required.*
36

37 **Additional Analyses to Inform a Primary (Health-Based) NAAQS for Lead**

38 Despite the dramatic decline in air lead levels and population blood-lead levels following
39 the phase-out of leaded gasoline, lead toxicity remains a major public health problem. As
40 discussed above, there is increasing evidence of lead-induced toxicity at the lowest levels of
41 exposure, including IQ deficits in children (11–14), ADHD (11) and cardiovascular disease (6–
42 19). Although less definitive, there is evidence that lead exposure is a risk factor for spontaneous
43 abortion, renal disease and immunologic effects (20–21).
44

1 Although relatively few counties in the United States are out of compliance, the greatest
2 benefit to public health will be realized by broad reductions in air lead levels across the U.S.
3 population because:

- 4 1. The adverse consequences are proportionately greater at lowest increments of lead
5 exposure;
- 6 2. Lead exposure is cumulative; and
- 7 3. Airborne lead exposure, in contrast with exposure to lead-based paint, is more widely
8 dispersed. Thus, reducing exposure from air lead will broadly reduce population blood
9 lead levels.

10
11 In 1978, the EPA established a NAAQS of $1.5 \mu\text{g}/\text{m}^3$ to ensure that 99.5% of the public
12 did not exceed a blood-lead level of $15 \mu\text{g}/\text{dl}$. The existing lead NAAQS was instrumental in
13 producing dramatic reductions in air lead and blood-lead concentrations over the last 30 years
14 due to phase out of leaded gasoline use. However, this lead NAAQS is totally inadequate for
15 assuring the necessary reduction of lead exposures in sensitive U.S. populations below those
16 current health hazard markers identified by a wealth of new epidemiological, experimental and
17 mechanistic studies. Consequently, *it is the CASAC's judgment that the NAAQS for Lead should*
18 *be lowered to fully-protect both the public health for children and adult populations.*
19

20 The EPA pilot-phase human health risk assessment focused on three case study locations
21 (*i.e.*, primary lead smelter, secondary lead smelter, and near-roadway urban). While the case
22 study approach undertaken in the risk assessment is enlightening and provides a potentially
23 useful framework for understanding lead exposure for some discrete populations within the U.S.,
24 there are some additional considerations and analyses we strongly feel could help inform a
25 scientifically defensible NAAQS for lead. In particular, the CASAC believes that the risk
26 assessment could be better informed with a “population based” risk assessment to supplement
27 the current case study approach. A population-based risk assessment would include two key
28 components:

- 29 1. A quantitative description of the relationship between concentrations of lead in national
30 ambient air and distributions of resulting blood lead levels; and
- 31 2. A quantitative description of the relationship between blood lead levels and impacts on
32 IQ.
33

34 For each of these factors, the EPA should fully and quantitatively evaluate the associated
35 uncertainties. For instance, the EPA should evaluate how the first factor has been informed by
36 historical lead exposures in the U.S. (resulting from a fairly homogeneous source of lead via
37 leaded gasoline) and how it might apply to current conditions in the U.S. now that lead exposures
38 are not dominated by such a homogeneous and ongoing source of lead in air. For the second
39 factor, the EPA should fully evaluate the uncertainties associated with the relationship between
40 blood lead levels and IQ decrements.
41

42 There are multiple ways in which EPA could conduct a population-based analysis, and
43 we illustrate some possibilities in the next section of this letter. Please note that this work does
44 not represent a complete analysis on the part of the Committee; rather, it is meant to illustrate the

1 CASAC's thinking in this area. It will be important for EPA to consider these approaches and to
2 fully evaluate their pros, cons, and associated uncertainties. An adequately comprehensive
3 analysis should characterize the uncertainty, preferably in a quantitative manner, in two key
4 areas: 1) the relationship between a change in the NAAQS for lead and the distribution of
5 population blood lead levels; and 2) the relationship between blood lead levels and the risk of
6 adverse health effects. This thoughtful review will help highlight the strengths and weaknesses
7 of the available approaches and help to elucidate a NAAQS that is scientifically defensible and
8 health protective
9

10 The CASAC considered three separate, but related analytical approaches as examples to
11 be considered in deriving an acceptable range of levels, on the basis of the scientific evidence,
12 for setting a new NAAQS level for ambient lead. Each of these approaches (referred to as A, B
13 and C) uses existing data to derive estimated risk of blood lead not exceeding a given value with
14 consideration that 99.5% of the population of children would be below that level. The
15 approaches are all based on risk for the entire population (rather than selected sites).
16

17 The first approach (A) uses a modified empirical-deterministic approach that is
18 essentially the same approach used in previous Lead NAAQS documents, except, because of the
19 recent additional scientific evidence, the level of blood lead shown to be harmful has dropped
20 from 30 µg/dl (in 1978) to 15 µg/dl (in 1990) to less than 5 µg/dl (Final Lead AQCD). Therefore
21 the derivation of the ranges of exposure from the current standard set in 1978 must be lowered
22 by at least a factor of six. (See, in particular, the individual review comments of Lead Panel
23 members Dr. Paul Mushak and Dr. Ian von Lindern in Appendix E.)
24

25 As derived in several attached comments, the CASAC determined what selected range of
26 ambient air lead levels should be a "not-to-be-exceeded level," the range of which is dependent
27 not only on the 99.5% protective level but also on several additional key parameters. These
28 include the *maximal acceptable blood level* (range between 2.5–5.0 µg/dl but might be
29 considered lower); an *appropriate geometric standard deviation* (range 1.3–2.0); *non-air*
30 *background* (1.0–1.4 µg/dl or lower range should be considered); and the *slope factor* for the
31 relation between air lead and blood lead for levels of blood lead below 10 µg/dl. (Candidate
32 values considered were 2.0 m³/dl used in 1978, 5.0 m³/dl used by the World Health Organization
33 [WHO] in 2000, 10.0 m³/dl noted in recent studies, and 20.0 m³/dl as a maximum.)
34

35 These considerations are summarized in tables found in Appendix D and also contained
36 in the individual review comments from various Panel members attached as Appendix E. It is
37 clear that, depending on the slope factor selected between 5 and 20 m³/dl, the estimate of PbB
38 from various air lead levels varies by a factor of four (see Table 1 in Appendix D). From the
39 Lanphear 2005 analysis, the linear estimate of IQ loss associated with PbB below 7.5 µg/dl, the
40 CASAC estimated that, over the range of PbB from 0.5–4.0 µg/dl (an eight-fold range), the loss
41 of IQ would similarly increase from 1.5 to 11.6 IQ points (see Table 2 in Appendix D).
42

43 *The CASAC believes that a population loss of 1.5 IQ points is highly significant from a*
44 *public health perspective.* Therefore, the CASAC has considered this level as the change in IQ
45 not to be exceeded. Depending upon the slope factor selected, this results in a range of 0.025–

1 0.200 $\mu\text{g}/\text{m}^3$ (*i.e.*, about a 7.5- to 60-fold reduction) as the estimated air lead levels to consider
2 under approach A, in combination with the use of data on the relation between PbB and IQ.
3

4 Option B is a “top-down” approach. That is, instead of estimating the effect of inhalation
5 alone, the effect of air lead on deposition into dust, food, *etc.* and the uptakes from those
6 pathways, an epidemiologic approach is used to derive an adjusted slope factor taking into
7 account all pathways between air lead and blood lead. This is based on the changes in blood lead
8 observed when lead began to be phased-out of gasoline. This analysis relies on the results of
9 Schwartz and Pitcher (22).

10
11 The Schwartz and Pitcher analysis showed that in 1976, the midpoint of the National
12 Health and Nutrition Examination Survey (NHANES) II, gasoline lead was responsible for 9.1
13 $\mu\text{g}/\text{dl}$ of blood lead in children. This is based on their coefficient of 2.14 $\mu\text{g}/\text{dl}$ per 100 metric
14 tons (MT) per day of gasoline lead use, and usage of 426 MT/day in 1976. This provides the
15 first part of the CASAC’s estimate, a change in blood lead resulting from a change in air lead
16 (via the elimination of lead from gasoline in the late 1980s). Between 1976 and the elimination
17 of lead from gasoline, air lead concentrations in U.S. cities fell a little less than 1 $\mu\text{g}/\text{m}^3$. These
18 two facts imply a ratio of 9–10 $\mu\text{g}/\text{dl}$ per $\mu\text{g}/\text{m}^3$ reduction in air lead, taking all pathways into
19 account. Under this scenario, a reduction of mean air lead concentrations of 0.1 $\mu\text{g}/\text{m}^3$ could be
20 expected to produce a further reduction in average blood lead concentrations of 0.9–1.0 $\mu\text{g}/\text{dl}$.
21 Assuming a slope of three IQ points per $\mu\text{g}/\text{dl}$ reduction in blood lead, which is indicated by the
22 studies of low level lead exposure (13), this further reduction would be expected to raise the
23 average IQ of children in U.S. cities by approximately three IQ points, a significant positive
24 health impact. . Put another way, the derivation above empirically justifies the use of the slope
25 factor of 10 in approach A and C, and the resulting estimates that an air quality standard of 0.11
26 $\mu\text{g}/\text{m}^3$ (that is, a 13-fold reduction) would be required to keep 99.5% of the children below a
27 blood lead of 5 $\mu\text{g}/\text{dl}$.
28

29 In approach C a direct estimate of the geometric mean PbB level for a 99.5% level of 2.5
30 $\mu\text{g}/\text{dl}$ ranges between 0.42–0.74 $\mu\text{g}/\text{dl}$ for a range of geometric standard deviations (GSD) of
31 2.0–1.6 (see Comments of Dr. Bruce Lanphear dated February 19, 2007 in Appendix E). In
32 addition, between the ranges of 1–7.5 $\mu\text{g}/\text{dl}$ in the linear model for both concurrent and lifetime
33 exposure suggests a three-point decrement in IQ for each unit change in PbB (see Table 4 in Dr.
34 Lanphear’s comments in Appendix E). The log-linear model varies from 2–0.5 IQ points over
35 the same range of blood values below 7.5 $\mu\text{g}/\text{dl}$. Clearly, this approach, although more directly
36 measured from the existing data than the Approach A above, provides a similar range of IQ
37 effects, depending upon the choice of slope factors considered, and suggests that even lower
38 levels of air lead may be required to reduce IQ effects in children less than 1.5 points attributable
39 to air lead.
40

41 *These three approaches provide similar results. Pending a more thorough analysis and,*
42 *in particular, a careful evaluation of uncertainties in these analyses, the above population-*
43 *based, scientific analyses indicate the need for a reduction of the current primary (health-based)*
44 *lead NAAQS of from about 7.5- up to 60-fold to provide an adequate margin of safety for*
45 *99.5% of the sensitive pool of the U.S. population.*
46

1 **Possible Revision to Lead Indicator from TSP to Low Volume PM₁₀**

2 As revisions to the level, form and averaging time of the Lead NAAQS are considered, it
3 may also be timely for EPA to consider revisions to the indicator. Currently, Lead NAAQS
4 monitoring is predominantly based on atomic absorption analysis of fiberglass filters run on hi-
5 volume total suspended particulate (TSP) samplers. Most other TSP sampling was discontinued
6 after PM₁₀ standards were promulgated in 1987. TSP samplers capture particles with an
7 imprecise and variable upper particle cut size in the range of approximately 30 to 50 microns on
8 fiberglass filters which are not well-suited for analysis by inexpensive, multi-elemental surface
9 beam techniques like particle-induced X-ray emission (PIXE) or X-ray fluorescence (XRF).
10 Consequently TSP sampling by imprecise samplers is primarily conducted only for lead analysis
11 and these filters are rarely analyzed for other species.

12
13 If lead NAAQS monitoring was based on (low volume) PM₁₀ sampling on Teflon filters,
14 the resulting data would be highly correlated with TSP lead, as shown in the current Policy
15 Assessment Document, but would have substantially improved sampling precision. Other
16 advantages of low volume PM₁₀ sampling include:

- 17 1. Focus on the most biologically-relevant particles deposited in the thoracic region;
- 18 2. Larger spatial-scale representativeness for population exposures to monitored particles
19 which remain airborne longer;
- 20 3. Could utilize more widespread PM₁₀ and “air toxics” metals sampling networks, leading
21 to collection of more data at lower costs;
- 22 4. Potential for inexpensive multi-elemental analysis by XRF or PIXE would provide useful
23 supplemental metals information for health effects studies and source apportionment;
- 24 5. Potential for automated sequential PM₁₀ samplers (not available for TSP) would be
25 especially useful if sampling frequency is increased from once every six days; and
- 26 6. Weighing filters would provide useful information on PM₁₀ mass; and, if collocated with
27 PM_{2.5} Federal Reference Methods (FRM), could provide needed information on PM_{10-2.5}
28 mass and speciation.

29
30 Reasons for retaining the current TSP lead indicator include preservation of a long-term
31 historical record at some sites, and inclusion of very coarse (> 10 micron particle) lead which
32 may deposit in upper sections of the respiratory system and ultimately be ingested. Such coarse
33 particles might be missed by PM₁₀ samplers. Presumably a downward scaling of the level of the
34 Lead NAAQS could accommodate the loss of very coarse mode lead, and some short period of
35 concurrent PM₁₀ and TSP lead sampling could help develop site-specific scaling factors at sites
36 with highest concentrations where long-term historical records are important.

37
38 *Given the advantages of using PM₁₀, CASAC recommends that the Agency consider*
39 *revising the lead indicator to utilize low volume PM₁₀ sampling, and also develop equivalent*
40 *analytical methods to allow use of XRF and Inductively-Coupled Plasma Mass Spectrometry*
41 *(ICP-MS) analysis.*

1 **Possible Revision to Averaging Time Used for the Lead NAAQS**

2 A second change that should be considered with a change in the lead NAAQS is to use a
3 different averaging time. Currently, quarterly averaging is used. However, studies suggest that
4 blood levels respond at shorter time scales than would be captured completely by quarterly
5 values. Here, the CASAC suggests that the Agency consider *monthly* averaging in addition to
6 quarterly.

7
8 One consideration involved in using a shorter averaging period is sampling frequency.
9 Currently, many of the samplers operate with sampling frequencies less than once per day, and
10 as infrequently as every sixth day. In the most extreme case, as few as four samples may be
11 involved in determining a monthly average (assuming no samples are considered invalid). This
12 would make the average susceptible to anomalously high events. On the other hand, this may
13 motivate more frequent sampling in those areas near the standard, which would increase the
14 protection of public health and significantly reduce the impact of a single high period. One
15 could also consider having the standard based on the second highest monthly average, a form
16 that appears to correlate well with using the maximum quarterly value.

17
18 *CASAC recommends adopting monthly averaging as being more protective of human*
19 *health in light of the response of blood levels that occur at sub-quarterly time scales, and further*
20 *recommends that the most protective form would be the highest monthly average in a year. An*
21 *area could choose to increase sampling frequency to make the monthly average less susceptible*
22 *to more extreme events. Such a change is consistent with either using TSP or PM₁₀ sampling.*

23
24 **Secondary (Welfare-Based) NAAQS for Lead**

25 Chapter 6 of the 1st Draft Lead Staff Paper and Chapter 7 of the “Pilot Phase” Draft Lead
26 Exposure and Risk Assessments technical support document present compelling scientific
27 evidence that current atmospheric lead concentrations and deposition — combined with a large
28 reservoir of historically-deposited lead in soils, sediments and surface waters — continue to
29 cause adverse environmental effects in aquatic and/or terrestrial ecosystems, especially in the
30 vicinity of large emission sources. These effects persist in some cases at locations where current
31 airborne lead concentrations are below the levels of the current primary and secondary lead
32 standards. *Thus, from an environmental perspective, there are convincing reasons to both retain*
33 *lead as a regulated criteria air pollutant and to lower the level of the current secondary*
34 *standard.*

35
36 Since concentrations of historically deposited lead in soils throughout the U.S. (averaging
37 0.5 to 4 g/m² of land area) are changing only slowly — with a half-life exceeding a century —
38 these concentrated deposits of lead are expected to remain accessible for exchange with the
39 atmosphere and the rest of the biosphere into the foreseeable future. Fires, changes in land use,
40 or climatic events such as regional dust storms could mobilize significant quantities of lead that
41 would be harmful both to human health and ecosystems downwind. This potential for harm is
42 not adequately recognized in the 1st Draft Lead Staff Paper and the Draft Lead Exposure and
43 Risk Assessments technical support document, but is a concern that warrants careful continued
44 monitoring in the future.

45

1 In addition, while neither the 1st Draft Lead Staff Paper Lead Exposure nor the Draft Risk
2 Assessments document provide a clear quantitative basis for identifying a specific lower level at
3 which a more protective secondary (welfare- or environmental-based) Lead NAAQS should be
4 set, there are no reasons to expect that humans are uniquely sensitive to lead pollution among the
5 millions of animal and plant species. *Therefore, at a minimum, the level of the secondary Lead*
6 *NAAQS should be at least as low as the lowest-recommended primary Lead standard. The EPA*
7 *is also encouraged to identify the necessary funds to support needed continuing research on the*
8 *ecological effects of airborne lead pollution and to develop alternative secondary standards such*
9 *as critical loads for lead, which may be different from primary standards in indicator, averaging*
10 *time, level or form.*

11
12 The CASAC continues to be pleased to provide advice to you concerning the scientific
13 basis for the setting of the primary and secondary Lead NAAQS. In addition, the CASAC looks
14 forward to continued dialog with Agency officials and staff aimed at improving EPA's NAAQS
15 review process in a manner that enhances the efficiency of the process while maintaining its
16 integrity and adherence to the stipulations of the Clean Air Act. Finally, the Committee also
17 looks forward to reviewing the 2nd draft of the Agency's Lead Risk/Exposure Assessment. As
18 always, we wish Agency staff well in this important task.

19
20 Sincerely,

21
22 /signed/

23
24 Dr. Rogene Henderson, Chair
25 Clean Air Scientific Advisory Committee
26

27
28 Appendix A – Roster of the Clean Air Scientific Advisory Committee

29 Appendix B – Roster of the CASAC Lead Review Panel

30 Appendix C – Agency Charge to the CASAC Lead Review Panel

31 Appendix D – Tables on Analyses for the Primary Lead NAAQS

32 Appendix E – Review Comments from Individual CASAC Lead Review Panel Members **[not**
33 **attached to this V1-1 Working Draft]**

34
35
36 **References**

- 37 1. Pirkle JL, Kaufmann RB, Brody DJ, Hickman T, Gunter EW, Paschal DC. Exposure of the
38 U.S. population to lead, 1991–1994. *Environ Health Perspect* 1998;11:745–50.
- 39 2. Mahaffey KR, Annet JL, Roberts J, Murphy RS. National estimates of blood lead levels:
40 United States, 1976–1980. Association with selected demographic and socioeconomic
41 factors. *New Engl J Med* 1982;307:573–579.

- 1 3. Committee on Toxicology, Assembly of life Sciences, National Research Council.
2 Recommendations for the prevention of lead poisoning in children. *Nutrition Rev*
3 1976;34:321–327.
- 4 4. Bolger PM, Carrington CD, Capar SG, Adams MA. Reductions in dietary lead exposure in
5 the United States. *Chem Spec Bioavail* 1991;3:31–36.
- 6 5. Jacobs DR, Friedman W, Clickner RP, et al. The prevalence of lead-based paint hazards in
7 U.S. Housing. *Env Health Perspect* (in press).
- 8 6. Needleman HL, Gunnoe C, Leviton A, et al. Deficits in psychologic and classroom
9 performance of children with elevated dentine lead levels. *N Engl J Med* 1979;300:689–95.
- 10 7. Needleman HL, Schell A, Bellinger D, Leviton A, Allred EN. The long-term effects of
11 exposure to low doses of lead in childhood: An 11-year follow-up report. *N Engl J Med*
12 1990;322:83–88.
- 13 8. Denno D. *Biology and Violence*. New York: Cambridge University Press, 1990.
- 14 9. Needleman HL, Reiss JA, Tobin MJ, Biesecker GE, Greenhouse JB. Bone lead levels and
15 delinquent behavior. *JAMA*. 1996;275:363–369.
- 16 10. Dietrich K, Ris M, Succop P, Berger O, Bornshein R. Early exposure to lead and juvenile
17 delinquency. *Neurotox Teratol* 2001;23:511–518.
- 18 11. Braun J, Kahn RS, Froehlich T, Auinger P, Lanphear BP. Exposures to environmental
19 toxicants and attention deficit hyperactivity disorder in U.S. children. *Environ Health*
20 *Perspect* 2006;114:1904–1909.
- 21 12. Canfield RL, Henderson CR, Cory-Slechta DA, Cox C, Jusko TA, Lanphear BP.
22 Intellectual impairment in children with blood lead concentrations below 10 micrograms
23 per deciliter. *N Engl J Med* 2003;348:1517–1526.
- 24 13. Lanphear BP, Hornung R, Khoury J, et al. Low-level Environmental Lead Exposure and
25 Children’s Intellectual Function: An International Pooled Analysis. *Environ Health*
26 *Perspect* 2005;113:894–899.
- 27 14. Kordas K, Canfield RL, Lopez P, et al. Deficits in cognitive function and achievement in
28 Mexican first-graders with low blood lead concentrations. *Environ Res*. 2006;100:371–386.
- 29 15. Tellez-Rojo MM, Bellinger DC, Arroyo-Quiroz C, et al. Longitudinal associations between
30 blood lead concentrations lower than 10 µg/dl and neurobehavioral development in
31 environmentally exposed children in Mexico City. *Pediatrics*. 2006;118:e323–330.
- 32 16. Schwartz J. Lead, blood pressure, and cardiovascular disease in men. *Arch Environ Health*
33 1995;50:31–37.
- 34 17. Nash D, Magder L, Lustberg M, Sherwin RW, Rubin RJ, Kaufmann RB, Silbergeld EK.
35 2003. Blood lead, blood pressure, and hypertension in perimenopausal and postmenopausal
36 women. *JAMA* 289:1523–1532.
- 37 18. McDonald JA Potter NU. Lead’s legacy? Early and late mortality of 454 lead- poisoned
38 children. *Arch Environ Health* 1996;51:116–121.
- 39 19. Menke A, Muntner P, Batuman V, Silbergeld EK, Guallar E. Blood Lead Below 0.48
40 µmol/L (10 µg/dl) and mortality among U.S. Adults. *Circulation* 2006;114:1388–1394.

Working Draft V1-1 Dated 03/07/2007 – Do Not Cite or Quote

- 1 20. Lin JL, Lin-Tan DT, Hsu KH, Yu CC. 2003. Environmental lead exposure and progression
2 of chronic renal diseases in patients without diabetes. *N Engl J Med* 348:277–286.
- 3 21. Borja-Aburto VH, Hertz-Picciotto I, Rojas Lopez M, Farias P, Rios C, Blanco J. Blood
4 lead levels measured prospectively and risk of spontaneous abortion. *Am J Epidemiol*
5 1999;150:590–597.
- 6 22. Schwartz J, Pitcher H. The relationship between gasoline lead and blood lead in the United
7 States. 1989 *J. Official Stat.* 5: 421-431.

Appendix A – Roster of the Clean Air Scientific Advisory Committee

U.S. Environmental Protection Agency Science Advisory Board (SAB) Staff Office Clean Air Scientific Advisory Committee (CASAC)

CHAIR

Dr. Rogene Henderson, Scientist Emeritus, Lovelace Respiratory Research Institute, Albuquerque, NM

MEMBERS

Dr. Ellis Cowling, University Distinguished Professor-at-Large, North Carolina State University, Colleges of Natural Resources and Agriculture and Life Sciences, North Carolina State University, Raleigh, NC

Dr. James D. Crapo, Professor, Department of Medicine, National Jewish Medical and Research Center, Denver, CO

Dr. Douglas Crawford-Brown, Director, Carolina Environmental Program; Professor, Environmental Sciences and Engineering; and Professor, Public Policy, Department of Environmental Sciences and Engineering, University of North Carolina at Chapel Hill, Chapel Hill, NC

Mr. Richard L. Poirot, Environmental Analyst, Air Pollution Control Division, Department of Environmental Conservation, Vermont Agency of Natural Resources, Waterbury, VT

Dr. Armistead (Ted) Russell, Georgia Power Distinguished Professor of Environmental Engineering, Environmental Engineering Group, School of Civil and Environmental Engineering, Georgia Institute of Technology, Atlanta, GA

Dr. Frank Speizer, Edward Kass Professor of Medicine, Channing Laboratory, Harvard Medical School, Boston, MA

SCIENCE ADVISORY BOARD STAFF

Mr. Fred Butterfield, CASAC Designated Federal Officer, 1200 Pennsylvania Avenue, N.W., Washington, DC, 20460, Phone: 202-343-9994, Fax: 202-233-0643 (butterfield.fred@epa.gov) (Physical/Courier/FedEx Address: Fred A. Butterfield, III, EPA Science Advisory Board Staff Office (Mail Code 1400F), Woodies Building, 1025 F Street, N.W., Room 3604, Washington, DC 20004, Telephone: 202-343-9994)

Appendix B – Roster of the CASAC Lead Review Panel

**U.S. Environmental Protection Agency
Science Advisory Board (SAB) Staff Office
Clean Air Scientific Advisory Committee (CASAC)
CASAC Lead Review Panel**

CHAIR

Dr. Rogene Henderson*, Scientist Emeritus, Lovelace Respiratory Research Institute, Albuquerque, NM

MEMBERS

Dr. Joshua Cohen, Research Associate Professor of Medicine, Tufts University School of Medicine, Institute for Clinical Research and Health Policy Studies, Center for the Evaluation of Value and Risk, Tufts New England Medical Center, Boston, MA

Dr. Deborah Cory-Slechta, Director, University of Medicine and Dentistry of New Jersey and Rutgers State University, Piscataway, NJ

Dr. Ellis Cowling*, University Distinguished Professor-at-Large, North Carolina State University, Colleges of Natural Resources and Agriculture and Life Sciences, North Carolina State University, Raleigh, NC

Dr. James D. Crapo [M.D.]*, Professor, Department of Medicine, National Jewish Medical and Research Center, Denver, CO

Dr. Douglas Crawford-Brown*, Director, Carolina Environmental Program; Professor, Environmental Sciences and Engineering; and Professor, Public Policy, Department of Environmental Sciences and Engineering, University of North Carolina at Chapel Hill, Chapel Hill, NC

Dr. Bruce Fowler, Assistant Director for Science, Division of Toxicology and Environmental Medicine, Office of the Director, Agency for Toxic Substances and Disease Registry, U.S. Centers for Disease Control and Prevention (ATSDR/CDC), Chamblee, GA

Dr. Andrew Friedland, Professor and Chair, Environmental Studies Program, Dartmouth College, Hanover, NH

Dr. Robert Goyer [M.D.], Emeritus Professor of Pathology, Faculty of Medicine, University of Western Ontario (Canada), Chapel Hill, NC

Mr. Sean Hays, President, Summit Toxicology, Allenspark, CO

Dr. Bruce Lanphear [M.D.], Sloan Professor of Children’s Environmental Health, and the Director of the Cincinnati Children’s Environmental Health Center at Cincinnati Children’s Hospital Medical Center and the University of Cincinnati, Cincinnati, OH

Dr. Samuel Luoma, Senior Research Hydrologist, U.S. Geological Survey (USGS), Menlo Park, CA

Dr. Frederick J. Miller, Consultant, Cary, NC

Dr. Paul Mushak, Principal, PB Associates, and Visiting Professor, Albert Einstein College of Medicine (New York, NY), Durham, NC

Dr. Michael Newman, Professor of Marine Science, School of Marine Sciences, Virginia Institute of Marine Science, College of William & Mary, Gloucester Point, VA

Mr. Richard L. Poirot*, Environmental Analyst, Air Pollution Control Division, Department of Environmental Conservation, Vermont Agency of Natural Resources, Waterbury, VT

Dr. Michael Rabinowitz, Geochemist, Marine Biological Laboratory, Woods Hole, MA

Dr. Armistead (Ted) Russell*, Georgia Power Distinguished Professor of Environmental Engineering, Environmental Engineering Group, School of Civil and Environmental Engineering, Georgia Institute of Technology, Atlanta, GA

Dr. Joel Schwartz, Professor, Environmental Health, Harvard University School of Public Health, Boston, MA

Dr. Frank Speizer [M.D.]*, Edward Kass Professor of Medicine, Channing Laboratory, Harvard Medical School, Boston, MA

Dr. Ian von Lindern, Senior Scientist, TerraGraphics Environmental Engineering, Inc., Moscow, ID

Dr. Barbara Zielinska, Research Professor, Division of Atmospheric Science, Desert Research Institute, Reno, NV

SCIENCE ADVISORY BOARD STAFF

Mr. Fred Butterfield, CASAC Designated Federal Officer, 1200 Pennsylvania Avenue, N.W., Washington, DC, 20460, Phone: 202-343-9994, Fax: 202-233-0643 (butterfield.fred@epa.gov)

* Members of the statutory Clean Air Scientific Advisory Committee (CASAC) appointed by the EPA Administrator

Appendix C – Agency Charge to the CASAC Lead Review Panel

Charge to the CASAC Pb Panel

Within each of the main sections of the first draft Staff Paper, questions that we ask the Panel to focus on in their review include the following:

Ambient Pb information and analyses (Chapter 2):

1. To what extent are the emissions and air quality characterizations and analyses clearly communicated, appropriately characterized, and relevant to the review of the primary and secondary Pb NAAQS?
2. Does the information in Chapter 2 provide a sufficient ambient Pb-related basis for the exposure, human health and environmental effects, health risk assessment, and environmental assessment presented in later chapters?

Pb-related health effects (Chapter 3):

1. To what extent is the presentation of evidence from the health studies assessed in the Pb AQCD and the integration of information from across the various health-related research areas drawn from the Pb AQCD technically sound, appropriately balanced, and clearly communicated?
2. What are the views of the Panel on the appropriateness of staff's discussion and conclusions in Chapter 3 on key issues related to quantitative interpretation of epidemiologic study results, including, particularly, the form of a blood Pb-response function for neurocognitive effects, and the form of the associated blood Pb metric?
3. What are the Panel's views on the adequacy and clarity of the discussion of potential thresholds in concentration-response relationships presented in Chapter 3?

Human Exposure and Health Risk Analysis, Pilot-Phase (Chapter 4):

1. To what extent are the assessment, interpretation, and presentation of the results of the pilot exposure analysis, including characterization of Pb concentrations in media, the modeling of multi-pathway Pb exposure and application of biokinetic blood Pb models, as presented in Chapter 4 technically sound, appropriately balanced, and clearly communicated?
2. Are the methods used to conduct the pilot exposure analysis, including the modeling of population-level distributions of total blood Pb levels and the pathway-apportionment of those blood Pb levels (*e.g.*, air-inhalation, versus soil-ingestion versus dust-ingestion, versus

background) technically sound? Does the Panel have any suggestions for improvements in the methods used?

3. What are the Panel's views on the staff interpretation of the performance evaluation completed for the pilot analysis (and described in Chapter 4) with regard to the representativeness of individual modeling steps completed for the analysis (*e.g.*, characterization of ambient air and outdoor soil Pb levels and the estimation of blood Pb levels for specific case studies)?
4. In general, are the concentration-response functions and blood Pb metrics (*i.e.*, lifetime average, concurrent blood lead) used in the pilot analysis appropriate for this review?
5. Are the methods used to conduct the pilot health risk assessment, including the application of the cutpoints in relation to the concentration-response functions employed, technically sound? Does the Panel have any suggestions for improvements in the methods used?
6. To what extent does the sensitivity analysis completed for the pilot analysis (and described in Chapter 4) identify key sources of uncertainty and provide an assessment of their impact on risk results?
7. As part of the NAAQS review, there is interest in attempting to differentiate Pb exposure and health risk impacts for modeled populations between: (a) historically-deposited Pb (*e.g.*, near-roadway dust/soil lead from leaded gasoline); and (b) newly-emitted Pb. Does the Panel have specific recommendations regarding approaches that might be employed in the full-scale assessment for this purpose?
8. What are the Panel's views on the most important issues to be addressed in the subsequent full-scale human exposure and health assessment that will be presented in the revised documents?

The Primary Pb NAAQS (Chapter 5)

1. What are the Panel's views on the adequacy and clarity of the presentation of the basis for the existing standard and conclusions reached in the last review?
2. Based on the information contained in the first draft Staff Paper, as well as the AQCD, does the Panel have recommendations with regard to specific aspects of the standard to be considered in developing policy alternatives? For example, considering the prominence of the soil and dust pathways for ambient Pb exposures, and the evidence regarding environmental response times, is there reason to give more emphasis to consideration of an alternative (shorter or longer) averaging time; and, how might this be considered in the full-scale risk assessment given current capabilities?

Pb-related welfare effects and screening level ecological risk assessment (Chapter 6):

1. To what extent is the presentation of evidence from the ecological studies assessed in the Pb AQCD and the integration of information from across the various ecologically-related research areas drawn from the Pb AQCD technically sound, appropriately balanced, and clearly communicated?
2. Given the lack of quantitative information on Pb-related ecosystem effects, what are the Panel's views on the presentation of this topic in chapter 6?
3. What are the Panel's views of the data sources and models used to estimate current levels of Pb in soil, freshwater, and sediment for the case study locations?
4. To what extent are the methods used to conduct the exposure assessment and the interpretation and presentation of the results technically sound, appropriately balanced, and clearly communicated?
5. What are the Panel's views of the approach for addressing uncertainty in apportionment of Pb contributions in the national-scale screen by factoring out those locations with known non-air sources (*e.g.*, mining, point discharges)?
6. To what extent are the assessment, interpretation, and presentation of the results of the screening-level risk analysis, including characterization of lead concentrations in media and the comparisons to ecological screening values, as presented in Chapter 6 and the risk assessment report technically sound, appropriately balanced, and clearly communicated?
7. Does the Panel feel that adequate screening criteria (ecotoxicity screening values) were selected for each of the media?
8. What are the Panel's views on the derivation of the soil screening values for birds and mammals (*i.e.*, using the Eco-SSL methodology)? Do the resultant values adequately reflect current information on exposure characteristics of these organisms?
9. To what extent are the uncertainties associated with the exposure analysis clearly and appropriately characterized in Chapter 6 and the risk assessment report?

Appendix D –Tables on Analyses for the Primary Lead NAAQS

TABLE 1. Relationship of Blood Lead (PbB) to Air Lead (Pb-Air) by Differing Slope Factors

Pb-Air ($\mu\text{g}/\text{m}^3$)	PbB ($\mu\text{g}/\text{dl}$)		
	S.F.* = 5	S.F.* = 10	S.F.* = 20
0.010	0.05	0.10	0.20
0.025	0.13	0.25	0.50
0.050	0.25	0.50	1.00
0.100	0.50	1.00	2.00
0.200	1.00	2.00	4.00

*S.F. = slope factor (m^3/dl) = PbB/Pb-Air; S.F. value varies with increasing impact of indirect Pb-Air pathway (Dust Pb + Soil Pb)

TABLE 2. Relationship of IQ Point Losses to Increases in Pb-Air and Pb-Air-Based Blood Lead (PbB) Values Above Zero ^{a,b,c}

Pb-Air ($\mu\text{g}/\text{m}^3$)	S.F. = 5		S.F. = 10		S.F. = 20	
	PbB ^d	IQ Loss ^{e,f}	PbB	IQ Loss	PbB	IQ Loss
0	0	0	0	0	0	0
0.010	0.05	< 1	0.10	< 1	0.20	< 1
0.025	0.13	< 1	0.25	< 1	0.50	1.5
0.050	0.25	< 1	0.50	1.5	1.00	3.0
0.100	0.50	1.5	1.00	3.0	2.00	6.0
0.200	1.00	3.0	2.00	6.0	4.00	12.0

- a Pb-Air-related increases affecting IQ point loss through calculated PbB values using 3 slope factors per Table 1
- b IQ vs. PbB dose-response relationships based on Lanphear *et al.*, 2005: sub-7.5 $\mu\text{g}/\text{dl}$ linear segment, slope = 3, combining slopes of 2.9 and 3.1 for concurrent and lifetime average dose metrics, respectively
- c Slope factors as defined in Table 1 and text
- d PbB as derived in Table 1
- e Rounding for values < 1 IQ point
- f Population, not individual, IQ loss/gain projections; U.S. CDC 2007 estimates 23,380,860 U.S. children 0-71 months of age. Source: U.S. Centers for Disease Control, 2007. CDC Surveillance Data. (Last updated 2/16/2007). URL: <http://www.cdc.gov/nceh/surv/stats/htm> [accessed 3/8/2007]

**Appendix C – Review Comments from
Individual CASAC Lead Review Panel Members**

This appendix contains the preliminary and/or final written review comments of the individual members of the Clean Air Scientific Advisory Committee (CASAC) Lead Review Panel who submitted such comments electronically. The comments are included here to provide both a full perspective and a range of individual views expressed by Panel members during the review process. These comments do not represent the views of the CASAC Lead Review Panel, the CASAC, the EPA Science Advisory Board, or the EPA itself. The views of the CASAC Lead Review Panel and the CASAC as a whole are contained in the text of the report to which this appendix is attached. Panelists providing review comments are listed on the next page, and their individual comments follow.

Working Draft V1-1 Dated 03/07/2007 – Do Not Cite or Quote

<u>Panelist</u>	<u>Page #</u>
Dr. Joshua Cohen.....	D-3
Dr. Deborah Cory-Slechta.....	D-x
Dr. Ellis Cowling.....	D-x
Dr. James Crapo.....	D-x
Dr. Douglas Crawford-Brown.....	D-x
Dr. Bruce Fowler.....	D-x
Dr. Andrew Friedland	D-x
Dr. Robert Goyer	D-x
Mr. Sean Hays	D-x
Dr. Bruce Lanphear.....	D-x
Dr. Samuel Luoma	D-x
Dr. Frederick J. Miller	D-x
Dr. Paul Mushak	D-x
Dr. Michael Newman	D-x
Mr. Rich Poirot	D-x
Dr. Michael Rabinowitz	D-x
Dr. Armistead (Ted) Russell.....	D-x
Dr. Joel Schwartz	D-x
Dr. Frank Speizer	D-x
Dr. Ian von Lindern	D-x
Dr. Barbara Zielinska	D-x

NOTICE

This report has been written as part of the activities of the U.S. Environmental Protection Agency's (EPA) Clean Air Scientific Advisory Committee (CASAC), a Federal advisory committee administratively located under the EPA Science Advisory Board (SAB) Staff Office that is chartered to provide extramural scientific information and advice to the Administrator and other officials of the EPA. The CASAC is structured to provide balanced, expert assessment of scientific matters related to issue and problems facing the Agency. This report has not been reviewed for approval by the Agency and, hence, the contents of this report do not necessarily represent the views and policies of the EPA, nor of other agencies in the Executive Branch of the Federal government, nor does mention of trade names or commercial products constitute a recommendation for use. CASAC reports are posted on the SAB Web site at: <http://www.epa.gov/sab>.