COMMENTS OF THE
COARSE PARTICULATE MATTER COALITION
ON
DRAFT INTEGRATED SCIENCE ASSESSMENT
FOR
PARTICULATE MATTER
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Introduction and Summary

The Coarse Particulate Matter Coalition is an organization of industry groups dedicated to scientifically sound regulation of coarse particulate matter (PM) in air.[1] We appreciate the opportunity to submit these comments on the first draft of EPA's Integrated Science Assessment (ISA) for particulate matter (PM). Our comments may be summarized as follows:

1. The next draft of the ISA should include clear criteria for selection and evaluation of key studies.

2. The next draft should focus more clearly on the limitations of the health effects evidence for PM10 and PM10-2.5, as in the current CD. It should also clarify that: (a) positive associations with PM10 increasingly are tied to PM2.5 components associated with traffic or other combustion sources; and (b) positive associations have not been clearly demonstrated for PM 10-2.5.

3. The studies indicating absence of harm from exposure to coarse crustal material should be included, and issues with potential contamination of coarse PM by toxins in fine PM should be addressed.

4. The coarse PM monitor bias should be addressed in more detail.

These issues are discussed below in detail.

[1] Current members of the Coalition include the National Cotton Council, National Oilseed Processors Association, National Stone, Sand & Gravel Association and Kennecott Utah Copper Corporation.

Criteria for Selection of Studies

A recurrent theme throughout the recent reviews of the national ambient air quality standards (NAAQS) for PM has been the absence of a clear and comprehensive set of criteria for selection and evaluation of the key studies, and particularly health studies, on which the Criteria Document and now the ISA will focus. This was discussed in a recent Report of EPA's Board of Scientific Counselors (BOSC) on the Agency's Human Health Risk Assessment Program:

> [T]he process of reviewing criteria air pollutants has been substantially changed and the HHRA Program should be particularly vigilant in assuring that the new process provides assessments of comparable quality to those developed under the previous process. Although the Subcommittee believed that the input from CASAC, stakeholders and Congressional staff provided confidence that ISA work products will continue to be of high quality, it also thought there were several important issues that needed attention. These included: (1) the need for specific criteria that clearly articulate a process for inclusion or exclusion of scientific studies; (2) a strategy for identifying gaps in the science and a plan for filling in the gaps; and (3) the potential challenges associated with creating a single document, the ISA, that both summarizes the recent research and prioritizes the "key" studies across the older and more recent research.[2]

Our review of the preliminary draft PM ISA did not reveal significant progress with respect to these issues. In the past, EPA has been criticized by both the public and the Clean Air Scientific Advisory Committee (CASAC) for appearing to "cherrypick" studies that tend to support the staff perspective. A related issue is the extent to which primary reliance should be placed upon foreign studies. As discussed further below, there are several instances in the draft ISA where EPA appears to be placing primary reliance on foreign studies finding effects in the face of domestic studies that did not.

Clear criteria for study selection and evaluation, subject to CASAC and public comment, could do much to eliminate these issues. We urge EPA to provide such criteria for CASAC and public review in the next draft of the ISA.

Limitations of Coarse PM Health Studies

The current PM Criteria Document (CD) places great emphasis on the limitations of the health data for coarse PM. They are described in CD Chapter 8 as follows:

> *Coarse-fraction particles*. A much more limited body of evidence is suggestive of associations between short-term (but not long-term) exposures (inferred from stationary air monitor measures) to ambient coarse-fraction thoracic particles (generally indexed by PM10-2.5) and

[2] BOSC Human Health Risk Assessment Subcommittee, *Review of the Office of Research and Development's Human Health Risk Assessment Program*, p. 6 (April 2008).

various mortality and morbidity effects observed at times in some locations. This suggests that PM10-2.5, or some constituent component(s) of PM10-2.5, may contribute under some circumstances to increased human health risks.

The strength of the evidence varies across endpoints, with somewhat stronger evidence for coarse-fraction particle associations with morbidity (especially respiratory) endpoints than for mortality. Reasons for differences among findings on coarse-particle health effects reported for different cities are still poorly understood, but several of the locations where significant PM10-2.5 effects have been observed (e.g., Phoenix, Mexico City, Santiago) tend to be in drier climates and may have contributions to observed effects due to higher levels of organic particles from biogenic processes (e.g., endotoxins, fungi, etc.) during warm months. Other studies suggest that particles of crustal origin are generally unlikely to exert notable health effects under most ambient exposure conditions. Some exceptions may include situations where crustal particles have come to be heavily contaminated by metals originally emitted as fine particles from smelting operations but deposited over many years on soils around smelters, steel mills, etc. (see Item 10, below). Also, in some U.S. cities (especially in the NW and the SW) where PM10-2.5 tends to be a large fraction of PM10, measurements, coarse thoracic particles from woodburning are often an important source during at least some seasons. In such situations, the relationship between hospital admissions and PM10 may be an indicator of response to coarse thoracic particles from wood burning.

These findings are then analyzed in Chapter 9:

Still fewer studies have used PM10-2.5 measurements. The effect estimates are almost all positive and similar in magnitude to those reported for PM2.5 and PM10, but few reach statistical significance. Measurement error likely contributes to greater uncertainty, reflected by wider confidence intervals, in effect estimates for PM10-2.5 than for PM2.5 and PM10 . . . For PM10-2.5, the evidence for associations with mortality is more limited and clearly not as strong, although it is important in interpreting these results to consider issues such as exposure error (which could cause the calculated effects to be lower and less significant than the true values) (p. 9-28).

The few available PM10-2.5 studies also provide some evidence for associations between hospitalization for cardiovascular and respiratory diseases with PM10-2.5. PM10-2.5-hospitalization effect estimates were similar in magnitude to those for PM10 and PM2.5, but with less precision. For PM10-2.5, the evidence for associations with mortality is more limited; the magnitude of the effect estimates is very similar to those for PM2.5and PM10, but in terms of precision, the evidence is not as strong (p. 9-32).

> Exposure relationships for PM10-2.5 have been less well studied, but exposure error and measurement error would be expected to have greater influence for associations with PM10-2.5 than for PM2.5; this likely contributes to larger confidence intervals around PM10-2.5 effect estimates (p. 9-38).
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> There are fewer studies available for PM10-2.5 and the magnitude of the effect estimates for associations with mortality and morbidity effects (especially respiratory morbidity) is similar to that for PM2.5, but the lesser precision reduces the strength of the evidence for PM10-2.5 effects. Little evidence is available to allow conclusions to be drawn about long-term PM10-2.5 exposures and morbidity (p. 9-46).

In commenting on the penultimate draft of the CD, which included the findings quoted above, EPA's Clean Air Scientific Advisory Committee (CASAC) likewise focused on the limitations of the coarse PM studies:

> [T]he information regarding $PM_{10\text{-}2.5}$ is not yet as clearly articulated . . . There is relatively limited information on $PM_{10\text{-}2.5}$, and those limits need to be made clear. <u>PM_{10} is not an appropriate substitute for $PM_{10\text{-}2.5}$ since it is possible that some of the adverse health effects of PM_{10}, most notably annual mortality, reflect only the $PM_{2.5}$ effects and that the coarse mode particles add little to health risk.</u> This issue and the data addressing it need to more clearly be brought out in the document (p. 3, emphasis added).[3]

The draft ISA takes a step in the opposite direction. The discussions above from the current CD have been omitted, though we are not aware of any significant improvement in the evidence and the reasons for this omission are not discussed. Although the draft ISA employs new criteria for causal determination, the summaries of causal determination often do not summarize the limitations of the key studies, particularly in cases where a causal relationship is found to be likely. This is true for both PM10 and PM10-2.5.

<u>Short Term Exposure</u>

<u>Cardiovascular morbidity.</u> The Summary of Causal Determination for PM10 finds that a causal relationship is likely to exist between short-term PM10 exposures under the current standard and cardiovascular morbidity. "The bulk of recent evidence" for this finding "is derived from epidemiologic studies of hospital admissions and ED visits" (p. 6-94). Little analysis is provided as to the relative strengths and weaknesses of these studies. The more detailed discussions of these studies indicate the following limitations and caveats:

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[3] EPA Science Advisory Board, *EPA's Fourth External Review Draft of Air Quality Criteria for Particulate Matter: A Peer Review by the Clean Air Scientific Advisory Committee Particulate Matter Review Panel* (October 2004).

- The U.S. MCAPS study is reported as showing increased risk from exposure to PM 10-2.5, but it is primarily a PM2.5 study that was subsequently extended by including counties with co-located PM10 and PM10-2.5 monitors. The discussions of this study report no results for PM10. Further, "in a 2-pollutant model adjusted for PM2.5, the association between PM10-2.5 and CVD hospitalization lost precision" (p. 6-76).

- Several European studies are reported as showing increased risk from PM10 exposure (p. 6-75). However, "non-significant increases in association with PM10-2.5 were reported" (*id*). Further, very little detail is provided with respect to these studies and the PM10 measurement methods they used. The draft ISA notes that in Paris, one of the cities studied, PM13 was used. Only TSP data were used in Milan and Rome. BS data apparently also were used (p. 6-73).

- The Atlanta-based SOPHIA study is reported as showing increased risk for PM10, but "in a more recent analysis from this study with an additional 4 years of data, ED visits for CVD were not significantly associated with PM10 or PM2.5, but were significantly associated with total carbon . . . EC . . . and organic carbon . . . components of PM2.5. More recently, Sarnat et al. (2008) used multiple source-apportionment methods to evaluate the association between all CVD ED visits and specific PM2.5 sources and found consistent positive associations with sources related to motor vehicles and biomass combustion" (p. 6-76).

- The study by Janssen et al. (2002) "found that in multivariate analyses PM10 coefficients increased significantly with increasing percentage of PM10 emissions from highway vehicles/diesels and oil combustion" (p. 6-76).

- Positive PM10 results from single-city studies were not statistically significant. The study in Darwin, Australia "found little or no evidence of an association between PM10 and cardiovascular disease hospital admissions in the general population" (p. 6-77).

The subsequent discussions of specific cardiovascular diseases rely on many of these same studies. Most of the reported results are for PM2.5. The PM10 results are mixed and virtually no results are reported for PM10-2.5. In a separate analysis, the draft concludes that "evidence is inadequate to determine if a causal relationship exists between PM10-2.5 exposures and cardiovascular morbidity" (p. 6-97), yet this is not discussed on evaluation of the PM10 studies.

<u>Respiratory Morbidity</u>. The draft ISA concludes that "consistent evidence from epidemiological studies alone is sufficient to conclude that a causal relationship is likely

to exist between relevant PM10 exposures and short-term respiratory morbidity" (p. 6-183). This conclusion is based primarily on studies of respiratory symptoms and medication use in asthmatic children, and on evidence of ED visits and hospital admissions for older adults.

Again, the evidence has significant limitations that should be reflected in this conclusion. With respect to the studies of symptoms and medication use in asthmatic children:

- In the CAMP study, one of two large new longitudinal urban U.S. studies, "in contrast to several past studies (Delfino et al., 1996; 1998), no associations were observed between PM10 and asthma exacerbations or medication use" (p. 6-112).

- The other large new study, NCICAS, reported an effect associated with PM10 exposure, but the effect was "attenuated in models including O3, SO2, and NO2" (p. 6-112).

- The Slaughter study, which involved a subset of the population in the CAMP study, apparently did not examine the co-pollutants that attenuated the results in the NCICAS study and found no effect on inhaler use in co-pollutant models (p. 6-113).

- The Marr study reported an effect for PM10 but not for PM10-2.5 (p. 6-113).

- In the first Delfino study, effects were found only in children not taking medication, and no analysis was performed for co-pollutants. In the second study, "associations between EC or OC and asthma symptoms were very similar to those for PM10" (p. 6-115).

The evidence with respect to ED and hospital admissions for older adults reflects similar limitations:

- The draft notes that "effect estimates are clearly heterogeneous, with evidence of both regional and seasonal differences at play" (p. 6-162).

- The studies of all respiratory disease are described only as "somewhat consistent," with several of them failing to report significant effects (p. 6-166).

- The MCAPS study "observed small imprecise increases in respiratory admissions with 24-h PM10-2.5 concentration . . . which decreased after adjustment for PM2.5" (p. 6-169).

- The results of other studies of PM10-2.5 were mixed and the effect estimates lost precision when adjusted for co-pollutants (p. 6-171)

- The results of the asthma studies are not consistent and are generally larger than those for all respiratory disease combined (p. 6-175).

- The results of the COPD data are inconsistent, found no significant effects for PM10-2.5 and are attributed in part to traffic sources and co-pollutants (pp. 6-177-179).

- The pneumonia studies primarily address PM2.5; the one study that reported effects from PM10 indicates that they were seasonal. Most of these studies reported no effects for PM10-2.5 (p. 6-180).

Mortality

As discussed above, the current CD finds that the mortality data for PM10-2.5 are "more limited and clearly not as strong." In addition, CASAC advised that "PM_{10} is not an appropriate substitute for $PM_{10-2.5}$ since it is possible that some of the adverse health effects of PM_{10}, most notably annual mortality, reflect only the $PM_{2.5}$ effects and that the coarse mode particles add little to health risk." While the draft ISA omits these conclusions, the evidence presented continues to support them.

The mortality causal determination for PM10 discusses a number of positive studies, but makes no mention of the possibility that the effects could be attributable to PM2.5 (p. 6-240). The evidence with respect to PM10-2.5 continues to be very limited, as the draft ISA notes:

> The results from newly available studies that examined the association between short-term exposure to PM10-2.5 and mortality are mixed, as was the case in the 2004 PM AQCD. Due to the relatively wide confidence bands for the mortality risk estimates from the single-city studies evaluated along with the city-to-city variation in the chemical components of PM10-2.5, a quantitative summary of PM10-2.5 effects may not be informative at this point. In addition, the mortality risk estimates associated with PM10-2.5 may also be influenced by effect modifying conditions (e.g., season, relative exposure error, and dust storms), and to date have not been extensively examined. Clearly, more data are needed to characterize the chemical and biological components that may modify the potential toxicity of coarse particles (p 6-227).

Despite these conclusions, the draft finds the evidence "suggestive" of a causal relationship between mortality and ambient PM10-2.5 concentrations (p. 6-242). Heavy reliance is placed on one Canadian study, in the face of mixed results in other studies, but it is noted that the effects in all of the studies "may be confounded by gaseous pollutants or influenced by effect modifying conditions

(e.g. season, relative exposure error, dust storms)" (p. 6-242). It is then suggested that "the consistent association between short-term exposure to PM10 (which includes PM10-2.5) and mortality provides some evidence for the presence of an association between PM10-2.5 and mortality" (*id*.). This flies in the face of the prior finding that the PM10 association is likely due to the presence of PM 2.5.

Long Term Exposure

As discussed above, the current CD concludes that the evidence is insufficient to support conclusions with respect to potential adverse effects from long term exposure to PM10 or PM10-2.5. The draft ISA would reverse this conclusion with respect to PM10. This is based primarily on recent studies of respiratory morbidity. The draft finds that "a major challenge to interpreting the results of these studies is that the PM measures and concentrations of other air pollutants are often correlated; however, the consistency of findings across different locations supports an independent effect of PM10" (p. 7-48). A similar conclusion is drawn with respect to studies of pulmonary function (p. 7-49). Again, there is no discussion of whether the reported effects may be attributable to the PM2.5 component, in addition to the other co-pollutants. There is also no discussion of the data for PM10-2.5, which continue to be deemed inadequate to support any conclusions with respect to long term effects.

Health Effects Conclusions

The next draft ISA should:

1. focus more clearly on the limitations of the evidence for PM10 and PM10-2.5, as in the current CD;

2. clarify that positive associations with PM10 increasingly are tied to PM2.5 components associated with traffic or other combustion sources; and

3. clarify that positive associations have not been clearly demonstrated for PM 10-2.5.

Coarse Crustal Material

Health Data

The current PM CD Staff Paper (SP) and related CASAC reports emphasize the absence of evidence that exposure to coarse crustal material is harmful at ambient concentrations. With respect to crustal material, the CD concludes:

> The above results, overall, mostly suggest that crustal particles (coarse or fine) per se are not likely associated with daily mortality. However, as noted in the previous section, three analyses of Phoenix, AZ data do suggest that PM10-2.5 was associated with mortality. The results from one of the three studies (Smith et al., 2000) indicate that coarse particle-mortality associations are stronger in spring and summer, when the anthropogenic metal (Fe, Cu, Zn, and Pb) contribution to PM10-2.5 is lowest as determined by factor analysis. However, during spring and summer, biogenic processes (e.g., wind-blown pollen fragments, fungal materials, endotoxins, and glucans) may contribute more to the PM10-2.5 fraction in the Phoenix area, clouding any attribution of observed PM10-2.5 effects there to crustal particles alone, per se (p. 8-66).

At a later point, the CD concludes:

> Other studies suggest that particles of crustal origin are generally unlikely to exert notable health effects under most ambient exposure conditions. Some exceptions may include situations where crustal particles have come to be heavily contaminated by metals originally emitted as fine particles from smelting operations but deposited over many years on soils around smelters, steel mills, etc. (see Item 10, below). Also, in some U.S. cities (especially in the NW and the SW) where PM10-2.5 tends to be a large fraction of PM10, measurements, coarse thoracic particles from woodburning are often an important source during at least some seasons. In such situations, the relationship between hospital admissions and PM10 may be an indicator of response to coarse thoracic particles from wood burning (p. 8-339).

Thus, with respect to crustal material – which is the primary component of fugitive dust and most other coarse PM – the CD concludes that there is virtually no evidence of harm, with the possible exception of local sites subject to unusual circumstances.

This is confirmed in the CASAC Reports and the EPA Staff Paper. This issue first arose in the June 6, 2005 CASAC report on the second draft SP, in which the Committee closed on all major aspects of the SP except the discussions of coarse PM. The Committee's report includes several passages emphasizing the differences

between the urban coarse PM studied in the epidemiological reports and others types of dusts:

> Because the evidence for the toxicity of PM10-2.5 comes from studies conducted primarily in urban areas and is related, in large part, to the re-entrainment of urban and suburban road dusts as well as primary combustion products, there is concern that the associations of adverse effects with PM10-2.5 may not apply to rural areas where the PM10-2.5 is largely composed of less-toxic components of windblown soil or products of agricultural operations for which there is either no or limited evidence of health issues (p. 7).
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> One of the major reservations expressed by the Panel in recommending a 24-hour PM10-2.5 NAAQS related to the non-specificity of the PM10-2.5 mass metric. Given that most evidence indicates that the component of the coarse fraction in most rural areas has little or no toxicity at environmental concentrations, it was felt important to qualify the PM10-2.5 standard by somehow allowing exceptions for regions where the coarse fraction was composed largely of material that was not contaminated by industrial- or motor vehicle traffic-associated sources. (p. 8).

In the final SP, EPA staff responded to the Committee's recommendations:

> Beyond the urban and rural distinctions discussed above, staff has also considered the extent to which there is evidence of effects with exposure to ambient thoracic coarse particles in communities predominantly influenced by agricultural or mining sources. For example, in the last review, staff considered health evidence related to long-term silica exposures from mining activities, but found that there was a lack of evidence that such emissions contribute to effects linked with ambient PM exposures (EPA, 1996b, p. V-28). Similarly in this review, there is an absence of evidence related to such community exposures . . . it is unlikely that such sources contribute to the effects that have been observed in the recent urban epidemiologic studies.
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> Ambient PM10-2.5 exposure is associated with health effects in studies conducted in urban areas, and the limited available health evidence more

> strongly implicates coarse particles from industrial and traffic-related sources than from uncontaminated soil or geologic sources. The limited evidence does not support either the existence or the lack of causative associations for community exposures to agricultural or mining industries. Given the apparent differences in composition and in the epidemiologic evidence, it is not appropriate to conclude that evidence of associations with health effects related to urban coarse particles would also apply to nonurban or rural coarse particles. Collectively, the evidence suggests that a more narrowly defined indicator for thoracic coarse particles should be considered that would protect public health against effects linked with thoracic coarse particles present in urban areas. Such an indicator would be principally based on particle size, but also reflect a focus on those thoracic coarse particles that are generally present in urban environments (p. 5-56-57).

The evidence presented in the draft ISA provides further support for these findings. The draft explains that the more recent studies of dust storms in the U.S. and Canada "found no excess risk of cardiac or respiratory hospital admissions despite hourly PM10 levels >100 µg/m3 (Bennett et al., 2006)" (p. 6-77). It also notes that studies of dust storms in Asia and Eastern Europe report increased risk associated with dust storms, but the PM10 concentration levels and likely composition in these studies are not discussed in detail.

Despite these findings, none of the conclusions reprinted above from the current CD and related proceedings is presented in the draft ISA. The next draft should include the discussions of crustal material presented in the current CD and other materials, as supplemented by the more recent evidence discussed in the current draft.

<u>Contamination by Fine PM</u>

In several places, the preamble to the 2006 PM NAAQS regulations states that coarse crustal PM may need to be regulated in urban areas because it can become contaminated after it is emitted from a primary source and then emitted again as reentrained dust. The Coalition had commented on the proposed rule that we were not aware of any evidence in the record supporting this conclusion. We submitted a letter prepared by Dr. John Richards demonstrating that the record does not provide a technical basis for concluding that coarse mode particles, and especially crustal coarse mode particles, serve as important carriers of urban area contaminants. However, EPA did not respond to these comments in the final rules.

The Coalition is once again submitting Dr. Richards' letter on this issue. His primary conclusions are as follows:

- Coarse particles have less capability than fine particles of serving as a carrier because (i) coarse particles have much less surface area per unit of mass than fine mode particles, (ii) the surfaces of most coarse particles are not effective adsorbents, (iii) coarse mode particles are available for mass transfer for much shorter time periods, and (iv) most mechanisms for mass transfer of chemical contaminants to the surfaces of particles are much more effective for fine mode particles.

- Coarse mode particles have less capability than fine mode particles to initiate adverse health effects because (i) coarse particles have a limited atmospheric residence time and have less opportunity to reach people in both outdoor and indoor areas, and (ii) coarse mode particles are captured and removed from the respiratory tract with much greater efficiency than fine mode particulate matter.

- Any attempt to regulate urban contaminants "carried" on the surface of coarse mode particles will be ineffective because (i) contaminants are captured and "carried" much more effectively on fine mode particles than on coarse mode particles, and (ii) the small amount of "carried" contaminants that might be present on coarse particles are subject to the relatively short atmospheric residence times, the limited dispersion distances, and the increased respiratory tract capture and removal efficiencies that are characteristic of coarse particles in general.

These issues should be discussed in the next draft of the PM ISA.

Coarse PM Monitor Bias

On several prior occasions, the Coalition has submitted evidence of a substantial oversampling bias inherent in the coarse PM monitoring regime. The draft ISA addresses this issue as follows:

> Several papers (Buser et al., 2007a, b, c) published since the 2004 PM AQCD claim that the EPA FRM samplers for PM10 (and PM2.5) "oversample certain agricultural and other source emissions." These claims are based on the erroneous assumption that the "true" PM10 concentration is what would be given by a PM10 sampler that excluded all particles greater than 10 µm aerodynamic diameter and included all particles less than 10 µm. As noted earlier (Section 2.2) the legal definitions for PM2.5 and PM10, as defined in the Code of Federal Regulations include both a 50% cut-point and a penetration curve. For PM10, the 50% cut-point of 10 µm diameter means that 50% of particles

> with aerodynamic diameter of 10 ± 0.5 µm are removed by the inlet and 50% pass through the inlet and are collected on the filter. The penetration curve specifies, as a function of particle size, the fraction of particles larger than 10 µm that pass through the inlet and the fraction of particles less than 10 µm that are intercepted by the inlet. No effort was made in the development of the FRM to have the PM10 sampler collect all particles less than 10 µm and no particles greater than 10 µm since the sampler was designed to collect a fraction of atmospheric particles similar to the "inhalable" or thoracic fraction, i.e., those particles that would pass through the nose and throat and reach the lungs (Miller et al., 1979). Thus, the FRM PM10 sampler correctly and intentionally collects particles greater than 10 µm (p. 3-17).

This argument greatly oversimplifies the evidence of coarse PM monitor bias, which is much larger than could be caused by these aspects of the FRM. The accuracy of the FRM for PM10 and PM2.5 was demonstrated on the basis of the samplers' performance for ambient particles with mass median diameters (MMD) of 10 and 2.5 micrometers, respectively. However, some agricultural, mining or industrial dusts have much larger MMDs. For example, agricultural dusts have reported MMDs as large as 24 micrometers. Data presented in published, peer-reviewed studies demonstrate that use of FRM samplers to measure PM10 concentrations of dusts with MMDs significantly above 10 micrometers will result in concentrations as much as 300% higher than the measured concentrations of ambient PM with MMDs equal to or less than PM10.[4]

This phenomenon is referred to as "over-sampling" errors for dusts with larger MMD (Buser, et. al, 2002, Wang, et. al., 2004 and Capareda, et al., 2004). The pre-separator for a PM10 or PM2.5 FRM sampler will remove some of the larger particles, but some of those large particles will also pass through the pre-separator to the filter (or measuring sensor such as the TEOM). The penetration of these larger particles – which are much larger than the thoracic fraction -- can result in measurements of PM10 and PM2.5 concentrations that are substantially higher than the concentrations that would otherwise be measured .

PM10 samplers, as defined in 40 CFR Part 50, Appendix J, collect all of the fine particles and part of the coarse particles. As discussed in the draft ISA, the upper cut-point is defined as having a 50% collection efficiency (d50) at 10+0.5 µm. The slope of the collection efficiency is defined in amendments to 40 CFR, Part 53. Hinds (1982)

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[4] This section is based on a "White Paper" prepared by the faculty of the Texas A&M University Center for Agricultural Air Quality Engineering and Science (CAAQES). A copy of the paper, titled "CAAQES Position Paper on EPA's PM Coarse Standards, White Paper #2" (July 15, 2004) is attached. While this paper is marked as a draft, the Coalition has received the permission of the CAAQES faculty to rely on it here. The White Paper includes a bibliography of the studies cited in this section. The White Paper is reprinted on the CAAQES website, as are copies of many of the cited studies. It is our impression that the CAAQES faculty already has provided EPA staff with copies of all or most of these studies. The Coalition will work with the CAAQES faculty to provide EPA staff with copies of these or any other relevant material that EPA staff may wish to review.

reported a slope of 1.5 µm which would mean that some particles larger than 10 µm are captured and at 50% efficiency, the amount of larger particles captured will be equal to that of smaller particles that were not captured, making the weight measurements for PM10 correct at 10 micrometers. But that is only the case when the PM10 sampler is sampling PM with a 10 µm MMD. When the PM has a MMD larger than 10 µm, the amount of larger particles captured will be greater than that of smaller particles that were not captured, thus over sampling error will occur (Buser, et al. 2001).

These studies also indicate that PM10 over-sampling error is compounded as a result of a shift in the particle size cutpoint when the sampler is exposed to dusts of larger particle size. As particle size increases, the sampler cut-point shifts and the sampler captures more large particles, creating another over-sampling bias in PM10 concentration measurements (Wang, et al, 2003). This also has been demonstrated for the FRM PM2.5 sampler, which is assumed to have a d50 of 2.5 µm and a slope of 1.18 (EPA, 2001). Studies by Buch (1999) and Pargman, et al. (2001) showed that there was a shift in the cut-point for FRM PM2.5 to 2.7+0.41 µm and a slope of 1.32+.03 µm. This shift in the cutpoint, therefore, creates a similar over sampling problem for PM 2.5.

The effects of the over-sampling bias have been demonstrated in field sampling performed at the Texas A&M University Center for Agricultural Air Quality Engineering and Science (CAAQES). The CAAQES field samples demonstrate that the cut-point of PM10 FRM samplers shifted from 10 micrometers to much larger particles in the presence of dust with a mid-size of 20 micrometers. In effect, the PM10 concentration measurements with FRM PM10 samplers measured 600 $\mu g/m^3$ whereas the actual concentration was approximately 200 $\mu g/m^3$.

The CAAQES findings demonstrate that measurements of PM10 concentrations dominated by rural windblown dust, soils, agricultural and other similar operations (e.g., cotton gins, grain elevators, feed mills and oil mills) using federal reference method (FRM) samplers are biased. The magnitudes of these biases are large and will result in inappropriate regulation of these operations. The next draft of the ISA should contain a more detailed discussion of this coarse PM sampling bias.

Conclusion

For the reasons presented above, the next draft of the PM ISA should be modified to:

1. Provide clear criteria for selection and evaluation of key studies;

2. Focus more clearly on the limitations of the health effects evidence for PM10 and PM10-2.5, as in the current CD;

3. Include the studies discussed in the current CD indicating absence of harm from exposure to coarse crustal material; and

4. Address the coarse PM monitor bias in more detail.

Respectfully submitted,

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