Prepared Statement of
Anne E. Smith, Ph.D.
before the
Committee on Environment and Public Works
United States Senate
Washington, DC
July 19, 2006

Mr. Chairman and Members of the Committee:

Thank you for your invitation to participate in today's hearing. I am Anne Smith, and I am a Vice President of CRA International. I am a specialist in environmental risk assessment and integrated assessment to support environmental policy decisions, which was a core element of my Ph.D. thesis at Stanford University in economics and decision sciences. I have performed work in the area of risk assessment over the past thirty years, including as an economist in the USEPA's Office of Policy, Planning, and Evaluation, as a consultant to the USEPA Air Office, and in many consulting engagements since then for government and private sector clients globally while employed first at Decision Focus Incorporated and then CRA International. I have also served as a member of several committees of the National Academy of Sciences focusing on risk assessment and risk-based decision making.

I have been deeply involved in assessment of the evidence on risks from ambient fine particulate matter ($PM_{2.5}$) since EPA first turned to the task of identifying an appropriate National Ambient Air Quality Standard (NAAQS) for $PM_{2.5}$ over ten years ago. I testified to this same committee in 1997 on the nature of the scientific evidence underlying the $PM_{2.5}$ NAAQS proposed at that time. I thank you for the opportunity to share my perspective today on the current scientific evidence and associated risk assessment for $PM_{2.5}$ and how it has evolved since 1997. My written and oral testimony today provide a statement of my own research and opinions, and does not represent a position of my company, CRA International.

I would like to start by summarizing what I think are the most important and overarching considerations that should be accounted for when considering whether to alter the current $PM_{2.5}$ NAAQS, which include an *annual average* limit of 15 $\mu g/m^3$ and a *24-hour average* limit where the 98^{th} percentile of observations over all days must be below 65 $\mu g/m^3$. I will then summarize results of analyses I have done to synthesize the recent $PM_{2.5}$ health studies into an assessment of risks. Complete details and documentation of my analyses are in my written comments on the current Proposed Rule for a revised $PM_{2.5}$ NAAQS, which were submitted into the $PM_{2.5}$ docket in April, 2006. I am attaching a copy of my written comments to EPA to further substantiate the points that I make in my testimony today.

The key points that I wish to make about the scientific evidence on risks of $PM_{2.5}$ that are relevant for making a decision on the standard are:

- EPA and the courts recognize that the $PM_{2.5}$ NAAQS must be set at a level that still has some positive level of risk, because the science has yet to advance far enough to identify any threshold exposure level for effects, below which risk would be indistinguishable from zero. This was true in 1997 and it remains true today.

- EPA's own quantitative estimates of mortality risk at attainment of the current NAAQS are lower today than they were when EPA set that standard in 1997 "with an adequate margin of safety," after accounting for the many uncertainties. This is true for both long-term ("chronic") exposures to $PM_{2.5}$ (which are addressed by the annual average limit) and short-term ("acute") exposures (which are addressed by the 24-hour average limit).

- The reduction in the quantitative estimates of risk is apparent even in EPA's own risk analysis, but most of the reasonable alternative results reported *in the same studies that EPA has relied on* imply even lower quantitative risk estimates for $PM_{2.5}$.

- Looking more broadly beyond quantitative risk estimates, the many additional studies of $PM_{2.5}$ mortality risks since 1997 have demonstrated that many of the risk estimates become "statistically insignificant" when re-estimated in reasonable alternative ways. A "statistically insignificant" result directly implies a positive probability that there is no effect at all. Thus, when we look at all of the data in the new studies as a group, we find more statistical evidence now than was available in 1997 that $PM_{2.5}$ may not be the culprit pollutant, and that there may be no causal relationship at all between $PM_{2.5}$ and mortality.

In thinking about whether to tighten either the annual or daily standard, one might ask, *what has changed in our knowledge since 1997 that would undermine the Administrator's 1997 judgment that the current $PM_{2.5}$ NAAQS are neither more nor less stringent "than necessary to protect the public health with an adequate margin of safety?* A thorough review of the new evidence suggests that the margin of safety that the Administrator selected in 1997 is likely to be larger than was thought at the time.

Quantitative Estimates of Risk Remaining at the Current Standard Have Fallen

EPA has acknowledged that the $PM_{2.5}$ NAAQS cannot be set at a level that corresponds to zero risk.[1] However, EPA has also argued that its quantitative risk estimates cannot be used to identify a specific point where it should set a standard:

> "[I]n the Administrator's view, a risk assessment based on studies that do not resolve the issue of a threshold is inherently limited as a basis for standard setting, since it will necessarily predict that ever lower standards result in ever lower risks, which has the effect of masking the increasing uncertainty inherent as lower levels are considered. As a result, while the Administrator views the risk assessment as providing supporting evidence for the conclusion that there is a need to revise the current suite of $PM_{2.5}$ standards, he judges that it does not provide a reliable basis to determine what specific quantitative revisions are appropriate."[2]

[1] See, for example, the *Proposed Rule*, p. 2622 (*i.e.*, 71 FR 2622).
[2] 71 FR 2648.

I concur that a risk assessment that makes no attempt to incorporate the uncertainty on where a threshold may exist will indeed only serve to promote ever lower standards without a sound basis. Since EPA has not incorporated such uncertainty into its risk assessment, that risk assessment is indeed incapable of helping to identify where to set the standard. However, since EPA views the risk assessment as supporting a conclusion on whether there is a need to *revise* the standard, it is appropriate and relevant to compare EPA's current quantitative risk estimates and the associated statistical measures of a $PM_{2.5}$ effect to those estimates that were available in 1997. In the *Proposed Rule,* EPA partially acknowledges that risk estimates are lower today than in 1997 for the two cities that were included in both its 1997 and current risk analyses. With respect to short-term exposure risk estimates, EPA states that "the magnitude of the estimates associated with just meeting the current annual standard... is similar in one of the locations... and the current estimate is lower in the other location."[3] With respect to the long-term exposure risks, EPA states that the risk estimates "are very similar for the two specific locations included in both the prior and current assessments."[4]

EPA does not provide the actual numerical estimates for these two cities. They are:

- For acute risks in Los Angeles, in 1997 EPA estimated that 1.7% of mortality would continue to be attributable to $PM_{2.5}$ once Los Angeles would be in attainment with the current NAAQS. Today EPA's risk estimate has fallen to 0.5% *and this current estimate is statistically insignificant* (which means that there is a fairly large chance that this particular estimate suggests that there is really no $PM_{2.5}$ effect at all).

- For acute risks in Philadelphia, in 1997 EPA estimated that 1.5% of mortality would continue to be attributable to $PM_{2.5}$ at attainment of the current NAAQS. The risk estimate that EPA now uses for Philadelphia is 2.2%. Although this is higher than in 1997, EPA has selected a single estimate out of a very large number of estimates reported in the epidemiological study it is relying on for Philadelphia. In fact, that study actually concluded that $PM_{2.5}$ did not appear to explain the mortality risk as well as ozone, and the residual risk for $PM_{2.5}$ after simultaneously accounting for the role of ozone would have produced a lower estimate – about 0.8% – which is lower than in 1997. This more thoroughly-controlled estimate also is not statistically significant.

- Chronic risk estimates do not vary from city to city, because the statistical method to estimate relative chronic risks produces a single value that applies to all cities. I will therefore only relate the results for Los Angeles here. For chronic risks, in 1997 EPA estimated that 2.0% of mortality would continue to be attributable to $PM_{2.5}$ at attainment of the current NAAQS. Today, EPA's risk estimate for the same attainment status is 1.8% – in other words, the chronic risk estimate also is lower now, even though the quote from the *Proposed Rule* above suggests that the estimate has not changed.

The *Proposed Rule* only referred to a comparison of risks for these two cities. However, it is actually possible to make the same comparison for the other six cities that EPA has included in its

[3] 71 FR 2640.
[4] 71 FR 2640.

current risk analysis. This is because there was only one $PM_{2.5}$ acute mortality study it could have used for each of those cities back in 1997 – the same one that it used for Los Angeles and Philadelphia.[5] For five of the other six cities in the current risk analysis, EPA's acute risk estimates today are lower than they would have been estimated to be in 1997, and all the cities have lower chronic risk estimates. Table 1 summarizes the cities and the results of my comparison of their risk estimates.

When I reviewed the original papers that EPA is relying on, I also found that EPA's risk analysis has selectively used the highest or near-highest risk estimates supported by each paper. This means that risks estimates that more fully reflect the body of evidence are likely lower still than EPA's risk analysis suggests. Additionally, as for Philadelphia, I found that San Jose would have had a much lower risk estimate than in 1997 – literally zero now – if EPA had chosen to use the one reasonable alternative result for $PM_{2.5}$ reported in the San Jose study.[6] Thus, the full body of evidence can support risk estimates that would be lower now than in 1997 *for every one of the eight cities in EPA's current risk analysis*.

Table 1. Comparison of EPA's Risk Estimates for Attainment of the Current Standard Now Versus in 1997.

		Has EPA's risk estimate gone up or down since 1997?	Is statistical significance of estimate robust to alternative model choices?
Acute risk	Philadelphia	Up	Not robust
	Los Angeles	Down	Not robust
	Phoenix (*)	Down	Not robust
	St. Louis (*)	Down	Not robust
	Boston (*)	Down	Not robust
	Detroit (*)	Down	Not robust
	Pittsburgh (*)	Down	Not robust
	San Jose (*)	Up	Not robust
Chronic risk	All Cities	Down	Not robust

(*) Although this city was not in the 1997 Risk Analysis, it is possible to determine what risk would have been estimated for each, using the only data that were available in 1997, following EPA's same decisions for Philadelphia and Los Angeles. Specifically, the 1997 risk estimate is based on the "combined" estimate in Schwartz, Dockery, and Neas (1996) for all cities except for Boston and St. Louis, for which it is the city-specific estimate in that paper.

[5] This was the paper by Schwartz, Dockery, and Neas (1996) on acute risks in six US cities.

[6] That is, EPA's risk estimate for San Jose is based on a 1-pollutant regression that associated mortality with $PM_{2.5}$ on the *same day* as death. The study also reported results of a comparable 1-pollutant regression that was identical in all ways except that it associated mortality with $PM_{2.5}$ from the *day before* death. The latter regression produced a *negative* risk estimate, which I interpret to be evidence of no effect at all (rather than evidence of a beneficial effect of $PM_{2.5}$).

Table 1 also reports that the $PM_{2.5}$ findings are not statistically significant across all of the alternative reasonable risk estimates in each underlying study. This was not the case in 1997. At that time, there was a much more limited set of studies – and estimates within each study – but for some cities, all the estimates available at the time were statistically significant. Today, the opposite it true. Every single study that EPA has relied on for its current risk analysis contains alternative estimates that indicate that $PM_{2.5}$ does not have a statistically significant association with mortality, yet EPA chose not to use this part of the new information.

In conclusion, EPA has stated that the risk assessment's role is to provide "supporting evidence" on whether there is a need to revise the $PM_{2.5}$ standard. In this role, EPA's own risk analysis provides no evidence supporting a decision to tighten the standard now. The risks are lower now than they were when the standard was set in 1997. The higher estimates of risks were determined to be "requisite to protect the public health with an adequate margin of safety" in 1997, and the quantitative risk analysis suggests that that margin of safety has grown, not narrowed, as a result of the many more recent $PM_{2.5}$ health effects studies.

The question then remains whether other aspects of the new evidence provide an overriding reason for tightening the standard. The other part of EPA's reasoning for how to set the standard relies on what EPA calls an "evidence-based approach." Simply put, EPA looks at all of the studies that estimate the statistical relationship of $PM_{2.5}$ with health effects, and seeks to identify a level of $PM_{2.5}$ above which statistically significant effects are found, and below which statistically significant effects are not found.

In applying the evidence-based approach, EPA states that the large quantity of new studies of acute effects justifies the use of acute studies to set the 24-hour standard, and that chronic studies should be used to determine where to set the annual standard:

> "Given the extensive body of new evidence based specifically on $PM_{2.5}$ that is now available, and the resulting broader approach presented in the Staff Paper, the Administrator considers it appropriate to use a different approach from that used in the last review to select appropriate standard levels. More specifically, the Administrator's proposal relies on an evidence-based approach that considers the much expanded body of evidence from short-term exposure $PM_{2.5}$ studies as the principal basis for selecting the level of the 24-hour standard and the stronger and more robust body of evidence from the long-term exposure $PM_{2.5}$ studies as the principal basis for selecting the level of the annual standard."[7]

I will next discuss how the evidence in the long-term exposure studies of $PM_{2.5}$ has weakened since 1997, thus removing any necessity to tighten the annual standard under EPA's evidence-based approach. I will then discuss how the evidence in the short-term exposure studies of $PM_{2.5}$ that are the basis for the 24-hour standard also has weakened.

[7] 71 FR 2648.

The Evidence in Long-Term Exposure Studies Has Weakened Since 1997

In 1997, the two prominent long-term exposure studies (one based on a sample population, or "cohort" in 154 US cities that was tracked by the American Cancer Society, and one based on a sample population in just six US cities that was tracked by Harvard School of Public Health) both had published findings of a statistically significant relationship between long-term exposure to $PM_{2.5}$ and life expectancy. These studies were subjected to an extensive process of reanalysis under the auspices of the Health Effects Institute (HEI) that was released in 2000.[8] This reanalysis is widely reputed to have confirmed the original studies' results; however, a complete reading of the actual report shows that some major statistical concerns underlying those results were unearthed. Although a positive $PM_{2.5}$ effect was still found in those data sets, the ability to interpret those results as clearly causal in nature was weakened.

EPA acknowledges that the concerns identified in the HEI reanalyses of the long-term exposure studies for $PM_{2.5}$ remain unresolved to the present time:

> The Administrator also recognizes a contrasting view as to the interpretation of and weight to be accorded to the results from the ACS-based studies (Pope *et al*., 1995; Krewski *et al*., 2000; Pope *et al*., 2002). In this view, the ACS-based studies are not sufficiently robust to support a policy response that would tighten the annual $PM_{2.5}$ standard based on the evidence. This view emphasizes the sensitivity of the results of these studies to plausible changes in model specification with regard to accounting for the geographical proximity of cities and the correlation of air pollutant concentrations within a region, effect modification by education level, and inclusion of SO_2 in the model. In this view, these sensitivities suggest potential confounding or effect modification that has not been taken into account. For example, concern has been raised about the sensitivity of results in the reanalysis of data from the ACS cohort study (Krewski *et al*., 2000) to inclusion of SO_2 in the models. …[T]he reanalysis found that $PM_{2.5}$, sulfates, and SO_2 were each associated with mortality in single-pollutant models. However, in two-pollutant models with SO_2 and $PM_{2.5}$, the relative risk for $PM_{2.5}$ was substantially smaller and no longer statistically significant, whereas the effect estimates for SO_2 were not sensitive to inclusion of $PM_{2.5}$ or sulfates in two-pollutant models. In this view, the ACS-based risk estimates are more robust for SO_2 than for $PM_{2.5}$ or sulfates. In further extended analyses, Pope *et al*. (2002) reported that effect estimates were not highly sensitive to spatial smoothing approaches intended to address spatial autocorrelation, while findings of effect modification by education level were reaffirmed. Results of multi-pollutant models were not reported by Pope *et al*. (2002). Because the correlation coefficient between $PM_{2.5}$ and SO_2 was 0.50 in the ACS data, in this view it is plausible to believe that the independent effects of the two pollutants could be disentangled with additional study."[9]

The quote above is lengthy, which highlights that the concerns identified in the long-term exposure studies are many. The quote above also indicates that the new set of results using the American

[8] The report on findings of these reanalyses is Krewski *et al.* (2000).
[9] 71 FR 2652.

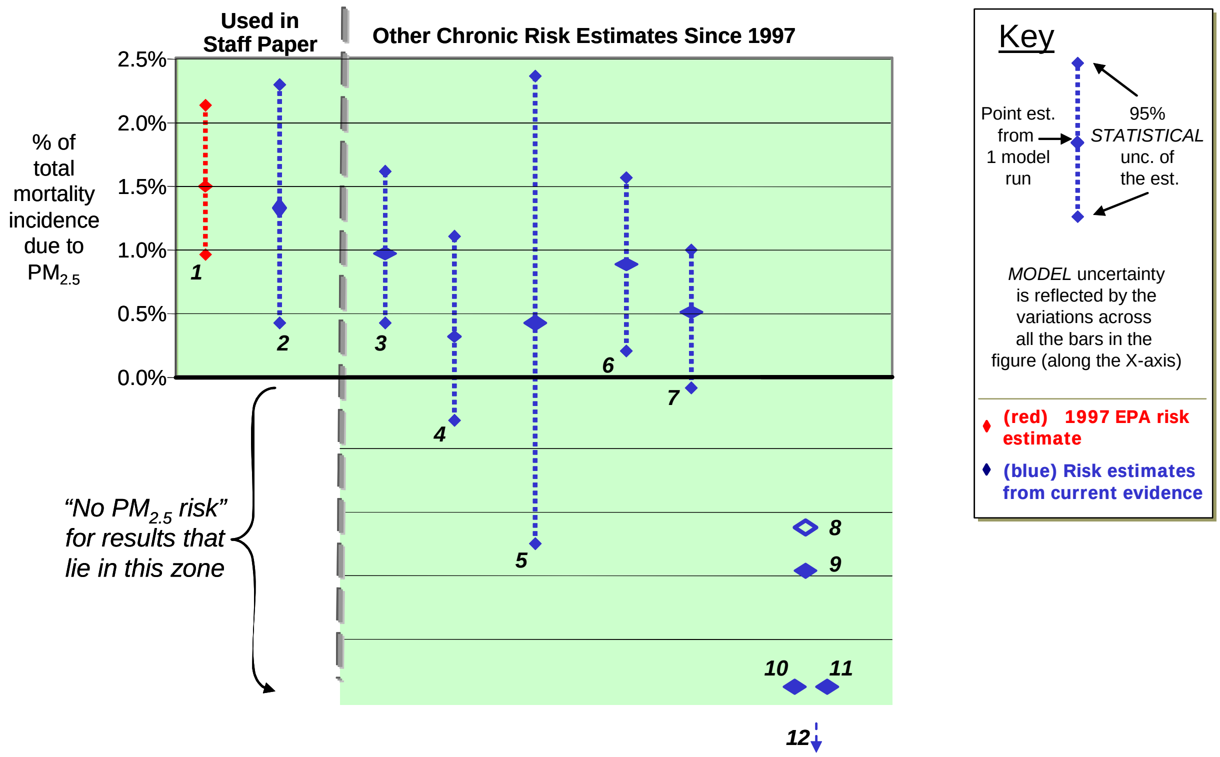
Cancer Society cohort that was published after the HEI reanalyses (*i.e*., Pope *et al.*, 2002) did not help resolve these issues. Specifically, the 2002 paper ignores concerns that the purported $PM_{2.5}$ effect instead might be attributable to the gaseous pollutant SO_2, and re-affirms a troubling finding that $PM_{2.5}$ only seems to create mortality risk only for individuals who have not continued their education beyond the high school level. (The latter finding is discussed further below.) Additionally, the 2002 paper still finds that the $PM_{2.5}$ effect is diminished and rendered insignificant when applying statistical methods to correct a clear statistical error that the HEI report found in the original results. Nevertheless, the Pope *et al.* (2002) paper continues to use the estimation method that is subject to error *except* in a sensitivity analysis; and EPA continues to rely on the uncorrected estimates in its risk analysis. Even with these dubious selections from the full body of literature, EPA's estimates of long-term exposure risk are lower than in 1997.

Figure 1 illustrates the degree to which the evidence on long-term exposure risk has fallen, both in the overall magnitude of the risk estimate, and also in terms of a greater degree of uncertainty in the estimate. Figure 1 uses the case of Los Angeles at attainment of the current standard, yet the relative patterns evident in this figure are the same for all cities in the US. All of the risk estimates in Figure 1 labeled "1" through "7" are based on the American Cancer Society cohort, which has received the majority of attention. The estimate on the far left of the figure, labeled "1" is the estimate from 1997 (note that the estimate is 1.5%, as reported for Los Angeles in the preceding section), and the estimates to the right are other key results from the HEI reanalyses and from the more recent Pope *et al*. (2002) paper. The estimate labeled "2" is the single result from the many new estimates that is used for the current EPA estimates of long-term risk (which is 1.3%, as I stated in the previous section).

It is quite apparent from the figure that the current risk estimate is among the highest that could be found among the more recent results. If any of the others (labeled "3" through "7") had been used for EPA's risk analysis, the current risk estimate of 1.3% for Los Angeles would instead be in the range of 0.3% to 1.0% – much lower than the original 1997 risk estimate that was available when the current standard was first set.

The set of results on the far right of the figure (labeled "8" through "12") reflect the findings based on a new study of a third sample population that had not been identified or studied as of 1997. It is known as the "Veterans' Cohort." I believe this study to be of some policy relevance regarding whether or not the annual standard needs to be tightened, given that this study finds no effect at all of $PM_{2.5}$ on life expectancy in this particular cohort. EPA has chosen to give "greatest weight" to results from the American Cancer Society and the Six Cities cohorts because they have been reanalyzed and scrutinized so thoroughly. While this may be a reasonable judgment, EPA has actually gone further than that, and accorded the Veterans' Cohort results *zero* weight. Its findings should be acknowledged with somewhat more than zero weight. When one does so, the overall evidence regarding long-term $PM_{2.5}$ risks is further weakened.

Figure 1. Evidence of the Eroding Strength of Association in Long-Term $PM_{2.5}$ Risk Studies (estimates are for Los Angeles at attainment of the current standard)

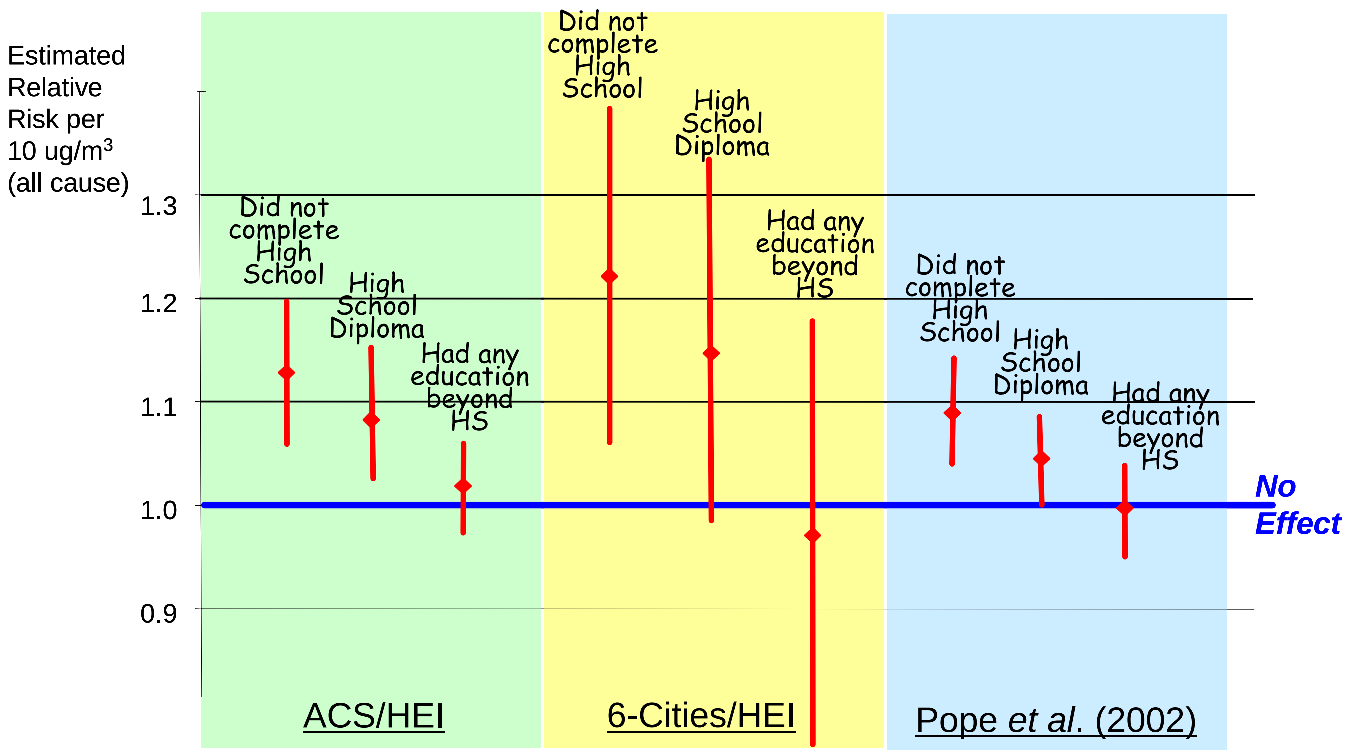


1. Pope *et al.* (1995), Table 3, row 1, column 4 – all combined, all cause mortality, for $PM_{2.5}$
2. Pope *et al.* (2002) Table 2, row 1, column 3 – single pollutant + "all covariates" using "averaged" PM
3. Krewski *et al.* (2000), Table ES-4 – row 3 column 1 (single pollutant)
4. Krewski *et al.* (2000), Table 1, row 18, column 4 – 2-P case (ecologic controls including SO_2)
5. Krewski *et al.* (2000), Table ES-6, row 5 – with "25%" ecologic covariates using regional adjustment
6. Pope *et al.* (2002), Table 2, row 1, column 1 – single pollutant + "all covariates" using 1979-1983 PM
7. Pope *et al.* (2002), Figure 3A – single pollutant + "highest P-value" spatial smoothing using 1979-1983 PM
8. Lipfert *et al.* (2000b), Table 7, deaths in 1976-81 associated with PM2.5 data from 1979-1981 (coeff=-5.28)
9. Lipfert *et al.* (2000b), Table 7, deaths in 1982-1988 associated with PM2.5 data from 1982-1984 (coeff= -6.11)
10. Lipfert *et al.* (2000b), Table 7, deaths in 1982-1988 associated with PM2.5 data from 1979-1981 (coeff= -10.07)
11. Lipfert *et al.* (2000b), Table 7, deaths after 1988 associated with PM2.5 data from 1982-1984 (coeff= -10.78
12. Lipfert *et al.* (2000b), Table 7, deaths after 1988 associated with PM2.5 data from 1979-1981 (coeff=-15.35

EPA also refers to the perplexing finding that level of education determines whether or not there is a $PM_{2.5}$ association. Figure 2 illustrates this finding, which was first identified in the HEI reanalyses, and which remains in the more recent Pope *et al*. (2002) study. Clearly education *per se* is not believed to be the cause of sensitivity to exposure to $PM_{2.5}$, yet the important (and still unanswered) question is: what is educational level indicating about risks that these sample populations face? What could possibly explain the complete lack of a $PM_{2.5}$ effect among those with higher educations? When such a pattern appears in epidemiological study results, it indicates that there is still an important explanatory factor that is missing from the statistical estimation method – something correlated with education. Until that factor is identified and included in the estimation of $PM_{2.5}$ risks, estimates of the effect of $PM_{2.5}$ are biased. The $PM_{2.5}$ estimate could be higher, or it could vanish altogether. Thus, the unexplained pattern related to education in all of these studies remains a very important warning about the pitfalls of making a causal interpretation regarding long-term exposure risks of $PM_{2.5}$.

In summary, the evidence against a need to tighten the annual standard is not just founded on the fact that the numerical long-term risk estimates are now lower than when the current standard was set. The more important point is that the basis for interpreting the long-term studies as unbiased evidence of a causal relationship between $PM_{2.5}$ and chronic mortality risk has weakened. This was a concern in 1997, and the reanalyses and new studies since then have done more to amplify these concerns than to allay them. In the face of this evidence of greater uncertainty, *combined with* the reduced quantitative risk estimates, there is no justification for tightening the annual standard on the basis of the long-term exposure studies.

Figure 2. New Analyses of Long-Term Exposures Risks Since 1997 Find that Educational Level Is an Important Determinant of Whether PM2.5 is Associated with Mortality
(Sources: "ACS/HEI" and "6-Cities/HEI" are based on numerical results reported in Krewski *et al.* (2000), Summary Table 3. Results cited from Pope *et al.* (2002) are in Figure 4A of that paper.)



A Summary CASAC's Case for Tightening the Annual Standard

EPA's Clean Air Scientific Advisory Committee (CASAC) has made the case to tighten the annual standard with two lines of reasoning, neither of which is founded on the long-term risk studies.[10]

CASAC's first line of reasoning is that EPA reports substantial risk would remain at the current standard. As EPA and the courts have long established, the $PM_{2.5}$ NAAQS cannot be a zero-risk standard. CASAC was concerned by the estimates of remaining risk, but never deliberated the question of whether this risk estimate had risen or fallen since the standard was deemed "requisite to protect the public health with an adequate margin of safety." As I have shown above, the risk estimates *fell*, both for chronic and acute risks, but EPA never reported this fact to CASAC during CASAC's review of the *Staff Paper* and associated risk assessment. In the face of this fact, the only other argument to tighten the annual standard might be if there were stronger reason to *believe* that the effects found in these studies are causal in nature. However, EPA set the current standards with a *presumption* (precautionary in nature) that the estimated $PM_{2.5}$ risks *were* causal. This cannot therefore be the rationale to tighten the standards.

Hence, to argue that the standard should be tightened because there is evidence that risk remains at the current standard is a logic that would force a tightening of the standard in every future review cycle, *even if no new evidence were to have become available at all since the previous review.* There is nothing in the law or in precedent that dictates that the standard *has* to be tightened as the result of a NAAQS review.

CASAC's second line of reasoning comes closer to the heart of how EPA first set the standard. CASAC notes that there are three new acute studies that find $PM_{2.5}$ associations with mortality at annual averages below the current annual standard (all with reported annual averages in the range of 13 to 14 $\mu g/m^3$). These studies are: Burnett and Goldberg (2003) for 8 Canadian cities combined; Mar *et al.* (2003) for Phoenix, and Fairley (2003) for Santa Clara County, CA (referred to in the risk analysis as San Jose).[11]

The first thing to realize about this part of CASAC's case for a tightened annual standard is that it is using studies that consider only how day-to-day changes in $PM_{2.5}$ levels affect day-to-day numbers of deaths relative to the number of deaths that might otherwise be expected on each day (*e.g.*, relative to numbers of deaths that are expected to occur on each day based on established patterns related to the time of year, time of week, weather, etc.). Such acute effects of a pollutant are generally believed to be associated with spikes in $PM_{2.5}$, although studies to date rarely report evidence of any threshold level below which the association disappears. Nevertheless, there is no clear linkage between the annual average in a city, and the extent to which day-to-day spikes in $PM_{2.5}$ might be occurring. If an acute effect is found in a city that happens to have a low annual average, there is no reason to believe that the estimated association is not still due to sudden upward changes in $PM_{2.5}$ from one day to the next. The city may simply have a large number of very clean days that pull the annual average $PM_{2.5}$ down, while not eliminating the presence of many days of

[10] CASAC's reasoning is stated in a letter to the Administrator on March, 21 2006, pp. 3-4.
[11] CASAC actually cites a paper by Lipsett *et al.* (1997) in its letter, but that paper has nothing to do with $PM_{2.5}$ mortality. I interpret CASAC to have wanted to cite Fairley (2003).

sudden increases in pollution that are logically likely to be the cause of any acute risk that the study is finding.

Thus, it is not necessarily correct to assume that if acute risks are found in a city with a low annual average pollution, then such risks exist in all cities with low annual average pollution. In fact, if one believes that there must be a threshold where the acute risk from exposure to pollution drops off *somewhere* above zero, then the linkage between the annual average of pollution and existence of acute risk is not only unclear, but illogical. For reasons such as these, EPA has decided to use the plethora of acute risk studies now available to set the 24-hour standard that that type of study more meaningfully informs. EPA has decided not to use acute studies to set an annual standard.[12]

Nevertheless, even if one were to use acute effects studies to determine an "adequate margin of safety" for lower levels of long-term exposure to pollution, there are good reasons to believe that the annual average $PM_{2.5}$ reported for each of the three studies cited by CASAC may not be a good indicator of the long-term exposure levels that account for the risk findings in these studies. I explain why for each of the three:

- **Goldberg and Burnett (2003).** This study reports a $PM_{2.5}$ association for eight Canadian cities *combined*. The annual average of 13.3 $\mu g/m^3$ is an average over all of the eight cities, while the annual averages in the individual cities vary from 9.5 $\mu g/m^3$ to 17.7 $\mu g/m^3$. There are no city-specific results reported to help indicate whether the estimate of an acute effect is due to effects in each of the eight cities, or only in a few.[13] Evidence in the paper suggests that there may in fact be different effects in each city.

  Another concern with this study is that it is a reanalysis of a more comprehensive study that included consideration of the role of gaseous pollutants as well.[14] The original study concluded that the gaseous pollutants had a much greater ability to explain mortality risks than both $PM_{2.5}$ and $PM_{10\text{-}2.5}$ combined. However, when the paper had to be reanalyzed, the authors did not reanalyze the portions that considered gaseous pollutants in conjunction with particulate pollution, and so this finding is no longer discussed.

- **Fairley (2003)**. This study used data from Santa Clara County, CA, over a seven year period, and during that time pollution levels were falling dramatically. Although the annual average $PM_{2.5}$ that is attributed to this study is 13.6 $\mu g/m^3$, the annual average was as high as 18.4 $\mu g/m^3$ at the start, and fell progressively to 9.5 $\mu g/m^3$ by the end of the seven years studied.[15] Peak levels of $PM_{2.5}$ were also falling, starting at a 98^{th} percentile of 88 $\mu g/m^3$ for the first year and ending at 25 $\mu g/m^3$. Such a wide range within this one city's data set begs the question: are the reported acute effects relationship driven largely by the high levels in the early years, or are they also evident in the later years? This highly relevant question is

[12] See the quote on p. 5 above, taken from 71 FR 2648.
[13] The other multi-city $PM_{2.5}$ mortality studies (based on the Six Cities data set) report effects by individual city as well as for the combined set. This was the data on which EPA set the current standards, and in doing so, EPA used annual averages for only the individual cities that did have significant effects within the set of six. The lowest such city was Boston, with an annual average of 15.6 $\mu g/m^3$, which was the basis for the current annual average standard of 15 $\mu g/m^3$.
[14] The original paper was Burnett *et al.* (2000)
[15] To know this, one must go back and read the original study that this is a reanalysis of, Fairley (1999).

never mentioned, let alone analyzed, by the authors. Lacking any exploration of such an obviously relevant issue, it would seem a dubious proposition to use the annual average over the entire time period in this one study as the basis for a national ambient standard.

Another concern with this study is that it reports $PM_{2.5}$ risk estimates for two alternative methods of estimation, both of which are reasonable. One method considers whether deaths tend to fluctuate with the same day's $PM_{2.5}$ levels and the other method considers whether deaths tend to fluctuate with the previous day's $PM_{2.5}$ levels. The same-day estimate finds the positive association that this study is known for, but the estimate based on $PM_{2.5}$ on just the previous day is actually in the negative direction. *Complete* reversal of evidence of a $PM_{2.5}$ mortality effect by considering $PM_{2.5}$ levels only 24 hours apart in time presents a concern for interpreting the study's same-day estimate as a causal one. However, there is no discussion of what these conflicting results might mean.

- **Mar *et al.* (2003).** This study considered acute risks in Phoenix, AZ, with annual average $PM_{2.5}$ levels of 13.5 $\mu g/m^3$. There are ten estimates of $PM_{2.5}$ risk in the paper, and only three of them are significant. More importantly, this is not the only paper that studied the ability of this same set of $PM_{2.5}$ data to explain acute mortality risks in Phoenix. One of the other studies found that $PM_{2.5}$ did not have any explanatory power, and found instead that the coarse fraction of PM had explanatory power.[16] The third study found evidence that there is a threshold below which $PM_{2.5}$'s apparent ability to explain changes in daily mortality disappeared.[17] That threshold appeared to be above 20 $\mu g/m^3$. If there is a threshold, then the rationale for a linkage between annual average $PM_{2.5}$ and acute risks simply falls apart.

  A final concern with all three of the Phoenix studies is that none of them considered whether the $PM_{2.5}$ effect would remain if pollutants such as CO, SO_2, ozone, or NO_2 were also included in the analysis. This is a critical gap in many of the current studies because the new body of papers on $PM_{2.5}$ health effects reveals that $PM_{2.5}$ effects usually disappear when one of the gaseous pollutants is explored. This is addressed in the next part of my testimony.

In summary, CASAC makes its case to tighten the annual standard on the basis of acute, not chronic effects studies. There are logical problems with this approach to setting an annual standard; these logical problems become apparent when looking at each of the three acute studies that CASAC cites as its basis for recommending an annual standard that is tighter than the current one.

The Statistical Evidence on Acute Effects of $PM_{2.5}$ Has Also Weakened Since 1997.

In 1997, there existed only one study that had used actual measurements of $PM_{2.5}$ and estimated whether daily numbers of deaths might be associated with day-to-day variations in the $PM_{2.5}$. This was a study using the data from the Harvard study of six US cities reported in Schwartz *et al.* (1996), and it was used as the basis for the current standards. In that study, statistically significant associations of $PM_{2.5}$ and acute mortality were found in three of the four cities with the highest 98th percentile $PM_{2.5}$ levels, which ranged from 42 $\mu g/m^3$ to 44 $\mu g/m^3$. The city with the highest $PM_{2.5}$

[16] Clyde *et al.* (2000).
[17] Smith *et al.*, (2000)

98th percentile (which was 82 μg/m^3) did not produce a statistically significant association, nor did the two cities with the lowest 98th percentile levels of 32 and 34 μg/m^3. This was the best available information at the time,. Other than the anomaly for the city with the highest $PM_{2.5}$ exposures, it did at least suggest that there might be a range above which effects were more likely and below which they were more unlikely.

While this study was used as the primary basis for the current standards (including the annual standard), there were many concerns expressed with uncertainties in the estimation methods. In particular, there was concern that this study had not considered the explanatory role of any of the other common pollutants like CO, SO_2, ozone, and NO_2. (These are often called the "gaseous pollutants" because that distinguishes them from various forms of particulate pollutants that are regulated under the PM NAAQS.) It was argued that $PM_{2.5}$ might be simply playing a proxy role for a gaseous pollutant also present in the air in these cities.

As new acute $PM_{2.5}$ studies were performed after 1997, a number of these studies did strive to explore the respective roles of $PM_{2.5}$ and gaseous pollutants in the observed statistical associations. This was done by using "2-pollutant" or "multi-pollutant" methods, as contrasted to the "1-pollutant" method that only allows a single pollutant (*e.g.*, $PM_{2.5}$ in this case) to have any opportunity to explain mortality risk. One of the little recognized but important insights of this body of studies is that when gaseous pollutants also have been considered in a study, the gaseous pollutant has taken over the explanatory role from $PM_{2.5}$ in a majority of the cases.

I determined this in my review of the studies since 1997. Specifically, I attempted to identify all of the $PM_{2.5}$ health effects studies cited in the *Criteria Document* (including both mortality and morbidity effects studies) that had reported results of any estimates for $PM_{2.5}$ using a 2-pollutant method of estimation for at least one gaseous pollutant. I found ten such papers among all the new studies that did report a statistically significant association for $PM_{2.5}$. Of these ten, eight saw $PM_{2.5}$ lose its ability to explain mortality risk when studied using a 2-pollutant method. (In the other two studies, both the $PM_{2.5}$ and gaseous pollutant retained statistical explanatory power.)

Often it is suggested that 2-pollutant methods are not useful because it is impossible to unravel the effects of two pollutants that both move up and down together in near synchrony (*i.e.*, they are highly "correlated"). However, my review of these papers did not find evidence that this was a problem. If it were a problem, then *both* the $PM_{2.5}$ and gaseous pollutant would lose their explanatory power. What I found instead was that in seven of the eight studies where $PM_{2.5}$ lost its erstwhile explanatory power when it was the *only* pollutant considered, the gaseous pollutant *retained* its explanatory power. Otherwise stated, of the ten studies that I started with, only one seemed to be affected by intractable statistical problems making it impossible to unravel the separate effects of the two pollutants.

These papers are summarized in Table 2, which is more fully explained in my written comments to EPA of April 2006, which I am submitting with this testimony.

Table 2. Evidence from Recent Studies that Acute $PM_{2.5}$ Effect Estimate Is Often Lost When Estimated in a 2-Pollutant Model with a Gaseous Co-Pollutant

Paper	City	Effect Estimated	Was any $PM_{2.5}$ Coefficient Significant?		Gaseous Pollutant Signif in 2-P?	Gaseous Pollutant Included
			1-P	2-P		
Delfino *et al*., 1997	Montreal	ER visits	Yes	No	Yes	O_3
Sheppard, 2003	Seattle	Hosp adm	Yes	No (**)	Yes	CO
Lipfert *et al*., 2000a	Philadelphia	Mortality	Yes	No	Yes	O_3
Korrick *et al*., 1998	NH Mtns	Lung function indicators	Yes	No	No	O_3
Thurston *et al*., 1994	Toronto	Hosp adm	Yes	No	Yes	O_3
Moolgavkar, 2003	Los Angeles	Hosp adm	Yes	No	Yes	CO, NO_2
		Mortality	Yes	No	Yes	CO
Delfino *et al*., 1998	Montreal	ER visits	Yes	No	Yes	O_3
Peters *et al*., 2000	E. Mass	Arrhythmia symptoms	Yes	No	Yes	NO_2
Fairley, 2003	Santa Clara Co, CA (San Jose)	Mortality	Yes	Yes	Yes for peak O_3	NO_2, O_3, CO
Gold *et al*., 2003	Boston	HRV	Yes	Yes	Yes for O_3	O_3, NO_2, SO_2
Chock *et al*., 2000	Pittsburgh	Mortality	No	No	No	Several

(**) For Sheppard (2003), the reanalyzed GAM-based 1-P and 2-P results were both significant. However, the GAM code produces a biased standard error that overstates significance levels, and hence GLM-based results are viewed as more reliable and should be used when available. The GLM-based 1-P result is significant, while the GLM-based 2-P result in this paper is insignificant (albeit borderline), and the relative risk level is reduced. Additionally, all four of the seasonal coefficients for the 2-P GLM models are insignificant.

The above findings represent just one of many ways that the new body of acute effects evidence has been found to vary depending on the particular method of estimation. Other sources of variation in the evidence include the methods for accounting for time and weather considerations. The new studies have demonstrated that concerns with variability of epidemiological estimates of risk, which were expressed but not well explored in 1997, are real. Table 1 at the beginning of my testimony shows that even the "best" $PM_{2.5}$ health effects studies that EPA could select for its risk analysis present a highly uncertain picture of whether $PM_{2.5}$ is playing a causal role for acute effects. Even if there is a causal relationship, which is what the current standard assumed when EPA set it in 1997, there appears to be great difficulty in determining what the size of the effect is. The one trend that is clear is that the size of the $PM_{2.5}$ acute mortality estimates found in the many new studies since 1997 are generally lower than the estimates that were available when the current standards were set.

As I have already noted, EPA has chosen to use an evidence-based approach to set the 24-hour standard. EPA has proposed to tighten the 24-hour standard from the current level of 65 $\mu g/m^3$ to 35 $\mu g/m^3$. This decision was made even though the quantitative risk estimates based on these studies are lower and statistically weaker than they were when the standard was set. Nevertheless, it is true that there are many more studies available now than at the time of the standard, and it is relevant to ask if this new body of evidence might provide a better indication of a 98^{th} percentile $PM_{2.5}$ level where observed effects start to drop off. EPA has attempted to make such a case for a cut-off point of 35 μg/m^3 in the *Proposed Rule*.[18] I have gone through that case very carefully, and I have found it incomplete. I will state what I found in general terms here.[19]

EPA's verbal summary of its evidence-based approach used a selected subset of ten $PM_{2.5}$ mortality studies. I found another eight such acute studies of US or Canadian mortality cited in the *Criteria Document*, that used actual measurements of $PM_{2.5}$, and that did not appear to have any un-reanalyzed statistical problems associated with the GAM software. (If a single paper reports results for more than one city, I treat each city as a separate "study".) Six of the eighteen studies that I considered are the original "Six Cities" used to set the current standard. All of the others are studies published between 1997 and the cut-off time for consideration in this review cycle.

I read each study, and determined whether all the $PM_{2.5}$ estimates reported in a study were "more often insignificant than significant", "a near 50-50 mix", or "more often significant than insignificant." After categorizing them in this way, I found that there is no clear pattern where statistically significant results tend to be found for studies with higher $PM_{2.5}$ levels, and that increasingly mixed evidence is found in studies with progressively lower $PM_{2.5}$ levels. Figure 3 graphically summarizes my findings for the mortality studies. It shows that many of the datasets with the highest 24-hour average $PM_{2.5}$ levels demonstrate the least likelihood of a statistically-significant association with mortality. This is contrary to what EPA states in its discussion of the evidence-based approach in the *Proposed Rule*. I attribute the difference to the fact EPA considered only a selected set of the new studies, and not the more complete set that I identified. (The ten studies EPA considered are shown as blue diamonds in Figure 3, while the additional eight studies that I also considered are shown as red diamonds in Figure 3.)

Figure 3 also shows that I determined that only three of the eighteen studies found statistically significant $PM_{2.5}$ effects for a majority of the methods of estimation that they reported. Of these:

- One is for eight Canadian cities *combined* by Goldberg and Burnett (2003), which I described earlier in this testimony. Its 98th percentile value is about 39 μg/m^3, but this value has the same flaw that I described for its annual average – the actual peak exposures faced by people in the eight separate cities ranged from 27 to 48 μg/m^3, and there is no information to indicate which of the various city-specific 98th percentiles might be accounting for the effects estimated when all cities are combined.

- Another of these was Fairley (2003) for Santa Clara Co, CA, with a 98th percentile value of 59 μg/m^3, which I also discussed above. This study had very high exposures at first, and we have no idea whether the statistical significance is related to the earlier high levels, or equally attributable to later, lower $PM_{2.5}$ peaks.[20]

---

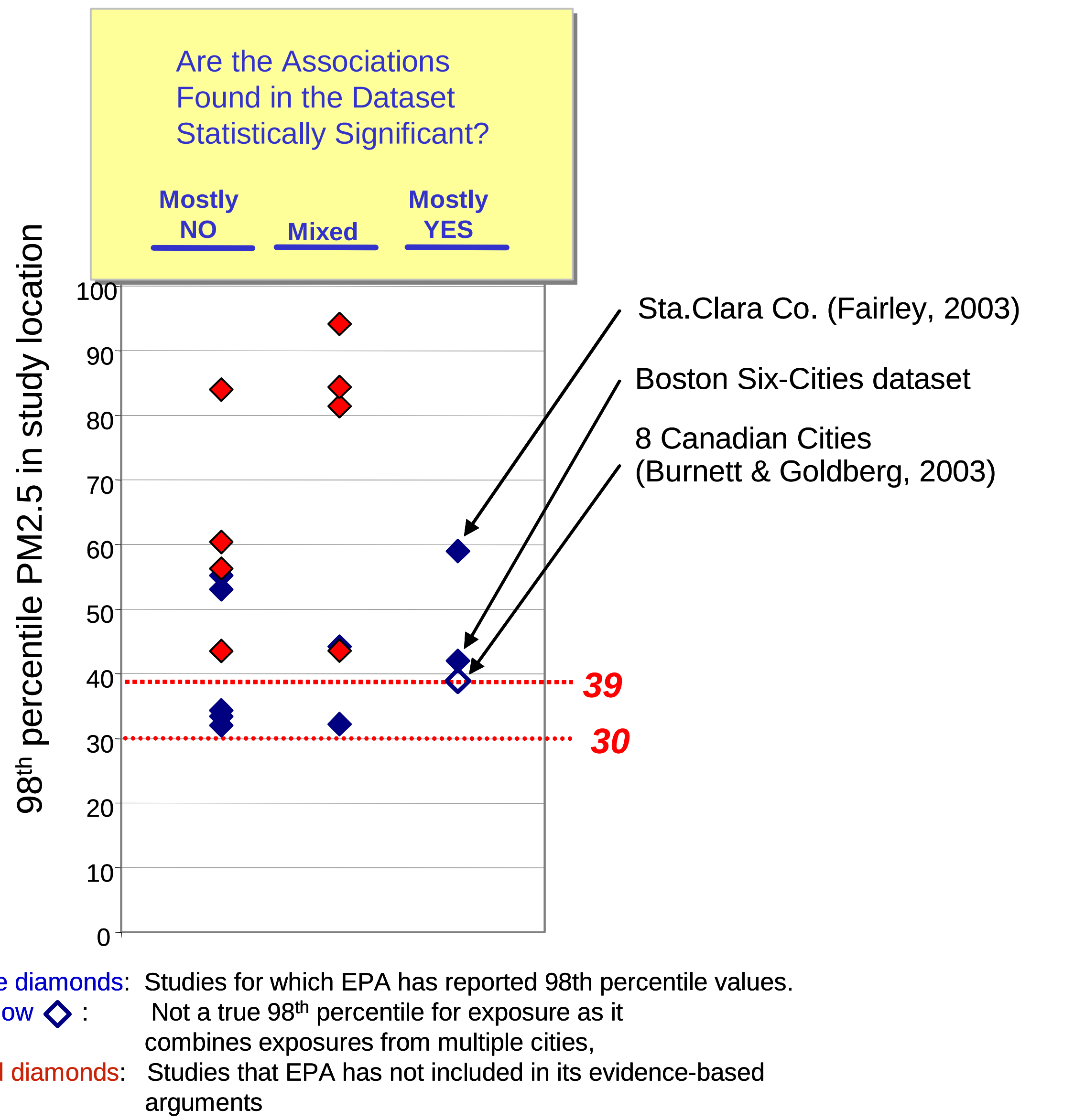
[18] See 71 FR 2649.

[19] My written comments to EPA that are being submitted into the record with this testimony provide complete documentation of my review of the literature and application of an evidence-based approach for the 24-hour standard. Although the full discussion includes both mortality and morbidity studies, I only summarize the mortality findings here. However, the patterns I describe are similar in studies of hospitalizations, emergency room visits, and frequency of "symptoms" that are not severe enough to entail a hospital visit.

[20] Further, although a majority of estimates are statistically significant because the paper focused on the lag period that was found to be significant in the simplest, 1-pollutant starting point of the analysis, the single other alternative 1-pollutant result found a negative risk estimate, implying no risk at all.

- The third is the Boston data from the Harvard Six Cities study that served as the basis for the current $PM_{2.5}$ NAAQS in 1997. The magnitude and statistical significance of the association observed in this dataset has been reduced in reanalyses since 1997, and none of these estimates include consideration of the potential role of any gaseous pollutants in explaining these associations.

Figure 3. Summary of the Evidence of Statistically Significant Associations between $PM_{2.5}$ and Acute Mortality in 18 Locations.
(Source: Written comments of Anne E. Smith on EPA's Proposed Rule, April 2006. Copy of these comments is submitted with this testimony)



In conclusion, the evidence regarding a causal relationship between short-term exposures to $PM_{2.5}$ and health has not strengthened since 1997. To draw this conclusion, one must consider more than just the *number* of new studies that have reported at least one statistically significant association; one must also explore the extent to which the effects reported in these studies remain statistically significant under a range of different plausible methods for making such estimates. In particular, the new evidence strongly suggests that many or most of these associations may actually be attributable to a gaseous pollutant, not $PM_{2.5}$.

But even setting aside the weaknesses in the statistical evidence, EPA's evidence-based approach for where to set a 24-hour standard for $PM_{2.5}$ leads us right back to the very dataset on which the current $PM_{2.5}$ NAAQS were based – the Boston "Six Cities" dataset. Thus, the evidence-based approach that EPA is trying to apply provides little additional insight beyond the simple point that I started my testimony with: the quantitative estimates of risks remaining at the current standards are *lower now* than when they were determined to offer an "adequate margin of safety." They therefore do not support a tightening of the current NAAQS.

Integrated Analyses of Alternative Results May Help Inform NAAQS Decisions Better

It is easy to feel lost regarding how to effectively interpret a plethora of alternative studies, and of alternative risk estimates within each study. EPA's method in performing risk analyses has been to rely on a single estimate that it selects from the large pool of alternatives, and to base its summaries of quantitative risk estimates on that single estimate. In many of these summaries, even the statistical errors associated with that one estimate are often not reported. Some, but not all, of the remaining alternative estimates are studied through "sensitivity analyses." However, these are usually relegated to the back pages of a technical support document. The result of this approach is that the degree of certainty about the risk estimates becomes greatly overstated by the time summary results reach the eyes of decision makers, advisors, and the public. Further, the method of selecting the single risk estimate to rely on for the primary analysis can lead to a substantial bias in the quantitative risk estimates reported.

There are alternative methods for performing risk assessments that integrate multiple alternative risk estimates, and even key uncertainties that remain purely judgmental. These methods are sometimes called probabilistic analysis, or integrated uncertainty analysis. EPA has not used such methods in the documents supporting the Administrator's decision on the $PM_{2.5}$ NAAQS, such as the *Staff Paper*. I believe that such methods could be very useful, and would reveal better the true extent of uncertainty that I have tried to characterize qualitatively in my testimony above.

In 2003, at an early stage of the drafting of the current risk assessment, I prepared some illustrative examples of an integrated uncertainty analysis to show how the reams and reams of sensitivity results in the risk assessment document could be condensed to more decision-relevant information. The results of that illustrative analysis remain of some interest:

- Using just the alternative long-term exposure studies in the *Criteria Document*, I found that there could be about a 40% probability that there would be no long-term mortality benefit from tightening the current NAAQS. I also estimated that the probability that actual long-term mortality would be less than the primary risk estimate that EPA reports in its risk analysis is about 75%.

- I did a less thorough example for the short-term mortality risk, based only on Los Angeles. (Short-term risk estimates – and their uncertainty – vary by city). For Los Angeles, I estimated a 42% probability there would be no benefits from tightening the standard from the current level when using only the risk estimates that EPA had itself cited in its risk analysis, and a 64% probability that acute risk reductions would be lower than EPA's primary risk estimate.[21]

These probability estimates were based solely on actual estimates in the new body of literature on $PM_{2.5}$ mortality, and do not include any external judgments such as whether any of these estimates can be interpreted as causal, whether some particles are more toxic than others, or the hypothetical presence of a threshold. (Consideration of these issues would raise the probabilities that I calculated.) They are strictly based in the published evidence reviewed in the *Criteria Document.* They are thus indicative of the degree of uncertainty that the published studies themselves reveal.

I believe that the process of decision making leading up to the point where a new rule is proposed would benefit greatly if such a synthesis of statistical and modeling uncertainties were to be developed as a part of that process. Controversies would remain regarding the judgments that are necessary for such estimates, but if they are conducted in an open manner, with ample opportunity for public review and comment, more insight about the overall implications of the body of scientific evidence would be created before a decision must be made than we have at the present moment. I emphasize that this should be done *during* the NAAQS review cycle, with opportunities for public review and comment, before a rule is proposed.

[21] For documentation of these calculations, see Smith (2003).

References to Papers Cited in This Testimony

Burnett, R. T.; Brook, J.; Dann, T.; Delocla, C.; Philips, O.; Cakmak, S.; Vincent, R.; Goldberg, M. S.; Krewski, D. (2000) "Association Between Particulate- and Gas-phase Components of Urban Air Pollution and Daily Mortality in Eight Canadian Cities" In: Grant, L. D., ed. PM2000: Particulate Matter and Health. *Inhalation Toxicology* 12(suppl. 4): 15-39.

Burnett, R. T.; Goldberg, M. S. (2003) "Size-fractionated Particulate Mass and Daily Mortality in Eight Canadian Cities." *Revised Analyses of Time-series Studies of Air Pollution and Health. Special report*. Boston, MA: Health Effects Institute; pp. 85-90.

Chock, D. P.; Winkler, S.; Chen, C. (2000) "A Study of the Association Between Daily Mortality and Ambient Air Pollutant Concentrations in Pittsburgh, Pennsylvania" *Journal of the Air & Waste Management Association* 50: 1481-1500.

Clyde, M. A.; Guttorp, P.; Sullivan, E. (2000) "Effects of Ambient Fine and Coarse Particles on Mortality in Phoenix, Arizona" Seattle, WA: University of Washington, National Research Center for Statistics and the Environment; NRCSE technical report series, NRCSE-TRS no. 040.

Delfino, R. J.; Murphy-Moulton, A. M.; Becklake, M. R. (1998) "Emergency Room Visits for Respiratory Illnesses Among the Elderly in Montreal: Association with Low Level Ozone Exposure" *Environmental Research* 76: 67-77.

Dockery, D. W.; Pope, C. A., III; Xu, X.; Spengler, J. D.; Ware, J. H.; Fay, M. E.; Ferris, B. G., Jr.; Speizer, F. E. (1993) "An Association Between Air Pollution and Mortality in Six U.S. Cities" *New England Journal of Medicine* 329: 1753-1759.

Fairley, D. (1999) "Daily Mortality and Air Pollution in Santa Clara County, California: 1989-1996" *Environmental Health Perspectives* 107: 637-641.

Fairley, D. (2003) "Mortality and Air Pollution for Santa Clara County, California, 1989-1996" *Revised Analyses of Time-series Studies of Air Pollution and Health, Special Report.* Boston, MA: Health Effects Institute; pp. 97-106.

Gold, D. R.; Schwartz, J.; Litonjua, A.; Verrier, R.; Zanobetti, A. (2003) "Ambient pollution and reduced heart rate variability. In: *Revised Analyses of Time-series Studies of Air Pollution and Health,* Special report. Boston, MA: Health Effects Institute; pp.107-112.

Korrick, S. A.; Neas, L. M.; Dockery, D. W.; Gold, D. R.; Allen, G. A.; Hill, L. B.; Kimball, K. D.; Rosner, B. A.; Speizer, F. E. (1998) "Effects of Ozone and Other Pollutants on the Pulmonary Function of Adult Hikers" *Environmental Health Perspectives* 106: 93-99.

Krewski, D.; Burnett, R. T.; Goldberg, M. S.; Hoover, K.; Siemiatycki, J.; Jerrett, M.; Abrahamowicz, M.; White, W. H. (2000) *Reanalysis of the Harvard Six Cities Study and the American Cancer Society Study of Particulate Air Pollution and Mortality,* a special report of the Particle Epidemiology Reanalysis Project. Cambridge, MA: Health Effects Institute.

Lipfert, F. W.; Morris, S. C.; Wyzga, R. E. (2000a) “Daily Mortality in the Philadelphia Metropolitan Area and Size-classified Particulate Matter” *Journal of the Air & Waste Management Association* 1501-1513.

Lipfert, F. W.; Perry, H. M., Jr.; Miller, J. P.; Baty, J. D.; Wyzga, R. E.; Carmody, S. E. (2000b) “The Washington University-EPRI Veterans' Cohort Mortality Study: Preliminary Results” *Inhalation Toxicology* 12(suppl. 4): 41-73.

Mar, T. F.; Norris, G. A.; Larson, T. V.; Wilson, W. E.; Koenig, J. Q. (2003) “Air Pollution and Cardiovascular Mortality in Phoenix, 1995-1997” *Revised Analyses of Time-series Studies of Air Pollution and Health, Special Report* Boston, MA: Health Effects Institute; pp. 177-182.

Moolgavkar, S. H. (2003) “Air Pollution and Daily Deaths and Hospital Admissions in Los Angeles and Cook Counties” *Revised Analyses of Time-series Studies of Air Pollution and Health, Special Report* Boston, MA: Health Effects Institute; pp. 183-198.

Peters, A.; Liu, E.; Verrier, R. L.; Schwartz, J.; Gold, D. R.; Mittleman, M.; Baliff, J.; Oh, J. A.; Allen, G.; Monahan, K.; Dockery, D. W. (2000) “Air Pollution and Incidence of Cardiac Arrhythmia” *Epidemiology* 11: 11-17.

Pope, C. A., III; Thun, M. J.; Namboodiri, M. M.; Dockery, D. W.; Evans, J. S.; Speizer, F. E.; Heath, C. W., Jr. (1995) “Particulate Air Pollution as a Predictor of Mortality in a Prospective Study of U.S. Adults” *American Journal of Respiratory and Critical Care Medicine* 151: 669-674.

Pope, C. A., III; Burnett, R. T.; Thun, M. J.; Calle, E. E.; Krewski, D.; Ito, K.; Thurston, G. D. (2002) “Lung Cancer, Cardiopulmonary Mortality, and Long-term Exposure to Fine Particulate Air Pollution” *Journal of the American Medical Association* 287: 1132-1141.

Schwartz, J.; Dockery, D. W.; Neas, L. M. (1996) “Is Daily Mortality Associated Specifically with Fine Particles?” *Journal of the Air & Waste Management Association* 46: 927-939.

Sheppard, L. (2003) “Ambient Air Pollution and Nonelderly Asthma Hospital Admissions in Seattle, Washington, 1987-1994” *Revised Analyses of Time-series Studies of Air Pollution and Health, Special Report* Boston, MA: Health Effects Institute; pp. 227-230.

Smith, A. (2003) “Comments on EPA’s ‘Particulate Matter Health Risk Assessment for Selected Urban Areas: Draft Report’” (submitted to EPA as an attachment to comments from Utility Air Regulatory Group), EPA Docket No. EPA-HQ-OAR-2001-0017-0200 (October 28, 2003).

Smith, R. L.; Spitzner, D.; Kim, Y.; Fuentes, M. (2000) “Threshold Dependence of Mortality Effects for Fine and Coarse Particles in Phoenix, Arizona” *Journal of the Air & Waste Management Association* 50: 1367-1379.

Thurston, G. D.; Ito, K.; Hayes, C. G.; Bates, D. V.; Lippmann, M. (1994) “Respiratory Hospital Admissions and Summertime Haze Air Pollution in Toronto, Ontario: Consideration of the Role of Acid Aerosols” *Environmental Research* 65: 271-290.