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U. S. Environmental Protection Agency
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Washington, DC 20460

RE: Draft Document Related to the Review of the National Ambient Air Quality Standards for Particulate Matter: Docket ID No. EPA-HQ-OAR-2007-0492

The American Chemistry Council (ACC)[1] appreciates the opportunity to submit comments on the U.S. Environmental Protection Agency's (EPA or the Agency) Policy Assessment for the Review of the Particulate Matter (PM) National Ambient Air Quality Standards (NAAQS) – Second External Review Draft (75 FR 39253, July 8, 2010). ACC supports health, safety and environmental protection policies that incorporate objective, realistic, comprehensive and scientifically balanced analyses. Our members operate facilities throughout the country, and are subject to compliance with PM emission limits.

We continue to believe that the process by which EPA reached its conclusions and recommendations is flawed. EPA did not address in adequate detail how studies for review were selected and, as important, why certain studies were left out. Numerous studies that question the

[1] *The American Chemistry Council (ACC) represents the leading companies engaged in the business of chemistry. ACC members apply the science of chemistry to make innovative products and services that make people's lives better, healthier and safer. ACC is committed to improved environmental, health and safety performance through Responsible Care®, common sense advocacy designed to address major public policy issues, and health and environmental research and product testing. The business of chemistry is a $674 billion enterprise and a key element of the nation's economy. It is one of the nation's largest exporters, accounting for ten cents out of every dollar in U.S. exports. Chemistry companies are among the largest investors in research and development. Safety and security have always been primary concerns of ACC members, and they have intensified their efforts, working closely with government agencies to improve security and to defend against any threat to the nation's critical infrastructure.*

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existence of an association between particulates and health effects were not mentioned at all. ACC has repeatedly submitted comments on these studies as well as on other important deficiencies in the Integrated Science Assessment, the Qualitative Risk Assessment, and the first draft of the Policy Assessment, and these comments have yet to be addressed in any EPA document pertaining to the PM NAAQS review. We have serious concerns about how this NAAQS review process has been conducted, particularly with the lack of EPA responses to any of the detailed technical comments submitted by stakeholders throughout this review.

The second draft Policy Assessment differs only slightly from the first draft released earlier this year. The second draft now contains a brief Executive Summary, and two very brief sections (2.5 and 3.5), that touch on key uncertainties and areas for future research. We are concerned that the EPA Administrator will be reviewing a final Policy Assessment that lacks a clear picture of the limitations of the available science, and making her determination on whether or not to revise the $PM_{2.5}$ NAAQS based on this document.

We continue to find the conclusions drawn in the second draft Policy Assessment and the documents upon which it relies to be equally problematic. We do not believe that EPA has sufficiently made the case for causality between exposure to $PM_{2.5}$ at current levels in the U.S. and any adverse health impacts in human populations. We continue to note that EPA primarily utilizes the American Cancer Society (ACS) II cohort for assessing the long-term impact of fine PM on mortality. However, the Krewski *et al.* (2000) study found that SO_2 and not fine PM was associated with mortality. All subsequent ACS II studies did not address this finding. Furthermore, the ACS II data is not publicly available. EPA should not base a rulemaking on information which cannot be scrutinized by the public. Finally, EPA has not acknowledged that concentration-response (CR) functions may not be linear, which would then result in different estimates of benefits of lowering the $PM_{2.5}$ NAAQS in the risk assessment.

For these reasons, we continue to believe that the recommended alternative $PM_{2.5}$ standards given in the second draft Policy Assessment are without scientific merit. Specifically, EPA has not demonstrated a justification for lowering the levels of either the current annual or 24-hour NAAQS $PM_{2.5}$ standard or for making the form of the annual standard more stringent.

Our detailed comments are attached for your consideration. If you would like to discuss any of the comments in more detail, please contact me at (703) 741-5219 or lorraine_gershman@americanchemistry.com.

Very truly yours,

Lorraine Krupa Gershman
Director, American Chemistry Council

Attachment

ACC COMMENTS ON POLICY ASSESSMENT FOR THE REVIEW OF PARTICULATE MATTER NATIONAL AMBIENT AIR QUALITY STANDARDS

Second External Review Draft, June 2010
Docket ID Number EPA-HQ-OAR-2007-0492

I. Introduction

These comments present the results of ACC's review of EPA's Second External Review Draft of the Policy Assessment for the Review of the Particulate Matter National Ambient Air Quality Standards ("Policy Assessment"), which was made publicly available in June 2010. Our review of the second draft of the Policy Assessment deals with the primary standard and focuses on whether or not the recommendations and criticisms we submitted to EPA on the first draft were addressed, as well as major revisions to the second draft.

To provide a framework for these comments, we summarize the major comments we submitted on the June 2010 draft.

1. *EPA's conclusions are not based on a fair and impartial review of the literature.*

   EPA failed to recognize the limitations of observational epidemiology studies, especially when the risks are small. Relevant publications that called into question the causal link between exposure to fine particulate matter (PM) at current ambient levels and adverse effects on human health were not considered.

2. *Sources of uncertainty in air pollution epidemiology were not fully explored.*

   EPA failed to address that many of the implausibly large reported risks are more plausibly explained by differences in socioeconomic status than fine PM concentrations. The Policy Assessment does not properly identify what constitutes a susceptible sub-population.

3. *Statistical models used are inadequate.*

   Many of the papers EPA relied upon to draw its conclusions were based on the Cox proportional model, a model that is generally deficient for use in epidemiologic studies of air pollution. EPA needs to recognize that simply entering a covariate representing a confounder into a statistical model does not indicate that the confounder has been adequately controlled.

4. *Results of the analyses are highly model dependent.*

   Inclusions of copollutants in analyses can make a significant difference to the estimate of the fine PM effect. While EPA discusses uncertainties in the air pollution epidemiology literature, it does not adequately recognize that some of the principal studies it relies on have shown an association not of fine PM with mortality, but of SO_2 with mortality.

5. *Serious inconsistencies exist both within and between studies.*

   EPA does not acknowledge nor address inconsistencies among studies it relies upon.

6. *Many of the recent epidemiologic studies report biologically highly implausible results.*

   The risks reported in some studies that EPA relies on are so large that they defy biological plausibility suggesting strongly that it is not the differences in air pollution levels that are responsible for the observed differences in death rates.

7. *Coefficients and exposure-response relationships adopted by EPA for the risk assessment are suspect.*

   Neither the choice of the concentration response (CR) functions nor the assumption of linearity without a threshold is supported by the science.

Mostly on the basis of the above issues, we concluded in our comments on the first draft of the Policy Assessment that the document was seriously flawed. We pointed out specifically that:

- The case for causality between exposure to fine particles at current levels in the U.S. and any adverse health outcomes in human populations has not been made.
- Studies relied upon by EPA should not form the basis for regulatory decisions.
- Relevant data relied upon by EPA were not made available to stakeholders.
- EPA has not demonstrated a justification for lowering the levels of either the current annual or 24-hour NAAQS $PM_{2.5}$ standard or for changing the form of the annual PM_{10} standard.

II. The Second Draft

With a few exceptions, the second draft echoes the EPA staff conclusions drawn in the first draft regarding the inadequacy of the current PM standard to protect human health. It proposes two alternative standards for the $PM_{2.5}$ standard that differ only slightly from those proposed in the first draft. Specifically, it proposes alternative annual standards covering a range of 13 to 11 $\mu g/m^3$ rather than first draft's recommendation of 13 to 10 $\mu g/m^3$. It also proposes alternative 24-hour standards in the range of 35 to 30 $\mu g/m^3$ instead of 35 to 25 $\mu g/m^3$ as proposed in the first draft. With respect to the coarse PM_{10} fraction, the second draft repeats the first draft's recommendation to either retain the current standard or change the form of the 24-hour standard. If the latter option were chosen, the second draft further recommends that a level of 85 to 65 $\mu g/m^3$ be set for the new standard. The second draft inserts an Executive Summary and provides more explanation of EPA's reasoning in selection of the alternatives. The second draft also adds sections describing key uncertainties and areas for future research and data collection that were not completed in the first draft.

EPA states in Chapter 2 that its approach in the second draft took into account advice from CASAC and public comments on the first draft. We cannot speak to the extent that public comments submitted by others were taken into account, but a review of the second draft reveals that the comments ACC submitted on the first draft, as well as similar comments ACC submitted on the Integrated Science Assessment, were virtually ignored in their entirety by EPA. The scientific case for revision of the current standards is made in sections 2.2.1, 2.2.2 and 2.2.3. These sections lay out the case made in the first draft with no acknowledgement of the concerns we expressed on the first draft. Thus our comments on the first draft are applicable in large part to the second draft. In these current comments, we show that they cast significant doubt on the major conclusions drawn in the second draft.

<u>Causality of the Association between Exposure to $PM_{2.5}$ and Adverse Effects</u>

EPA asserts that the epidemiological literature supports the existence of a causal relationship between long-term exposure to $PM_{2.5}$ and all-cause mortality and cardiovascular effects including mortality, and morbidity. To reach these conclusions, EPA relies on a number of large U.S. cohort studies published since the previous 2006 NAAQS $PM_{2.5}$ review. Among these are extended analyses of the American Cancer Society (ACS) and Harvard Six Cities Studies, and the Women's Health Initiative and Medicare studies. What EPA does not point out is that these studies are all observational in nature and accordingly suffer from the overall limitations of such types of studies, specifically when the estimated risks are small.

Conclusions drawn from observational epidemiology studies, whether time-series or long-term, are particularly subject to distortion by residual confounding because of the small risks being estimated and the complexity of confounders that must be controlled.

The limitations of observational epidemiology are illustrated by studies of postmenopausal hormone replacement therapy (HRT) and anti-oxidants and cancer and heart disease. In both of these cases, the initial interpretation of the studies and the recommendations they led to were shown to be strikingly incorrect. They illustrate the failure of epidemiology to reach unbiased (in the statistical sense) conclusions when estimated risks are small, even when epidemiology is bolstered by experimental evidence and biological plausibility.

Epidemiologic studies of air pollution are in some fundamental respects much weaker than the observational studies of HRT and of anti-oxidants in that exposure to air pollution is not known on the individual level. Thus, epidemiologic studies of air pollution use either an ecologic or semi-ecologic design, generally considered to be the weakest study designs in epidemiology. Recent attempts to characterize exposure to air pollution better, such as in the WHI study (Miller *et al*., 2007), which the agency relies on for its conclusions regarding cardiovascular morbidity, lead to estimates of risk that are far too large to be biologically plausible. This study and others, such as California Teachers' Study (Ostro *et al.*, 2009) and the Nurses' Health Study (Puett *et al.*, 2009) present estimates of risk that suggest that fine particle pollution at contemporary levels in the U.S. is far more toxic than a 20-40 pack-year smoking habit (a pack-year is equivalent to smoking one pack a day over the course of a year). EPA and CASAC need to take a hard look at these results and evaluate them critically before using them for regulatory decisions with far-reaching implications. If EPA believes that fine PM presents a risk greater than from 20-40 pack-years of cigarette smoking, it should clearly state as much.

We also note that in arriving at its determinations of causality, based on the lack of citations, EPA did not appear to consider several relevant papers calling into question the causal link between exposure to fine PM at current ambient levels and adverse effects on human health. For example, a 2005 publication by Grahame and Schlesinger questions the interpretation of the ACS II study and would appear to be highly relevant to the issues discussed. Papers by Moolgavkar (2005, 2006, and 2007) discussing methodological issues in the analyses of epidemiologic studies of air pollution are equally relevant. Another example is provided by the omission of a paper by Wittmaack (2007) calling into question the interpretation of the so-called 'interventional' study in Dublin by Clancy *et al.* A recent paper on the toxicity of sulfates and nitrates (Reiss *et al.*, 2007) clearly relevant to the ISA, does not appear to have been considered by the Agency. Similarly, a paper by Vedal and Dutton (2006) analyzing mortality in Denver in relation to high levels of fine PM following wild fires and reporting no associations does not appear to have been considered by the Agency. A recent HEI report by Peters *et al.* (2009), which finds no association between fine PM pollution and mortality in Erfurt, Germany, also is not addressed by the Agency. These are just some examples of relevant literature that should have been reviewed and addressed by EPA. These omissions were pointed out in comments we submitted on the ISA and first draft of the Policy Assessment, and remain absent from the second draft.

The Alternative Standards

The policy alternative standards recommended for consideration by the EPA Administrator in the second draft are strongly based on the results from a number of epidemiological studies - both long-term exposure studies (e.g., Krewski *et al.* 2009, Miller *et al.* 2007, Etfim *et al.* 2008) and short-term exposure studies, (e.g., Dominici *et al.* 2006, Bell *et al.* 2008, Zanobetti and Schwartz 2009) - that show effects from $PM_{2.5}$ exposure. As discussed below, each of these studies has serious limitations and/or inconsistencies that cast serious doubts on whether they should have been used to establish alternative standards.

Krewski et al. (2009)

Krewski *et al.* (2009) is an update and extension of the original ACS II study of Pope *et al.* (1995). Previous extensions include Krewski *et al.* (2000), and Pope *et al.* (2002 and 2004). The Krewski *et al.* (2000) analyses showed that including SO_2 markedly reduced the magnitude of the PM association to a level that was not statistically significant. After that original observation, and despite repeated requests that joint pollutant analyses be performed, none of the subsequent analyses of this cohort reported results of joint pollutant analyses even though, in single pollutant analyses, SO_2 was significantly associated with mortality. The external advisory panel of HEI opined that the lack of multi-pollutant analyses was a limitation of Krewski *et al.* (2009). EPA goes to great lengths to justify its choice of the CR coefficient from Krewski *et al.* (2009), but it completely ignores that in the only joint pollutant analyses of this data set ever performed, fine PM was not significantly associated with mortality. For this reason alone, the choice of a coefficient from the Krewski *et al.* (2009) analysis is indefensible. The saga of the many analyses of the ACS II cohort illustrates dramatically the importance of basing regulatory decisions on data that are available to all stake-holders. Despite repeated requests, crucial analyses were not conducted by individuals who were granted special access to the data. Agency regulatory policy should not be based on data that have been subject to incomplete, and possibly biased, analyses.

There are other problems as well with the Krewski *et al.* (2009) analyses. The authors conducted spatial analyses in the cities of New York and Los Angeles and found statistically significant associations of fine PM and mortality in Los Angeles, but not in New York. The original Krewski *et al.* (2000) analyses had reported much stronger association in the northeast than in California. Moreover, the original first-stage NMMAPS time-series analyses had reported statistically significant results in New York, but not in Los Angeles. It is incumbent upon the Agency to address these inconsistencies and infirmities if it is going to rely on these analyses for its regulatory decision-making.

Miller et al. (2007)

Researchers at the University of Washington (Miller *et al*., 2007) investigated the association between exposure to air pollution and the incidence of cardiovascular events, including mortality, among more than 65,000 post-menopausal women between the ages of 50 and 79 enrolled in WHI between 1994 and 1998. The women were followed up until August 2003. As in other long-term studies of air pollution, the principal analytical tool was the Cox proportional hazards model. Although this study is billed as an air pollution study, the main focus of investigation was fine PM. Other pollutants appear to have been considered only perfunctorily. In particular, the within-city analyses, which yielded the highest risks for fine PM, appear not to have been conducted for the other pollutants.

The investigators estimated risks for cardiovascular events including death, associated with fine PM pollution. They computed a between-city risk (risk associated with differences in average PM levels between cities) and also a within-city risk (risk associated with differences in fine PM levels within cities). Their overall risk (RR=1.76) and their between-city (RR=1.63) and within-city risks (RR=2.28) for cardiovascular death associated with a difference in fine PM concentration of 10 $\mu g/m^3$ were considerably higher than those reported in previous studies. A few points are worth noting here. If fine PM mass concentration is determining the risk, it is difficult to see why the within-city risk is so much larger than the between-city risk. Second, the reported risks are implausibly high. For example, the within-city relative risk of dying from cardiovascular disease is reported to be 2.28, as noted above. By way of comparison, the relative risk associated with smoking (which the authors do not report in this paper) in Pope et al. (2004) is 1.94 for a current smoker. In a direct examination of smoking-associated cardiovascular risks among women, Burns et al. (1996, chapter 3, table 13) reported that the relative risk of dying from coronary heart disease for a woman aged 65-69 and smoking 40 cigarettes per day is 1.86. Thus, in this paper, the within-city risk associated with a 10 $\mu g/m^3$ increase in fine PM is larger than, and the overall risk is about the same as, the risk associated with smoking 40 cigarettes per day, findings that defy plausibility.

To put this observation in perspective, each cigarette contains 20-40 mg of tar (IARC, 1986). Most of this tar is converted to fine PM when a cigarette is smoked. If 50% of the fine PM is inhaled, a single cigarette delivers about 10,000-20,000 micrograms of combustion fine PM to the lungs. By contrast, a normal adult inhales about 15 cubic meters of air every day (EPA, 2009). Even with an ambient fine PM concentration of 100 $\mu g/m^3$, far above the current standard, an adult would breathe in 1500 micrograms of fine PM daily, far less than the fine PM delivered by a single cigarette. In this study, the difference in fine PM concentrations between the most polluted and least polluted cities is about 25 $\mu g/m^3$. A simple calculation based on the overall result reported in the paper then shows that a woman moving from the least polluted city to the most polluted city would increase her risk of dying from heart disease four-fold, far higher than the risk imposed by smoking 40 cigarettes per day. Clearly,

these discrepancies in risks are far too large to be ignored. And yet, the Agency seems to accept these findings uncritically.

Other reported results are inconsistent with earlier epidemiological studies of fine PM and mortality. For example, whereas the reanalysis (Krewski *et al*., 2000) of the ACS II study found strong modification of the risk associated with fine PM by socio-economic status as measured by level of education, this study found no such effect. On the other hand, this study reports strong effect modification of fine PM effects by body mass index (BMI) with risk increasing with increasing BMI. In contrast, the reanalysis of the ACS II study reported no such finding.

Etfim et al. (2008)

Etfim *et al.* (2008) is a study based on Medicare data and was conducted in the same areas as the Harvard Six Cities and the ACS II studies. Therefore, it is not surprising that the authors reported similar results. However, the study design is much weaker than the design used for the Six Cities and ACS II studies in that no individual-level information on smoking was available. Smoking was controlled using crude proxy ecologic measures such as lung cancer and COPD rates. This study should not be touted as an independent verification of the findings of the Six Cities and ACS II studies.

Zanobetti and Schwartz (2009), Dominici et al. (2006) and Bell et al. (2008)

These studies are all extensions of the original NMMAPS studies. In comments on previous EPA documents, ACC has offered comprehensive reviews and critiques of the NMMAPS studies. These comments apply to these three studies as well.

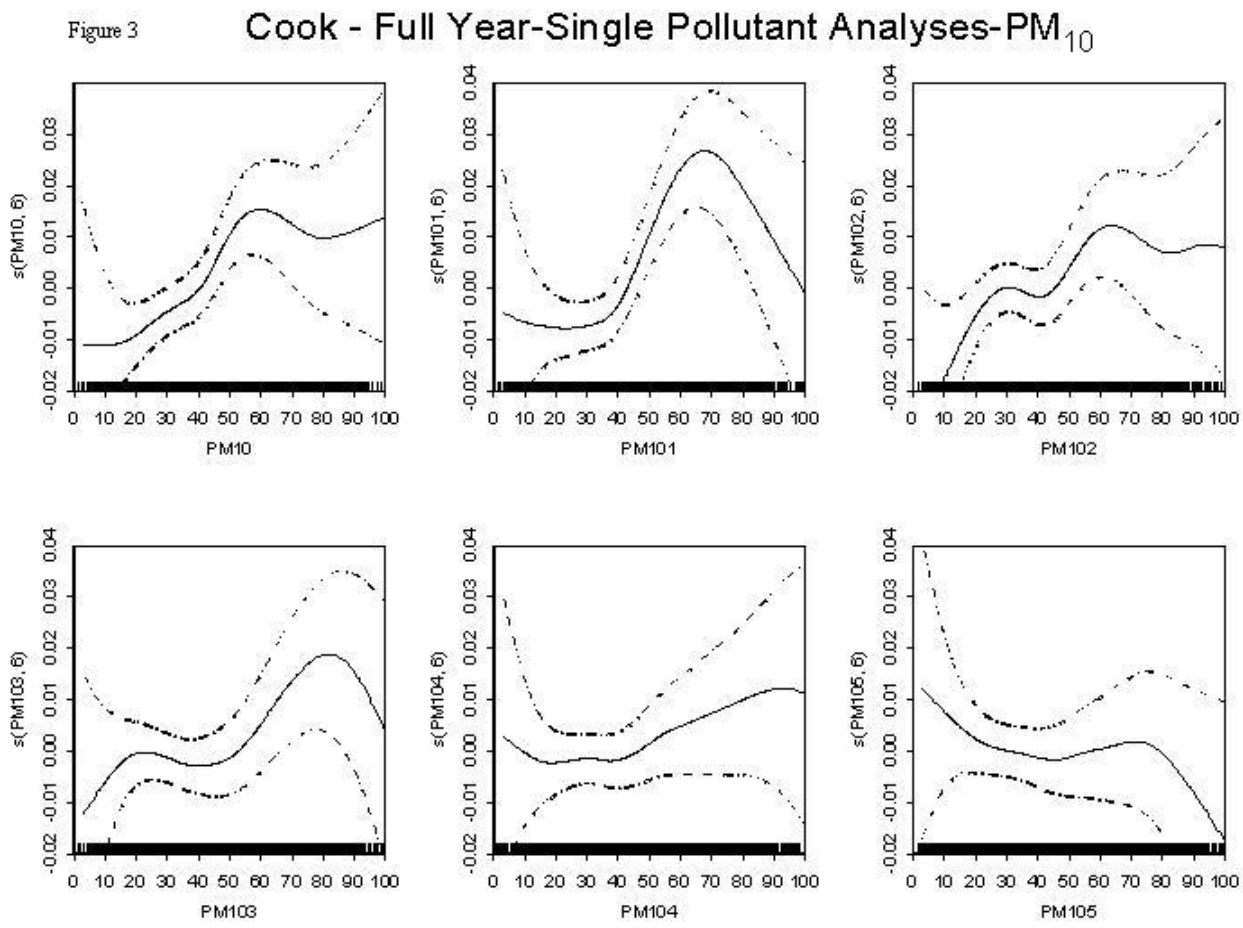
EPA assumes that concentration-response relationships between PM and adverse effects on human health are universally (log) linear[2] without a threshold concentration below which there is no effect. However, there is considerable difference of opinion in the scientific literature on this issue. Certainly there are papers that report a linear non-threshold concentration-response, but there are others that come to opposite conclusions. For example, Smith *et al.* (2000) investigated the issue of a threshold in the concentration-response relationship between PM and mortality in Birmingham, Alabama. The authors concluded, "[w]e also find evidence of a threshold effect, with the greatest increase in mortality occurring above 50 $\mu g/m^3$…" In addition, they reported that the results of statistical analyses were quite sensitive to the choice of model used for analyses, in particular to whether or not relative humidity was included in the analyses.

[2] It is widely acknowledged that fine PM is a mixture of components that determine its toxicity, with different components having different toxicities. The composition of the fine PM mixture varies from city to city (e.g., Laden *et al*., 2000) and from day to day within a given city. In fact, recent attention has moved away from fine PM mass to the specific components of fine PM. Simple algebra then shows that the exposure-response relationship for fine PM cannot be linear, even if we assume that the exposure-response relationship for each toxic component of fine PM is linear.

Although there may be some data sets in which concentration-response relationships for individual pollutants are linear, in general these can be highly non-linear. An example of such non-linearities is shown in Figure 1, based on analysis of mortality in Cook County (Moolgavkar, 2003). The figure shows concentration-response relationships for PM_{10} with lags of from zero to five days. These curves are highly non-linear and many of them show evidence of a threshold.

If PM were causally associated with mortality, one would expect the exposure-response relationship to be increasing, possibly with a threshold. One would not expect, however, the risk of mortality to level off or to decrease at high concentrations of PM. Yet, this is precisely the behavior exhibited by the curves in Figure 1. In particular, there is an actual decrease in risk with increasing concentration of PM_{10} above about 50 $\mu g/m^3$. This observation suggests the statistical associations seen in these analyses may not reflect a cause and effect relationship.

With co-pollutants in the models, the concentration-response relationship for PM is even more difficult to interpret. Because of the strong non-linearities in the concentration-response relationship, the estimated coefficient from a linear fit cannot be interpreted as a measure of the increase in adverse health effects to be expected for a unit increase in PM. That is, it is inappropriate to conclude that there is a linear exposure-response relationship without a threshold between PM mass concentrations and mortality.



<u>Figure 1</u>: Results of the smoothed exposure-response analyses in Cook County. For each panel, the concentration of PM_{10} (in micrograms per cubic meter) is shown along the x-axis, the response (log of the relative risk for daily mortality) along the y-axis. Six panels are shown corresponding to lags of 0 to 5 days. The density of the "rug" (i.e., the thick black line) along the bottom of the x-axis is an indicator of the amount of data on which the response curves are based. The dashed curves represent the 95% confidence intervals. Note the highly non-linear exposure-response relationship. There is a clear indication of thresholds at lags of 0, 1 and 4 days.

Although the linear CR function for long-term mortality adopted by EPA is taken from the latest analysis of the ACS II cohort, previous investigations of the issue of linearity based on the ACS II cohort have suggested that the relationship is considerably more complex. For example, Abrahamowicz *et al*. (2003) reported significant non-linearities in the exposure-response relationships for both fine PM and sulfates. Further, the authors reported evidence of a threshold for sulfates. In a recent paper, Pope *et al.* (2009) examined critically the exposure-response relationship between fine PM and

cardiovascular disease mortality and concluded, "[o]verall these results suggest a nonlinear exposure-response association with both dimensions of cumulative exposure, the level of ongoing contemporaneous exposure and the duration of exposure." Moreover, Pope *et al.* conclude that the CR function could be supra-linear. Therefore, all possibilities for the CR function - linear, sub-linear with a threshold, and supra-linear - have been described in the literature. In fact, the Agency should acknowledge that a (log) linear relationship is logically inconsistent with the currently held view that different constituents of fine PM have different toxicities (see footnote 1).

Other Interpretations of Studies EPA Relies On

EPA relies on a study by Laden *et al.* (2006) to assert that declines in fine PM pollution have led to decreases in death rates. It uses a recent study by Pope *et al.* (2009) to estimate the increase in life expectancy that would accrue if fine PM pollution were reduced. However, EPA does not recognize the deficiencies of these studies, neither does it recognize, let alone discuss, alternative, equally plausible explanations of the findings reported.

Laden et al. (2006)

Laden *et al.* (2006) extended follow-up of the Six Cities cohort by eight years to 1998. The authors continued to use proportional hazards modeling to analyze the extended follow-up data and concluded that "[i]mproved overall mortality was associated with decreased mean $PM_{2.5}$…" The ISA cites this paper in support of the contention that cleaner air leads to health benefits saying, "[c]oupled with the results of the original analyses (Dockery *et al.*, 1993), this study strongly suggests that a reduction in fine PM pollution yields positive health benefits." However, the ISA fails to cite and address comments by Gamble and Nicolich (2006) suggesting that the model used by Laden *et al.* (2006) is incorrect and overestimates risk. Moreover, the ISA fails to discuss major limitations of this study. $PM_{2.5}$ measurements were discontinued early; later $PM_{2.5}$ levels were estimated from PM_{10} levels rather than measured directly. For this reason alone, this study should not be used by EPA for risk assessment purposes. Furthermore, the investigators compared death rates of individuals in different birth cohorts 8 years apart. Specifically, individuals of a given age in the second period were born 8 years later than individuals of that age in the first period. There are a myriad of factors that determine death rates in specific birth cohorts and to conclude that the decline in death rates in the second period, which occurred *pari passu* with a decline in fine PM pollution, was due to the decline in fine PM pollution is a gross over-interpretation of the results. Finally, as the paper by Villeneuve *et al.* (2002) clearly shows, the RR associated with exposure to fine PM declines with increasing age. Laden *et al.* are looking at a population that has aged 8 years in the second period. The decline in the RR in the second period could very well be attributable to this fact.

Further, the Agency fails to note that the Laden *et al.* paper shows clearly that, in the period 1990-1998, mortality was not higher in the cities with higher concentrations of

fine PM. Table 1 below is constructed from the results shown in Tables 1 and 2 in Laden *et al.* (2006).

If fine PM were associated with mortality at any concentration, one would expect to see higher death rates in the cities with higher concentrations of fine PM even in the follow-up period. But there is clearly no trend in risk with increasing concentration of fine PM, which suggests strongly that either the result reported in the original Six Cities Study was simply a statistical artifact or that there is a concentration below which no fine PM association with mortality is discernible in this study. Finally, in the original Six Cities Study, mortality was associated not only with fine PM but also with each of the gaseous pollutants except ozone. These pollutants also declined over the course of this study. Therefore, at best, the decline in mortality can be attributed to a decline in general air pollution levels rather than a decline in fine PM pollution, though we have little confidence in this conclusion.

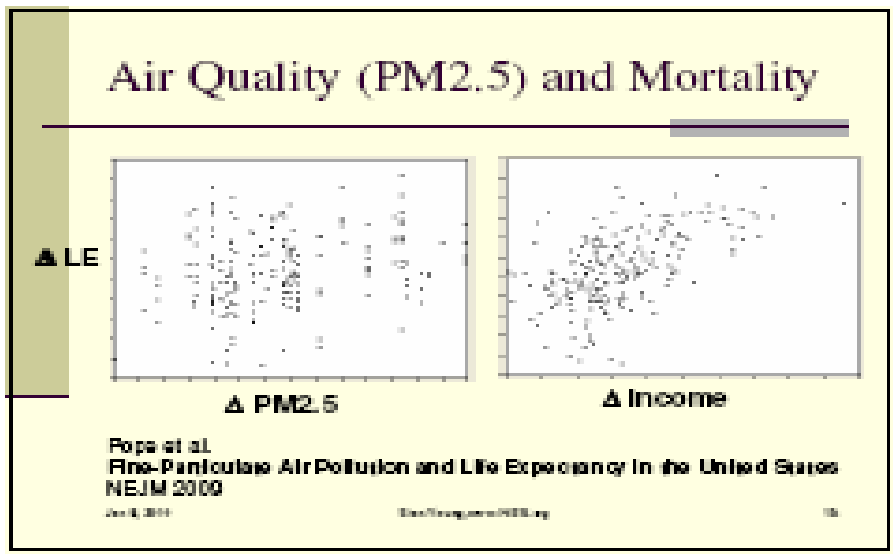
City	**Average fine PM concentration in micrograms per cubic meter.**	**Relative Risk**
Portage	10.2	1.00
Watertown	12.1	0.82
Topeka	13.1	1.01
St. Louis	13.4	0.96
Harriman	18.1	1.10
Steubenville	22.0	1.06

Table 1. ***Results compiled from tables 1 and 2 of Laden et al. 2006. The six cities in the study are arranged in order of increasing average concentrations of fine PM over the period 1990-1998. The relative risks are reported in the last column. There is no indication of an increase in risk with increasing fine PM concentration.***

EPA relies on a recent paper by Pope *et al.* (2009) to support its contention that a decrease in fine PM pollution leads to an increase in life expectancy. In fact, this paper presents an ecologic analysis covering essentially the same cities covered in the studies based on the ACS II cohort. It is, therefore, not surprising that there is an inverse correlation between fine PM pollution and life expectancy. However, the study design, being completely ecologic, is even weaker than the design of the ACS II study. Control of cigarette smoking is based on a surrogate measure and is particularly crude. Despite the association between SO_2 and mortality in the ACS II cohort, no attempt was made to consider any pollutant other than fine PM. It is remarkable that this study is being used to estimate the association between fine PM pollution and life expectancy when the hazard functions derived from the ACS II study could easily be used to estimate life shortening. Moreover, in view of the findings of a much stronger fine PM association in

the eastern U.S. than in the western U.S., Pope *et al.* should have looked at least these two regions separately. The results might have been quite different.

The following figure is taken from a recent talk by Dr. Stanley Young of the National Institute of Statistical Science at the 2010 annual meeting of the AAAS held at San Diego. It shows changes in life expectancy plotted against changes in income and changes in fine PM concentrations using the data from Pope *et al.* (2009). Clearly changes in income are much more strongly correlated with changes in life expectancy than are changes in fine PM concentrations. There have been large changes in smoking habits as well. ACC fails to understand how the Agency can believe that such strong confounders are adequately controlled in these simple regression analyses.



<u>Figure 2</u>: Particulate Air Pollution and Life Expectancy in the United States (New England Journal of Medicine, 2009)

Conclusions

EPA expends considerable effort in its cyclical review of priority pollutants. Years in the undertaking, highly technical Integrated Scientific Assessment (ISA) and Quantitative Health Risk Assessment (QRA) documents are prepared for each pollutant that form the scientific support for EPA staff's recommendations to the Administrator on deciding whether and how to revise the existing NAAQS. It is the Policy Assessment, however, that is the most critical document in EPA's review process. This document, in EPA's own words, is intended to "help 'bridge the gap' between the relevant scientific information and assessments and the judgments required of the EPA Administrator," (EPA, 2010). It is reasonable to expect that in making her judgments, the Administrator will rely primarily, if not entirely, on this document, rather than digesting the thousands of pages of detailed scientific information included in the ISA and QRA. For this reason, the Policy Assessment should present information in a manner that reflects the numerous uncertainties and shades-of-gray that accompany the interpretation of data. Not only would such an approach assist the Administrator in making decisions that rely on subjective judgment with far-reaching consequences, it would also go a long way in meeting EPA's stated goal of operating in an open and transparent manner.

It is therefore disappointing that the second draft of the Policy Assessment differs little from the first in presenting information without conveying to the reader the ambiguities and nuances that underlie many of the key studies that EPA relies upon. The two sections added to the second draft, Section 2.5 and Section 3.5, dealing with key uncertainties and areas for future research for fine and thoracic particulates respectively, cover a mere four pages, and convey nothing of the various interpretations that the data are subject to, many of which are discussed in these comments.

We are aware that many of our comments apply to conclusions drawn by EPA in the ISA and QRA, which EPA accepted uncritically and without further discussion in the Policy Statement. However, as indicated above, it is to the Policy Assessment that the Administrator will in all likelihood rely upon in making her decisions and it is here that the uncertainties must be laid out. For the very same reason, it is not sufficient for EPA to produce another, after-the-fact and stand-alone document to respond to external comments, as was done with SO_2. We believe that it is essential for the Policy Assessment to provide an evenhanded review of the epidemiological evidence integrating counterbalancing views of the conclusions that EPA draws, something that the second draft does not do.

Along these lines we note that the Executive Summary, a new section added to the second draft, simply summarizes the recommendations for alternative primary and secondary standards, without any qualification. We believe that this section should contain a discussion of the strength of the data supporting the alternatives. Without this discussion, the recommendations lose their credibility, an especially important consideration given the attention that an Executive Summary ordinarily gets.

We strongly believe, therefore, that this second draft requires substantial revision before it is presented to the Administrator for decision-making. Specifically, the Administrator should be given a much clearer understanding of, among other things, the limitations of observational epidemiology in establishing causal associations and the limitations of the studies EPA relies upon in establishing causality between exposure to fine particles and certain adverse effects. She should know that the principal studies of the long-term impact of fine PM on mortality are based on the ACS II cohort while an earlier analysis of a key closely-held data set, never subsequently repeated, showed that SO_2 and not PM was associated with mortality. She should be made aware of the epidemiological literature that questions the absence of a threshold, an absence that EPA presents as uncontested. She should have a clear understanding that the concentration-response functions may be highly non-linear and therefore that EPA estimates of the benefits accruing from various roll-back scenarios are not necessarily accurate as calculated. Only with an appreciation of these and other matters touched upon in the second draft, can the Administrator be expected to make an unbiased decision based on a balanced review of the science.

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