

## **COARSE PARTICULATE MATTER COALITION**

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December 5, 2018

Dr. Louis A. ("Tony") Cox, Jr.  
Chairman  
Clean Air Scientific Advisory Committee  
EPA Science Advisory Board Staff Office(1400R)  
U.S. Environmental Protection  
Agency, 1200 Pennsylvania Avenue  
NW, Washington, DC 20460;

*Submitted electronically through SAB staff*

Re: CASAC Review of Integrated Science Assessment for Particulate  
Matter (First External Review Draft)

Dear Chairman Cox and Committee Members:

The Coarse Particulate Matter Coalition (CPMC), an organization of industries dedicated to scientifically sound regulation of coarse particulate matter (PM10-2.5) in air, offers the following comments in connection with the Clean Air Scientific Advisory Committee (CASAC) review of the First External Review Draft of EPA's Integrated Science Assessment (ISA) for Particulate Matter.<sup>1</sup>

### **The Proposed New Coarse PM Causality Determinations Should Be Revised**

As discussed in the draft ISA, the proposed causality findings must be based on the criteria for such findings presented in EPA's General Preamble for ISAs, which includes aspects to consider in judging causality. These are shown in Table 1 below, reprinted from the General Preamble. They include: consistency, coherence, biological plausibility, exposure-response relationship, strength of the observed association, experimental evidence, temporality, specificity and analogy. As noted in the Preamble, "one cannot simply count the number of studies reporting statistically significant results

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<sup>1</sup> Current members of the Coalition include the Corn Refiners Association, National Cotton Council, National Stone, Sand & Gravel Association, Rio Tinto Kennecott and the National Cattlemen's Beef Association.

or statistically nonsignificant results and reach credible conclusions about the relative weight of evidence and the likelihood of causality. Rather, these aspects provide a framework for systematic appraisal of the body of evidence, informed by peer and public comment and advice, which includes weighing alternative views on controversial issues” (p.19). While not meeting one or more of the principles does not automatically preclude a determination of causality, these aspects provide a framework for assessing the evidence.

Attached for your review are tables we have prepared, based on EPA’s findings in the draft ISA, showing how the causation aspects have been considered with respect to each new causality finding for coarse PM. These tables demonstrate that the draft ISA does not provide comprehensive, consistent and transparent analyses of EPA’s causal criteria. As a result, the “suggestive” causality findings for cancer and for long term cardiovascular, metabolic, nervous system and mortality effects from coarse PM exposure appear to us to be unjustified and should be revised to “inadequate” in the next draft. We will provide a more detailed analysis of these issues in our Comments to EPA on the draft ISA, which we will forward to the Committee when they are filed on December 11.

### **Coarse PM Health Findings Should be Limited to Urban Roadside Dusts**

The draft ISA finds that PM<sub>10-2.5</sub> throughout the US is almost entirely primary in origin, composed largely of crustal material, sea salt, and biological material, and also notes that national average PM<sub>10-2.5</sub> concentrations have changed little over the past decade (p. ES-4). The ISA also finds that concentrations are highest in southwestern U.S. and are observed to be largely dominated by crustal material, but organic material can also represent a substantial contribution to mass, as well as biological material like bacteria, viruses, fungal spores, pollen, and plant debris (p. 1-12).

In contrast, the profile of urban roadside emissions presented in the draft ISA is quite different (pp. 2-70-71). The draft reports higher concentrations of PM components near roads with heavy traffic, including carbonaceous aerosols, PAHs, steranes, chromium, copper, iron and black carbon.

The 2009 Integrated Science Assessment (ISA) for PM noted that in the prior review, "the CASAC PM Panel was also in general agreement 'that coarse particles in urban or industrial areas are likely to be enriched by anthropogenic pollutants that tend to be inherently more toxic than the windblown crustal material which typically dominates coarse particle mass in arid rural areas'" (p. 1-9). The 2009 ISA discussed a number of new studies involving crustal material, but nearly all of them involved road dust, combustion sources or other external sources of potential contamination (see Table 6-17). The only reference to potential harm from exposure to crustal material involved studies of dust storms, with concentrations well above the current standards (p. 6-97). Accordingly, the Policy Assessment (PA) in the last review found that evidence of harm from exposure to crustal material was limited to studies involving high concentrations (p. 3-29).

This also was recognized by CASAC members in the last review. For example, in his individual comments on the second draft PA Dr. Joe Brain stated:

There is also a continuing cry for a more thoughtful assessment of particle composition. There is increasing evidence that the extent of particle toxicity relates to the composition and solubility of the particles. There is also concern about the most appropriate metric. Should standards really be mass-based or should they reflect numbers or surface area of particles? The composition issue is particularly relevant to discussions of coarse particles. How do we make the distinction between those derived from fossil fuel combustion and resuspended crustal dust? There is consensus that resuspended crustal dust is less toxic than combustion products. There are clear regulatory implications as well. It's hard to regulate dust storms, but easier and more appropriate to regulate stationary and mobile sources (emphasis added).

The only evidence for coarse crustal health effects discussed in the current draft ISA concerns dust storm events during which concentrations well above the current standards were linked to increases in cardiovascular ED visits and hospital admissions (p. 6-248). The draft ISA notes that even with respect to these studies, there are concerns with respect to the potential for exposure measurement error and copollutant confounding (*id.*).

As shown in the attached tables we have prepared, the vast majority of the new health studies for coarse PM, even if accepted at face value, are studies of contaminated roadside dusts. They are not studies of coarse crustal material uncontaminated by roadside emissions. The next draft of the ISA should limit the coarse PM findings to the urban roadside dusts that were the subject of the key studies, and should exclude coarse PM dominated by crustal material.

Our Coalition and its members rely on this Committee to ensure that EPA's interpretations of the scientific evidence are reasonably balanced and necessary to protect public health and welfare. We ask you to give close examination to the issues discussed above, which are vital to the future of our industries, and we thank you for the time and effort you have contributed to the CASAC deliberations on these important public health issues.

Respectfully submitted,

Kurt E. Blase

Counsel for the Coarse PM Coalition

**Table I Aspects to aid in judging causality.**

| Aspect   | Description  |
|--|--|
| Consistency  | An inference of causality is strengthened when a pattern of elevated risks is observed across several independent studies. The reproducibility of findings constitutes one of the strongest arguments for causality. Statistical significance is not the sole criterion by which the presence or absence of an effect is determined. If there are discordant results among investigations, possible reasons such as differences in exposure, confounding factors, and the power of the study are considered.   |
| Coherence  | An inference of causality from one line of evidence (e.g., epidemiologic, controlled human exposure, animal, or ecological studies) may be strengthened by other lines of evidence that support a cause-and-effect interpretation of the association. There may be coherence in demonstrating effects from evidence across various fields and/or across multiple study designs or related health endpoints within one scientific line of evidence. For example, evidence on welfare effects may be drawn from a variety of experimental approaches (e.g., greenhouse, laboratory, and field) and subdisciplines of ecology (e.g., community ecology, biogeochemistry, and paleontological/historical reconstructions). |
| Biological plausibility                              | An inference of causality is strengthened by results from experimental studies or other sources demonstrating biologically plausible mechanisms. A proposed mechanism, which is based on experimental evidence and which links exposure to an agent to a given effect, is an important source of support for causality.  |
| Biological gradient (exposure-response relationship) | A well-characterized exposure-response relationship (e.g., increasing effects associated with greater exposure) strongly suggests cause and effect, especially when such relationships are also observed for duration of exposure (e.g., increasing effects observed following longer exposure times).   |
| Strength of the observed association                 | The finding of large, precise risks increases confidence that the association is not likely due to chance, bias, or other factors. However, it is noted that a small magnitude in an effect estimate may or may not represent a substantial effect in a population.  |
| Experimental evidence                                | Strong evidence for causality can be provided through "natural experiments" when a change in exposure is found to result in a change in occurrence or frequency of health or welfare effects.  |
| Temporality of the observed association              | Evidence of a temporal sequence between the introduction of an agent and appearance of the effect constitutes another argument in favor of causality.  |
| Specificity of the observed association              | Evidence linking a specific outcome to an exposure can provide a strong argument for causation. However, it must be recognized that rarely, if ever, does exposure to a pollutant invariably predict the occurrence of an outcome, and that a given outcome may have multiple causes.  |
| Analogy  | Structure activity relationships and information on the agent's structural analogs can provide insight into whether an association is causal. Similarly, information on mode of action for a chemical, as one of many structural analogs, can inform decisions regarding likely causality.   |

**Table 2: Draft ISA Coarse PM Cancer Analysis**

|                                |  |
|--------------------------------|--|
| <i>Consistency</i>             | Two epidemiological studies, imprecise results, significant exposure uncertainty |
| <i>Coherence</i>               | No direct experimental evidence of carcinogenicity*                              |
| <i>Biological plausibility</i> | Few studies, mixed results   |
| <i>Exposure-response</i>       | No relationship discussed, data likely inadequate                                |
| <i>Experimental evidence</i>   | None discussed*  |
| <i>Temporality</i>             | No discussion  |
| <i>Specificity</i>             | Confounding PM2.5 exposures, studies focused on urban road dusts                 |
| <i>Analogy</i>                 | Not discussed  |

\*As discussed in EPA's General Preamble for ISAs, "coherence" refers to animal or human exposure data that replicate or confirm epidemiological results. "Experimental evidence" refers to "natural experiments" where a change in exposure is found to result in a change in health effects.

**Table 3: Draft ISA Coarse PM Long-Term Cardiovascular Analysis**

|                                |   |
|--------------------------------|---|
| <i>Consistency</i>             | No consistent pattern of associations, significant exposure uncertainty |
| <i>Coherence</i>               | No direct experimental evidence of association*                         |
| <i>Biological plausibility</i> | Evidence insufficient to establish                                      |
| <i>Exposure-response</i>       | No relationship discussed, data likely inadequate                       |
| <i>Experimental studies</i>    | None discussed*   |
| <i>Temporality</i>             | No discussion   |
| <i>Specificity</i>             | Confounding PM2.5 exposures, studies focused on urban road dusts        |
| <i>Analogy</i>                 | Not discussed   |

\*As discussed in EPA's General Preamble for ISAs, "coherence" refers to animal or human exposure data that replicate or confirm epidemiological results. "Experimental evidence" refers to "natural experiments" where a change in exposure is found to result in a change in health effects.

**Table 4: Draft ISA Coarse PM Long-Term Metabolic Analysis**

|                                |  |
|--------------------------------|--|
| <i>Consistency</i>             | One study, “results did not provide strong evidence of an association    |
| <i>Coherence</i>               | No direct experimental evidence of association*                          |
| <i>Biological plausibility</i> | Limited evidence   |
| <i>Exposure-response</i>       | No relationship discussed, data likely inadequate                        |
| <i>Experimental studies</i>    | None discussed*  |
| <i>Temporality</i>             | No discussion  |
| <i>Specificity</i>             | Confounding PM2.5 and NO2 exposures, studies focused on urban road dusts |
| <i>Analogy</i>                 | Not discussed  |

\*As discussed in EPA’s General Preamble for ISAs, “coherence” refers to animal or human exposure data that replicate or confirm epidemiological results. “Experimental evidence” refers to “natural experiments” where a change in exposure is found to result in a change in health effects.

**Table 5: Draft ISA Coarse PM Long-Term Nervous System Analysis**

|                                |   |
|--------------------------------|---|
| <i>Consistency</i>             | Few epidemiological studies, inconsistent results, significant exposure uncertainty and spatial variation |
| <i>Coherence</i>               | No direct experimental evidence of association*   |
| <i>Biological plausibility</i> | One study, uncertain results  |
| <i>Exposure-response</i>       | No relationship discussed, data likely inadequate   |
| <i>Experimental studies</i>    | None discussed*   |
| <i>Temporality</i>             | No discussion   |
| <i>Specificity</i>             | No adjustment for copollutants, studies focused on urban road dusts                                       |
| <i>Analogy</i>                 | Not discussed   |

\*As discussed in EPA's General Preamble for ISAs, "coherence" refers to animal or human exposure data that replicate or confirm epidemiological results. "Experimental evidence" refers to "natural experiments" where a change in exposure is found to result in a change in health effects.

**Table 6: Draft ISA Coarse PM Long-Term Mortality Analysis**

|                                |  |
|--------------------------------|--|
| <i>Consistency</i>             | Epidemiological studies provide “no consistent evidence for positive associations,” significant exposure uncertainty |
| <i>Coherence</i>               | No direct experimental evidence of association*  |
| <i>Biological plausibility</i> | “Limited information”  |
| <i>Exposure-response</i>       | No relationship discussed, data likely inadequate  |
| <i>Experimental studies</i>    | None discussed*  |
| <i>Temporality</i>             | Absence of spatial and temporal information reduces confidence in epidemiological results                            |
| <i>Specificity</i>             | Copollutant confounding in epidemiological studies   |
| <i>Analogy</i>                 | Not discussed  |

\*As discussed in EPA’s General Preamble for ISAs, “coherence” refers to animal or human exposure data that replicate or confirm epidemiological results. “Experimental evidence” refers to “natural experiments” where a change in exposure is found to result in a change in health effects.