Alliance of Automobile Manufacturers
Comments on EPA's Draft Policy Assessment for Particulate Matter

Prepared for the Alliance of Automobile Manufacturers by

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Summary

As part of its current review of the adequacy of the current National Air Quality Standards (NAAQS) for particulate matter (PM), the U.S. EPA released a first draft of the Policy Assessment (PA)[1] in September 2019. Also contained within the PA document is the EPA's health risk assessment, which in the previous PM review was a separate document.

As is customary, the PA contains EPA Staff's preliminary recommendations for the indicator, level and form of the PM NAAQS. They considered the following existing PM standards: a primary (health based) annual standard for fine ($PM_{2.5}$) PM, a primary 24-hour standard for $PM_{2.5}$, a primary 24-hour standard for PM_{10}, and a secondary (welfare based) annual standard for $PM_{2.5}$.

For the 24-hour PM_{10} NAAQS and the secondary NAAQS, EPA Staff concluded that the available evidence does not call into question the adequacy of the existing the 24-hour PM_{10}[2] or annual secondary $PM_{2.5}$ standards,[3] so no changes are recommended for these. For the primary $PM_{2.5}$ NAAQS however, they state: "we reach the preliminary conclusion that the available scientific evidence, air quality analyses, and the risk assessment, as summarized above, can reasonably be viewed as calling into question the adequacy of the public health protection afforded by the combination of the current annual and 24-hour primary PM2.5 standards."[4]

Concerning the PM_{10} and the secondary $PM_{2.5}$ NAAQS, AIR, Inc. agrees the evidence does not support making these more stringent. However, AIR does not agree with EPA's assessment that the existing primary $PM_{2.5}$ are inadequate to protect public. For EPA to reach that conclusion,

[1] U.S. EPA (2019). Policy Assessment for the Review of the National Ambient Air Quality Standards for Particulate Matter, External Review Draft. Research Triangle Park, NC. Office of Air Quality Planning and Standards. U.S. EPA. EPA-452/P-19-001 September 2019. Available at: https://www.epa.gov/sites/production/files/2019-09/documents/draft_policy_assessment_for_pm_naaqs_09-05-2019.pdf.
[2] Ibid, p. 4-15.
[3] Ibid, p. 5-39.
[4] Ibid, p. 3-98.

EPA had to ignore the comments CASAC[5] and the Alliance[6,7] submitted on the draft Integrated Science Assessment (ISA)[8] and the Integrated Review Plan.[9]

Details

To support their conclusion, EPA cites old and new evidence of statistical relationships between $PM_{2.5}$ and health effects:

> As an initial matter, we note the longstanding body of health evidence supporting relationships between PM2.5 exposures (short- and long-term) and mortality or serious morbidity effects. The evidence available in this review (i.e., assessed in U.S. EPA, 2018 and summarized above in section 3.2.1) reaffirms, and in some cases strengthens, the conclusions from the 2009 ISA regarding the health effects of PM2.5 exposures (U.S. EPA, 2009). Much of this evidence comes from epidemiologic studies conducted in North America, Europe, or Asia that demonstrate generally positive, and often statistically significant, PM2.5 health effect associations. Such studies report associations between estimated PM2.5 exposures and nonaccidental, cardiovascular, or respiratory mortality; cardiovascular or respiratory hospitalizations or emergency room visits; and other mortality/morbidity outcomes (e.g., lung cancer mortality or incidence, asthma development).[10]

They further state that the epidemiology results are supported by clinical and laboratory studies:

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[5] Cox, LA. (2019). Letter from Louis Anthony Cox, Jr., Chair, Clean Air Scientific Advisory Committee, to Administrator Andrew R. Wheeler. Re: CASAC Review of the EPA's *Integrated Science Assessment for Particulate Matter (External Review Draft – October* 7 *2018)*. April 11, 2019. EPA-CASAC-19-002. U.S. EPA HQ, Washington DC. Office of the Administrator, Science Advisory Board. Available at: https://yosemite.epa.gov/sab/sabproduct.nsf/LookupWebReportsLastMonthCASAC/6CBCBBC3025E13B4852583D90047B352/$File/EPA-CASAC-19-002+.pdf.

[6] Wolff, GT and Heuss, JM (2018). Review and Critique of the U. S. Environmental Protection Agency's First External Review Draft of the "Integrated Science Assessment for Particulate Matter." December 7, 2018. Available at: https://yosemite.epa.gov/sab/sabproduct.nsf/EF1B960097E3EA7E8525835C00751A5C/$File/AIR+PM+ISA+Comments+12-18.pdf.

[7] Heuss, JM and Wolff, GT (2016). Comments on EPA's Draft Integrated Review Plan for Particulate Matter, prepared for the Alliance of Automobile Manufacturers, August 2, 2016. Available at: https://yosemite.epa.gov/sab/sabproduct.nsf/1E6D2E2D91F5C78485258005004EF660/$File/Heuss-Wolff+PM+Alliance+comments+8-2-16.pdf

[8] U.S. EPA. (2018). Integrated Science Assessment (ISA) for Particulate Matter (External Review Draft). Washington, DC. U.S. Environmental Protection Agency, Office of Research and Development, National Center for Environmental Assessment. U.S. EPA. EPA/600/R-18/179. October 2018. Available at: https://cfpub.epa.gov/ncea/isa/recordisplay.cfm?deid=341593.

[9] U.S. EPA. (2016). Integrated review plan for the national ambient air quality standards for particulate matter. Research Triangle Park, NC. Office of Air Quality Planning and Standards. U.S. EPA. EPA-452/R-16-005. December 2016. Available at: https://www3.epa.gov/ttn/naaqs/standards/pm/data/201612-final-integrated-review30plan.pdf.

[10] U.S. EPA, supra note 1, at 3-95.

> Recent experimental evidence strengthens support for potential biological pathways through which $PM_{2.5}$ exposures could lead to the effects reported in epidemiologic studies. This includes evidence from controlled human exposure and animal toxicological studies reporting cardiovascular effects and animal studies reporting respiratory, nervous system, and lung cancer-related effects.[11]

However, elsewhere in the document, they admit the exposure levels used in the clinical and laboratory studies that showed effects were well above ambient levels:

> While controlled human exposure studies support the plausibility of the serious cardiovascular effects that have been linked with ambient PM2.5 exposures (U.S. EPA, 2018, Chapter 6), the PM2.5 exposure concentrations evaluated in most of these studies are well above the ambient concentrations typically measured in locations meeting the current primary standards.[12]

In addition, EPA cannot identify a threshold concentration for effects:

> In addition to broadening our understanding of the health effects that can result from exposures to $PM_{2.5}$ and strengthening support for some key effects (e.g., nervous system effects, cancer), recent epidemiologic studies strengthen support for health effect associations at relatively low ambient $PM_{2.5}$ concentrations. Studies that examine the shapes of concentration response functions over the full distribution of ambient $PM_{2.5}$ concentrations have not identified a threshold concentration, below which associations no longer exist (U.S. EPA, 2018, section 2 1.5.3).[13]

Further, EPA states: "key studies indicate such associations consistently for distributions with long-term mean $PM_{2.5}$ concentrations at or above 8.1 $\mu g/m^3$."[14]

The logic EPA uses to conclude a lower standard is necessary to protect public health ignores the evidence that questions the ability of existing air pollution epidemiology studies to identify causal relationships between PM and a variety of health effects. EPA does recognize some causality determinations maybe faulty in a footnote in the PA:

> Based on its review, the CASAC questioned several of the causality determinations in the draft ISA. Specifically, the CASAC found that "the Draft ISA does not present adequate evidence to conclude that there is likely to be a causal relationship between long-term PM2.5 exposure and nervous system effects; between long-term ultrafine particulate (UFP) exposure and nervous system effects; or between long-term PM2.5 exposure and cancer" (Cox,

[11] Ibid, p. 3-95.
[12] Ibid, p. 3-106.
[13] Ibid, p. 3-95 to 3-96.
[14] Ibid, p. 3-96.

> 2019). Thus, while the causality determinations for these health outcome categories are listed as "likely to be causal" in Table 3-1, we recognize that the final ISA will reflect the EPA's consideration of CASAC advice and that, based on CASAC advice, some or all of these causality determinations could differ in the final ISA. The final PA will reflect these updates.[15]

This footnote, however, does not explain enough. Indeed CASAC (and other commenters) do question these "likely to be causal" determinations, CASAC (and other commenters) also question the legitimacy of EPA's "causal" determinations between $PM_{2.5}$ and mortality and other health effects. The remainder of these comments will focus on causality and other issues ignored by EPA.

In their verbal comments presented at the December 12-13, 2018 and March 28, 2019 meetings and in their April 11, 2019 report to the Administrator,[16] CASAC expressed numerous concerns with the conclusions EPA expressed in their October 2018 PM ISA and the approach that EPA used to evaluate the scientific literature. These are discussed below.

Most importantly "CASAC did not reach consensus on the causality determinations of mortality from $PM_{2.5}$ exposures."[17]

> The CASAC members had varying opinions on whether there is robust and convincing evidence to support the EPA's conclusion that there is a causal relationship between PM2.5 exposure and mortality.[18]

CASAC further went on to say:

> The EPA's mortality causality determination appears to be based almost exclusively on epidemiology studies, which cannot be used in isolation to determine causation. Further integration amongst epidemiology studies showing logical patterns in magnitude and types of health effects, as well as demonstrations of substantial health effects in animals exposed to high concentrations could provide some of the necessary justification for this causality conclusion.[19]

As AIR has pointed out in previous comments, we do not think EPA will be able to show such "logical patterns" because there is a disconnect between the epidemiology and laboratory studies.[20] The mechanisms of how $PM_{2.5}$ at ambient concentrations observed in the U.S. are causing premature mortality have not been demonstrated. As a result, CASAC states:

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[15] Ibid, p. 3-18 footnote.
[16] Cox, supra note 5.
[17] Ibid, p. 1.
[18] Ibid, cover letter p. 3.
[19] Ibid, p. 2.
[20] Wolff and Heuss, supra note 6.

> How do low concentrations of PM2.5 cause mortality? The EPA should discuss not just general, possible mechanisms, but specifically how ambient concentrations of PM2.5 can move into and through the biological systems in the body to activate a cascade of effects that ultimately lead to a person's death.[21]

CASAC also asks if there is any support from the animal studies:

> If the EPA has identified any short-term or long-term exposure studies in animals where PM exposure increased mortality, that would be a useful addition to the discussions in Chapter 11. If none has been identified, that would also be useful information, if put into the appropriate context of aging and differential susceptibility of rodents.[22]

The issue of geographical heterogeneity which the Alliance brought up in earlier comments,[23] was also a focus of CASAC:

> Heterogeneity. The EPA should also address the substantial unexplained geographic heterogeneity in effect estimates between PM2.5 exposure and mortality (e.g. Eftim et al., 2008, Baxter et al., 2017, and many others). In the previous PM NAAQS review, the EPA noted that uncertainty remained in the form of unexplained within- and between-city heterogeneity in responses to PM. The EPA also asked several policy-relevant questions related to geographical heterogeneity in the Integrated Review Plan for this current PM NAAQS review. Given the emphasis that the EPA has placed on this topic, they should include more discussion of geographic and other types of heterogeneity in this ISA. The implications of unexplained heterogeneity need to be discussed for those endpoints where many potential explanations have been tested, but none has been able to explain the observed heterogeneity (e.g. short-term PM2.5 exposure and total mortality). At what point does heterogeneity move from being an uncertainty, to impacting the causality conclusion or other policy-relevant issues such as the use of a single effect estimate for the whole nation?[24]

CASAC also questions EPA's conclusion of a linear dose-response function with no threshold:

> Some members of CASAC think that the EPA should do further work on C-R functions. In the Draft ISA, the EPA concludes that the evidence from epidemiology studies largely supports a linear, no-threshold association between PM2.5 and various health effects. However, a number of statistical studies have shown that the error (e.g., measurement error) in these types of epidemiology studies lead study authors to the erroneous conclusion that C-R functions are linear with no threshold when that is not, in fact, the case (Rhomberg et al., 2011; Brauer et al., 2002; Cox, 2018; Lipfert and Wyzga, 1996; Watt et al., 1995;

[21] Cox, supra note 5, at 1.
[22] Ibid, p. 3.
[23] Heuss and Wolff, supra note 7, at 6.
[24] Cox, supra note 5, at 2.

> Yoshimura, 1990). Therefore, the EPA should not be using these epidemiology studies to draw conclusions about the true shape of the relationship between PM2.5 and health effects, unless it can strongly argue (and provide evidence) that the referenced epidemiology studies can produce an unbiased estimate of the true shape of the C-R function. In addition, this conclusion is not consistent with the evidence of a threshold of effects demonstrated in human controlled exposure and animal toxicology studies.[25]

Another issue AIR has brought up in previous comments which EPA has largely ignored is the lack of consistency[26] between different epidemiology studies that examine both similar and dissimilar health endpoints. CASAC expresses similar thoughts:

> Concentration Concordance. When discussing the continuum of effects from PM2.5 exposure, the EPA should include a discussion of how this continuum is impacted by the concentrations at which different effects have been observed. For example, when the EPA states that mortality evidence provides coherence for a continuum of effects, this should be considered within the context of whether more serious effects occur at higher, lower, or similar concentrations as more mild effects. This comparison of concentrations of effect should be extended to comparisons between epidemiology, animal, and human controlled exposure studies.[27]

AIR believes that such an analysis will show the absence of "coherence for a continuum effects" which will further undermine EPA's conclusion for causal relationships. CASAC continues:

> Comparing results between and within studies. The EPA could improve the integration of evidence in this ISA by hypothesis-testing its conclusions by comparing PM2.5 effect estimates within and between studies. For example, if one expects that some subset of mortality is more affected by PM2.5 (e.g. cardiovascular mortality), then that mortality should have a larger and more significant association with PM2.5 than total mortality. Similarly, if all these effects are occurring at the same concentrations, then one would expect more mild effects (e.g. symptom exacerbation) to be more common and more likely to show an association than the more serious effects (e.g. hospital admission or mortality). One would also expect that long-term effects would occur at lower concentrations and would show stronger effects than short-term, because of cumulative exposure (if PM2.5 has an impact via cumulative exposure); and that health risks associated with PM2.5 would be higher in places with higher PM2.5 concentrations. Investigating these types of patterns could be done with the study information that the EPA has already collected for this ISA and would greatly strengthen the conclusions that are drawn.[28]

[25] Ibid, p.21.
[26] Heuss and Wolff, supra note 7, at 5.
[27] Cox, supra note 5, at 2.
[28] Ibid, p. 2.

Based on our familiarity with the heterogeneity of epidemiological results,[29] AIR does not believe such "patterns" exist. The lack of these patterns further questions the conclusion of causality.

Finally, there is another major shortcoming of EPA's review that CASAC identifies: "the CASAC finds that the Draft ISA does not provide a sufficiently comprehensive, systematic assessment of the available science relevant to understanding the health impacts of exposure to particulate matter (PM)."[30] They further comment:

> *Lack of comprehensive, systematic review* - some of the relevant and important scientific literature is not reviewed and study quality is not systematically considered. The revised ISA should provide a clearer and more complete description of the process and criteria for study quality assessment, including an explanation of how systematic assessments of individual study quality were used in preparing the ISA and the causality determinations.[31]

A framework that we believe deserves consideration for providing "a sufficiently comprehensive, systematic assessment of the available science" was contained in the comments presented at the December 2018 CASAC Meeting by National Council for Air and Stream Improvement.[32]

[29] Heuss and Wolff, supra note 7, at 3.
[30] Cox, supra note 5, at cover letter 1.
[31] Ibid.
[32] NCASI (2018). Comments on the Integrated Science Assessment for Particulate Matter, December 5, 2018. Available at: https://yosemite.epa.gov/sab/sabproduct.nsf/4CC636F22DED7F208525835B0050D685/$File/NCASI+Comments+on+Draft+PM+ISA_web.pdf.