



UNITED STATES ENVIRONMENTAL PROTECTION AGENCY  
WASHINGTON D.C. 20460

OFFICE OF THE ADMINISTRATOR  
SCIENCE ADVISORY BOARD

[Date]

EPA-COUNCIL-10-xxx

The Honorable Lisa P. Jackson  
Administrator  
U.S. Environmental Protection Agency  
1200 Pennsylvania Avenue, N.W.  
Washington, D.C. 20460

Subject: Review of EPA's DRAFT Health Benefits of the Second Section 812  
Prospective Study of the Clean Air Act.

Dear Administrator Jackson:

In response to a request from EPA's Office of Air and Radiation (OAR), the Advisory Council on Clean Air Compliance Analysis (Council) convened the Health Effects Subcommittee (HES) with additional experts from the Council to review EPA's draft benefits and uncertainty documents supporting the second prospective study of the benefits and costs of the Clean Air Act. The study was conducted in concordance with Section 812 of the Clean Air Act Amendments of 1990. Specifically, the HES provided advice on the data chosen for the analysis, the selection of models used to conduct the analysis, and the validity of resulting estimates of Clean Air Act program benefits and costs.

The HES compliments EPA on a thorough analysis of a very complex issue. The Second Prospective Study has been under development for several years. In the early planning phases, the analytical blueprint and preliminary data were reviewed by the Council and its subcomponents. EPA has thoughtfully and thoroughly considered previous advice in conducting this analysis.

The HES finds the data sources and analytical methodology to be generally sound and well conceived. In particular, the HES notes that reductions in particulate matter (PM)-related mortality that can be attributed to the Clean Air Act are a major driver of benefits. The benefit estimates that EPA derives for PM are based on two well-researched and highly respected cohort studies of air pollution health effects, the Harvard Six Cities Study and the American Cancer Study (ACS) Cancer Prevention Study. These are good foundations for the health benefits estimates for PM and these studies are supported by other recent studies and expert evidence gathered by the EPA project team. This is encouraging, and the HES makes some recommendations on how to express a best estimate

1 and the uncertainty around that estimate. The HES generally agrees with other decisions made by the  
2 EPA project team with respect to PM, in particular, the PM mortality effect threshold model, the  
3 cessation lag model, the inclusion of infant mortality estimation, and differential toxicity of PM.  
4

5 The HES supports the inclusion of mortality related to ozone exposure in the Section 812  
6 Prospective Analysis and supports EPA's data choices for estimating health benefits from  
7 reduction in ozone exposure. As with PM, the HES makes recommendations on a statistical  
8 approach to better capture the best estimate and uncertainty around the ozone concentration-  
9 response function.

10  
11 The HES finds that the draft documents presented to it for review require a fair amount of technical  
12 editing to make the analyses clearer. The details of the conclusions and recommendations are detailed  
13 in the Committee's report. We appreciate the opportunity to provide EPA advice on their analysis of  
14 the health benefits of the Clean Air Act regulations.  
15

16  
17 Sincerely,  
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21  
22 Dr. James Hammitt  
23 Chair  
24 Advisory Council on Clean Air Compliance Analysis

Dr. John Bailar  
Chair  
Health Effects Subcommittee

NOTICE

1  
2  
3 This report has been written as part of the activities of the EPA Advisory Council on Clean Air  
4 Compliance Analysis (Council), a public advisory group providing extramural scientific  
5 information and advice to the Administrator and other officials of the Environmental Protection  
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1  
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1 **EXECUTIVE SUMMARY**

2  
3 The Clean Air Act amendments of 1990 included specific language in Section 812 directing the  
4 EPA to perform benefit-cost studies of the overall effects of the Clean Air Act.

5  
6 In response to Section 812, on October 15, 1997, EPA issued a retrospective study (1970 – 1990)  
7 of benefits and costs of the Clean Air Act. EPA issued a first prospective study (1990 – 2010) on  
8 November 15, 1999 and is now completing a second prospective study (1990 – 2020).

9  
10 The Health Effects Subcommittee (HES) of the Advisory Council on Clean Air Compliance Analysis  
11 (Council) held a public meeting on December 15-16, 2009 to review and provide guidance to the  
12 EPA on the draft human health effect estimates in chapters from the second Section 812 Prospective  
13 Analysis benefits report (1990 – 2020) as well as the human health components of the draft  
14 standalone uncertainty analysis report. Specifically, the OAR asked the HES to consider: 1) the data  
15 choices and methodologies used to develop mortality estimates as a function of air pollution  
16 concentration for both particulate matter (PM) and ozone; 2) The methods and data used to develop  
17 estimates of the lag time between cessation of exposure and reduction in health effects for both PM  
18 and ozone; 3) the Agency’s assumptions about a threshold concentration for mortality effects for  
19 both PM and ozone; 4) the estimates of infant mortality related to PM exposure; 5) Baseline  
20 incidence and prevalence of relevant disease conditions in the population; 6) the sensitivity analysis  
21 of the analytical results to differences in PM composition; and 7) the approach for dynamic modeling  
22 of U.S. population demographic changes.

23  
24 Overall, the HES finds the EPA analyses, data choices, and methodologies to be sound. EPA bases  
25 its estimates of the mortality benefits of reducing fine particulate matter (PM<sub>2.5</sub>) on data analyzed  
26 from two large, long-running landmark studies on the health effects of air pollution, the Harvard Six  
27 Cities Study and the American Cancer Society (ACS) Cancer Prevention Study (CPS). The HES  
28 finds the selection of these cohort studies as the underlying basis for PM mortality benefit estimates  
29 to be a good choice. These are widely cited, well studied and extensively reviewed data sets. EPA  
30 further bolsters its PM mortality benefit estimates by gathering information on the PM mortality  
31 concentration-response function in an expert elicitation of twelve clinicians, epidemiologists, and air  
32 pollution scientists. HES suggests an alternative to EPA’s choice of the Pope et al. (2002) study  
33 (based on the ACS cohort) for the primary estimates of differences in incidence of PM-related  
34 premature mortality and the Laden et al. (2006) study (based on the Six Cities cohort) as an  
35 alternative estimate. EPA found that these choices fell at approximately the 25<sup>th</sup> and 75<sup>th</sup> percentile of  
36 the mortality effect estimates garnered from the expert elicitation. EPA proposed a statistical  
37 approach using copula functions to combine the 12 expert elicitation estimates into a single mortality  
38 effects distribution. The HES, however, recommends a simpler approach. EPA should define a  
39 distribution, perhaps a truncated normal distribution, with the Pope and Laden studies at the 25<sup>th</sup> and  
40 75<sup>th</sup> percentiles respectively. The mean of the new distribution should then be close to the mean of  
41 the central estimates of both Pope and Laden. This will be generally consistent with the distribution  
42 identified in the expert elicitation.

43  
44 The HES agrees with EPA’s contention that a majority of health effect benefits from reduction of  
45 exposure to PM occurs within the first few years following reduced exposure. The HES therefore  
46 supports EPA’s choice of 20-year distributed cessation lag structure, skewed towards benefit gains in

1 the first few years. However, EPA should further examine the decay function and determine whether  
2 a more rapid decay model is appropriate. Further, the HES fully supports EPA's use of a no-  
3 threshold model to estimate the mortality reductions associated with reduced PM exposure.

4  
5 EPA's estimates an association between PM exposure and respiratory inflammation and  
6 infection leading to mortality in children under 5 years of age, relying on a cohort study  
7 (Woodruff et al., 1997). The HES generally supports this approach with some caveats. The age  
8 range of interest for respiratory-related mortality should be 1 month of age (since infections  
9 earlier than this are not likely to be from air pollution) to about 12 months of age. There are  
10 limited data available to estimate these effects and EPA should better describe the data  
11 challenges and uncertainties in these estimates.

12  
13 The HES supports the inclusion of mortality due to ozone exposure in the Section 812  
14 Prospective Analysis, given the evidence in the time-series literature and the consistency of these  
15 findings with morbidity studies. With respect to the concentration-response estimates, it is  
16 premature to use the cohort mortality evidence from Jerrett et al., 2009 as the basis for the  
17 Primary Estimate, because it has not yet been subjected to the vigorous critique that now  
18 supports, for example, analyses using the Pope and Laden studies for PM. For the time-series  
19 ozone mortality evidence, the HES supports the consideration of both the multi-city studies and  
20 the literature meta-analyses when generating the Primary Estimate. As there is not a clear basis  
21 for choosing one set of studies over the other, the HES recommends that the Primary Estimate be  
22 derived as an intermediate value between the multi-city studies and the meta-analyses. The  
23 uncertainty distribution can then reflect methodological uncertainties as well as the uncertainties  
24 reported within individual studies.

25  
26 The HES supports the EPA decision not to apply a cessation lag for ozone and also supports the  
27 assumption of no threshold for ozone-related mortality effects, as evidence is not conclusive to  
28 demonstrate such a threshold.

29  
30 In their analysis, EPA incorporates data on the baseline incidence and prevalence of the various  
31 health endpoints that are the basis of the benefits. The HES generally supports the choices made  
32 by the EPA. The most important data, in terms of driving the benefit estimates, are the baseline  
33 mortality data, which are of relatively high quality. There is some concern about the noise and  
34 inconsistency in data used to estimate baseline school loss and work loss days. There may be too  
35 much noise to make reasonably reliable quantitative estimates of air pollution work loss days.

36  
37 The Council had previously recommended that EPA perform an analysis of the sensitivity of their  
38 benefit results to differing PM composition in terms of both size and chemical composition. EPA  
39 determined that there is insufficient data to perform such an analysis. The HES supports this  
40 conclusion, given the state of the evidence but encourages EPA to continually revisit this  
41 conclusion as the literature evolves.

42  
43 EPA developed a dynamic population simulation model to explore how changes in population  
44 age structure change pollution-related premature mortality risks. EPA asked HES to comment on  
45 the methodology and results and to consider providing advice on the potential utility of further  
46 development of this approach. Overall, the HES commends EPA for demonstrating how dynamic

**3/24/2010: Council Draft Report for Quality Review -- Do not Cite or Quote**

This draft is a work in progress, does not reflect consensus advice or recommendations, has not been reviewed or approved by the chartered Council, and does not represent EPA policy

1 population modeling can be used in estimating the mortality impacts of long-term exposure to  
2 PM<sub>2.5</sub>. If practical, the approach would be a preferred one. However, the HES recognizes that  
3 these methods are complicated and computationally intensive, necessitating trade-offs between  
4 resolution of the results and run-time for the models. The HES recommends that the EPA  
5 describe in more detail the methods used and the reasons why EPA chose these particular  
6 methods over others.

7  
8 Overall, the HES finds the EPA analysis thorough and generally consistent with previous  
9 recommendations from the Council. The HES makes some minor suggestions for improvement  
10 and notes that the draft documents presented to the HES need technical editing to make the  
11 analyses and associated uncertainties more clear.

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## **BACKGROUND**

The reauthorization and amendment of the Clean Air Act in 1990 included specific language in Section 812 directing the EPA to perform benefit-cost studies of the programs affiliated with the Clean Air Act as a whole, relative to a consistent baseline. Congress expressed their intent that the comprehensiveness of the 812 studies should encourage and enable EPA to develop and continually refine its capabilities in clean air program assessment. Congress' stated objective was to ensure that EPA could provide better information on clean air program benefits and costs in support of future rounds of Clean Air Act reauthorization, whenever they may occur.

Included in the Section 812 amendments was specific language requiring EPA to convene a panel of outside experts in a range of relevant disciplines to advise the Administrator on the data chosen for the analysis, the selection of models used to conduct the analysis, and the validity of resulting estimates of Clean Air Act program benefits and costs. The Advisory Council on Clean Air Act Compliance Analysis (Council) was established in 1991 to fulfill this goal of providing multi-disciplinary outside expert review. Separate subcommittees have since been established to advise the parent Council on particular technical aspects of the Section 812 studies. The Air Quality Modeling Subcommittee (AQMS) was formed to advise the Council on issues of emissions estimation and air quality modeling. A Health Effects Subcommittee (HES) and an Ecological Effects Subcommittee (EES) exist today to advise the Council on issues associated with human health effects and ecological effects components, respectively.

In response to the Section 812 legislation, on October 15, 1997, EPA issued a retrospective study (1970 – 1990) of the benefits and costs of the Clean Air Act. EPA issued a first prospective study (1990 – 2010) on November 15, 1999 and is now completing a second prospective study (1990 – 2020).

The purpose of this Advisory is to review and provide guidance on the EPA draft human health effect estimates in chapters from the Second Section 812 Prospective analysis benefits report (1990 – 2020) as well as the human health components of the draft standalone uncertainty analysis report.

The Health Effects Subcommittee (HES) held a public meeting on December 15-16, 2009 to receive briefings and discuss the charge questions provided by the Agency. In addition to the Chair of the HES, who represents the HES on the Council, several additional member of the Council participated in the meeting, including Dr. James Hammitt, Chair of the Council, Dr. Michelle Bell, Dr. Jonathan Levy, and Dr. Arden Pope. A subsequent conference call of this committee was held on March 2, 2010, for the purposes of discussing recommendations made in this report.

1 **GENERAL CHARGE AND SUMMARY**

2

3 Charge question: EPA requests that the Council HES review the human health-related chapters  
4 and appendices of the draft Section 812 Second Prospective Study benefits and uncertainty  
5 reports. Consistent with the statutory language defining the role of the Council in reviewing the  
6 812 studies—and consistent with the role of the HES as advisor to the Council on human health  
7 effect estimation—EPA respectfully submits the following general charge questions to the HES:

8

9 a. Does the Council HES support the data choices made by the 812 Project Team for the  
10 development of the human health-related chapters and appendices of the draft benefits and  
11 uncertainty reports? If not, are there alternative data sets the Council HES recommends should  
12 be applied instead?

13

14 b. Does the Council HES support the methodological choices made for analyzing those data and  
15 developing the human health effect estimates for the relevant scenarios, and for characterizing  
16 their uncertainty? If not, are there alternative methodologies the Council HES recommends  
17 should be applied instead?

18

19 c. What advice does the HES have for the Council regarding the validity and utility of the human  
20 health effect analyses incorporated in the draft benefits report and the uncertainty analyses  
21 incorporated in the draft uncertainty report? If the validity and/or utility of the reports and their  
22 underlying analyses could be improved, what specific improvements does the Council HES  
23 recommend that the 812 Project Team consider, either for the present analysis or as part of a  
24 longer term research and development program?

25

26 HES response: The Council HES found the draft report to be generally sound, and members  
27 commend EPA on the quality of the present version. The very quality of this draft has made  
28 detailed criticism more feasible. EPA had to make an enormous number of choices of data  
29 collected from a variety of sources, and, using a variety of models, further processed the data.  
30 The scope and complexity of the data cannot be fully documented in a report of manageable size.  
31 Insofar as we have been able to probe the data, the Council HES generally supports the data  
32 approaches and judgments that have been made.

33

34 In particular, the HES supports the presentation of the PM mortality effect estimates and the data  
35 choices using the Pope et al., 2002 and Laden et al., 2006 studies. The HES notes that using the  
36 Pope studies (which are based on the ACS studies) for the Primary Estimate would provide  
37 lower health benefits estimates than the Six Cities study, which is considered by EPA to be  
38 equally applicable for health benefits analysis. The HES supports EPA's aim to give unbiased  
39 benefits estimates where possible. The HES proposes an alternative simple combination of the  
40 Pope and Laden studies. This alternative is discussed in the HES response to charge question 2a  
41 on the PM mortality concentration response function. With respect to PM, the HES also  
42 generally supports the Agency's proposed 20-year distributed cessation lag model, though most  
43 of the benefits of cessation appear to occur in the first few years after cessation of exposure. The  
44 HES commends the efforts to quantify infant mortality related to PM, even though, as the EPA  
45 acknowledges, these do not add a large amount to the overall benefits in the 812 Analysis, and

1 the estimates are based on only a small number of studies. The HES also supports the Agency's  
2 choice of a non-threshold model for PM-related effects.

3  
4 The generation of a concentration response function to estimate mortality effects of ozone is  
5 more difficult. The HES supports EPA's data choices for the Primary Estimates. Because time  
6 series studies were used for the Primary Estimate, a cessation lag for effects is not relevant, and  
7 the HES supports the Agency's use of a no-lag assumption for the primary mortality effect  
8 estimates for ozone. Finally, the HES supports EPA's use of a no-threshold model for ozone  
9 mortality effects, based on the time-series results. The HES supports the inclusion of, as an  
10 alternate estimate of benefits, a concentration-response function (CRF) from long-term exposure  
11 based on Jerrett et al. (2009).

12  
13 There should be a short but thoughtful discussion of the purposes of this report and how it may  
14 be used beyond the initial determination of the overall impact of the Clean Air Act and its  
15 amendments. There should also be some consideration of the likely background of expected  
16 readers (Congressional staffers? State health officials? Other?), and the report should be written  
17 to be comprehensible to them. This includes spelling out the meaning of, and assumptions  
18 underlying concepts such as Value of Statistical Life (VSL), which appear easy to understand but  
19 which have quite precise and non-intuitive technical meanings. Overall the report needs technical  
20 editing, and two sections (Chapter 6, PM mortality cessation lag and Chapter 7, Dynamic  
21 population modeling) are difficult to follow; these sections may need a complete re-write to  
22 achieve better clarity for non-experts.

23  
24 The HES makes some general observations and recommendations for improvement of the  
25 presentation in the draft documents. EPA generally models health effects in ways that imply  
26 multiplicative effects. The report should discuss this and say why other models (e.g., additive)  
27 were not used. The report should also explicitly say why it is limited to PM and ozone, when  
28 EPA regulates a much larger number of pollutants.

29  
30 There is no discussion of the differences between values at central monitors and what people  
31 actually inhale. The HES recognizes that broad data on inhaled levels of pollutants do not now  
32 exist, but some comment is in order. (Elementary considerations of epidemiology suggest that  
33 better measures of what is inhaled would probably lead to bigger estimates of effects.)

34  
35 The characterization of uncertainty is confusing and incomplete. It would be helpful to have an  
36 integrated assessment of overall uncertainty. Are the estimates within 10% of the true value?  
37 50%? 200%? Does it matter? Missing is a sense of what level of accuracy is needed in this  
38 report and how close the report may come to that. It is understandable that this is difficult to say,  
39 so uncertainty is broken down into components. However, the appendices are not comprehensive  
40 and the scope for "comprehensiveness" is not stated. It is not clear how the authors determined  
41 "key uncertainties". Rather than brief footnoted statements, it would be helpful to have  
42 introductory text describing the scope of the uncertainties listed, the significance classification  
43 definitions (e.g., "potentially major" and "probably minor"), and rationales for these choices.  
44 Further, it would be helpful to know the likely direction of bias and EPA's confidence in the  
45 assessment components.

1 On a related note, there are inconsistencies in presenting assumptions between the Benefits and  
2 Uncertainty drafts. A comparison of the assumptions (using a simple search on the “assum” in  
3 both documents) reveals a mismatch that may not be a problem but is a matter for clarification.  
4 Two sets of authors may have used different approaches or boundaries for identifying and/or  
5 recording assumptions. Perhaps the Benefits document authors sought to present all assumptions  
6 they made in their estimation processes; e.g., in extending study-specific or regional results to  
7 the national scale. If so, the rationale for some assumptions is not always clear (e.g., use of  
8 mother’s median wage rather than an average of mother and father median wages, which may  
9 yield a higher cost estimate). In contrast, the Uncertainty authors indicate that they intended to  
10 record only the most important assumptions made during their estimation processes.  
11

12 The HES suggests that a section be added to the report that compiles, in a brief form, the primary  
13 and secondary data sources and the model sources used. This section should include links to  
14 detailed descriptions of the data and models used. Finally, the report reveals a large number of  
15 research needs. It would help readers to add some discussion of the most important gaps,  
16 especially if EPA is to repeat this exercise at some future time.  
17

18 Overall, total mortality benefits from the Clean Air Act appear large in relation to total US  
19 mortality (about 7%). The report should note that this result is driven in part by the rise in  
20 expected pollution-related mortality in the absence of Clean Air Act, and not entirely by actual  
21 reductions in mortality. If EPA can estimate these separately, the report will be stronger and  
22 perhaps more credible.  
23

## 24 **SPECIFIC CHARGES**

25  
26  
27 Charge question 2a: PM Mortality Concentration-Response Function (CRF).  
28

29 Agency-supplied background: The current draft benefits report reflects adoption of the Pope et  
30 al. 2002 study as the basis for the Primary Estimates of the difference in incidences of PM-  
31 related premature mortality. Also within the main benefits report, an Alternative Estimate is  
32 presented prominently which is based on the Laden et al. 2006 study. Furthermore, the Project  
33 Team is currently assessing the potential significance of the recent Krewski et al. (2009)  
34 publication since it appears to strengthen the evidence for PM-related ischemic heart disease and  
35 lung cancer mortality and could provide the basis for a revised Primary Estimate or an additional  
36 Alternative Estimate. Uncertainty in the Primary Estimate is further described in the draft  
37 uncertainty report through graphical presentation of results obtained by applying each of the 12  
38 expert elicitation study functions to the differences in PM exposure estimated for the with-  
39 CAAA90 and without-CAAA90 core scenarios. In addition, the Project Team has recently been  
40 considering an approach developed by Industrial Economics, which uses a copula function to  
41 generate results representing the 12 expert functions. This approach is summarized in a draft  
42 briefing which the Project Team proposes to present to the HES on December 15 for its  
43 consideration.  
44

45 Charge question: Does the Council HES support these study selections and the organization and

1 presentation of PM mortality estimates in the draft benefits and uncertainty reports? In addition,  
2 a particular question for which the Project Team seeks HES advice is whether the application of  
3 mortality risk coefficients drawn from the Krewski et al. (2009) study should be considered for  
4 use in generating the Primary Estimate, or at least as the foundation for an Alternative Estimate.  
5 If the answer to either or both of these two questions is negative, are there alternative study  
6 choices and/or methods for generating, organizing, and presenting results which the Council  
7 HES recommends EPA consider?

8  
9 HES response: The HES emphasizes the continued importance of the American Cancer Society  
10 (ACS) and Harvard Six Cities cohort studies for underpinning our understanding of the mortality  
11 effects of PM<sub>2.5</sub> in the U.S. Findings from both cohorts have been robust to extensive analyses  
12 and independent re-analyses. The HES believes that the Pope et al. (2002) and Laden et al.  
13 (2006) analyses of the ACS and Six Cities cohorts, respectively, are at present the most useful  
14 findings from these studies. The Krewski et al. (2009) findings, while informative, have not yet  
15 undergone the same degree of peer review as have the aforementioned studies. Thus, the HES  
16 recommends that EPA not use the Krewski et al. (2009) findings for generating the Primary  
17 Estimate.

18  
19 The HES also reviewed the findings from EPA's Expert Elicitation (EE) study. The central  
20 effect estimates from the 12 experts cover a range that encompasses the Pope et al. (2002) effect  
21 estimate at the low end, and the Laden et al. (2006) effect estimate at the high end. This is  
22 illustrated in a figure (reproduced below, from an EPA Technical Memorandum dated November  
23 15, 2009 and presented to the HES), which shows the monetized PM-related mortality benefits  
24 associated with meeting a hypothetical 50 ppb NO<sub>2</sub> standard in 2020 for the studies of Pope al.  
25 and Laden et al. as well as the twelve experts consulted in the EE study. Further, a quantitative  
26 analysis by EPA consultants demonstrates that the Pope et al. (2002) effect estimate falls at about  
27 the 25<sup>th</sup> percentile of the EE distribution; the Laden et al. (2006) effect estimate falls at about the  
28 75<sup>th</sup> percentile of the EE distribution. HES reviewed the proposed copula function approach to  
29 combine the 12 response functions from the EE. While supporting EPA's investigation of  
30 alternative methods for combining evidence using advanced quantitative methods, the HES feels  
31 that adoption of the copula function approach as a prominent feature of the current prospective  
32 analysis might hinder interpretability and transparency of the findings for a general audience,  
33 given the very technical nature of the method.

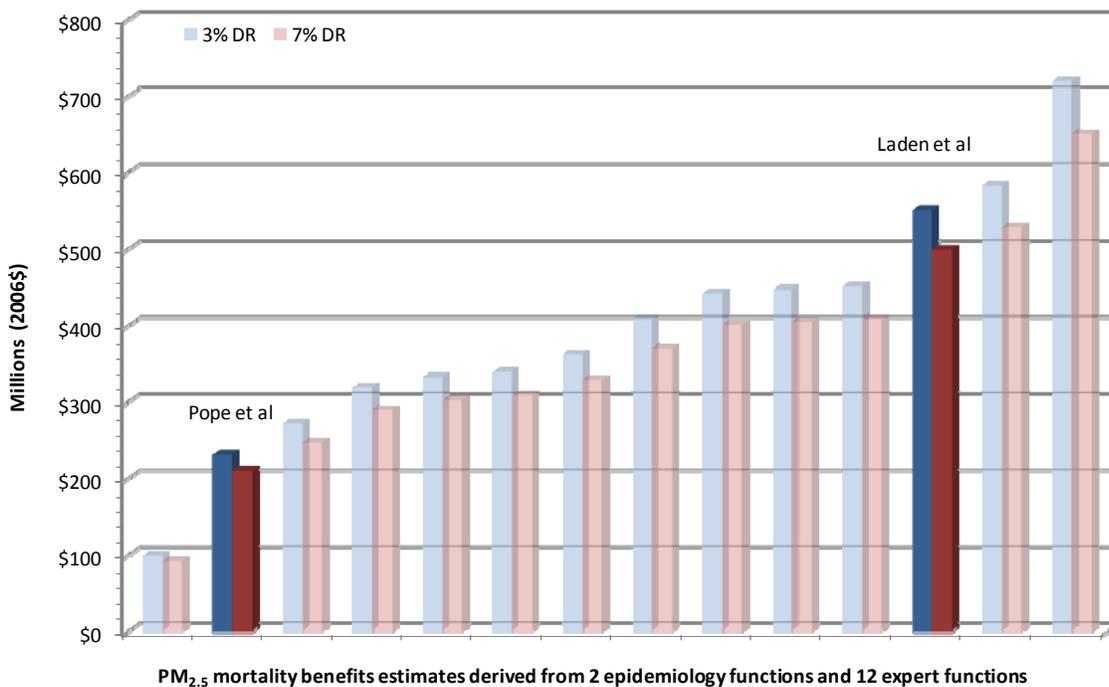
34  
35 The HES finds that the EE results from 2006 remain relevant; i.e. that new evidence since 2006  
36 informs, but does not fundamentally contradict, the data gathered then. On that basis, the HES  
37 supports the EE as the most comprehensive assessment to date of the mortality effect of PM<sub>2.5</sub>  
38 and so, given EPA's stated objective of generating unbiased benefits estimates in the Section 812  
39 prospective analysis, HES recommends that EPA adopt a new approach for developing its  
40 primary mortality benefits estimates. The new approach should reflect the range of EE opinions  
41 in a transparent way, while grounding the risk estimates in results from the two major  
42 contributing US cohorts. We suggest an approach along the following lines:

43  
44 Define a distribution of possible coefficients (perhaps a truncated normal distribution  
45 with zero probability below a value of zero, or a gamma distribution), with 25<sup>th</sup> percentile  
46 equal to a 0.6 percent change in mortality per 1 µg/m<sup>3</sup> change in annual average PM<sub>2.5</sub>

(the Pope et al., 2002 central estimate), with 75<sup>th</sup> percentile equal to 1.5 percent change in mortality per 1 µg/m<sup>3</sup> change in annual average PM<sub>2.5</sub> (the Laden et al., 2006 central estimate), and with mean equal to approximately the mean of these two values. Such a distribution would be adopted for the primary estimates.

The HES considered a formal method of integrating the opinions of the elicited experts, using a copula function. However, the HES decided not to recommend this approach, favoring instead a simpler and more transparent approach as described above.

### PM-related monetized benefits of attaining an NO<sub>2</sub> standard of 50ppb nationwide in 2020



Source: Technical Memorandum, Neal Fann, EPA/OAR/OAQPS to Jim DeMocker, EPA/OAR/OPAR Re: Estimating PM<sub>2.5</sub> and Ozone-related Premature Mortality Based on Risk Estimates from the Jerrett et al. (2009) and Krewski et al. (2009) Studies, November 15, 2009, Figure 3, page 4. Blue bars are based on a 3% discount rate; red bars represent a 7% discount rate

Charge question 2b: Cessation lag

Agency-supplied background: The Primary Estimates for PM mortality reflect an assumed lag between cessation of exposure and realization of the change in health effect incidence. Based in part on prior Council HES advice, the primary estimates in the draft benefits report reflect a 20-year distributed lag. Specifically, 30 percent of the total reduced incidences is assumed to occur in the first year following the exposure change. Another 50 percent of the total incidence changes

1 is be spread evenly over years two through five. The remaining 20 percent of the incidence  
2 change is spread evenly over years six through twenty. The effect of the cessation lag is realized  
3 through discounting (at a 5 percent rate) of the monetized value of future-year incidence changes  
4 (i.e., there is no need, and no intent, to represent the discounted values as reflecting direct  
5 discounting of incidences *per se*). In addition, the draft uncertainty report evaluates the effect of  
6 alternative lag structures. These alternatives include the 5-year distributed lag applied in the First  
7 Prospective Study and a set of smoothed lag functions derived from consideration of the results  
8 of available cohort and intervention studies.

9  
10 Charge question: Does the Council HES support the use of the 20-year distributed lag structure  
11 described above for generation of the Primary Estimates of the monetary value of PM mortality  
12 incidence reduction and the specific alternative lag functions presented in the draft uncertainty  
13 report? If not, are there alternative study choices and/or methods for organizing and presenting  
14 results that the Council HES recommends EPA consider?

15  
16 HES response: EPA has done an admirable job responding to the suggestions of earlier reviews  
17 by the Council and NAS. However, EPA should cite and include information from the recent  
18 analyses of the Nurses' Health Study (Puett et al., 2009) and the Harvard Six Cities Study  
19 (Schwartz et al., 2008; Laden et al., 2006). These studies suggest that most of the health effects  
20 of exposure (and benefits from reduction) occur within a few years. EPA assumes that 80% of  
21 the risk reduction occurs in the first five years. However, the EPA analysis of alternative  
22 assumptions about the lag using a given cohort study indicates that the 20-year distributed lag  
23 default assumption generates a result that is close to the mean of a range of reasonable  
24 assumptions. Therefore, in the face of uncertainty, this lag structure is appropriate.

25  
26 The HES suggests that if the decay function approach is used, EPA should ensure that its choice  
27 of parameter  $k$  is consistent with its choice of risk coefficient, in terms of the cohort studies used  
28 to generate both.

29  
30  
31 Charge question 2c: PM Infant Mortality

32  
33 Agency-supplied background: EPA's current approach to estimating the association between PM  
34 exposure and respiratory inflammation and infection leading to premature mortality in children  
35 under 5 years of age relies on the cohort study conducted by Woodruff et al. (1997). This is  
36 based in part on prior (SAB-HES) advice, which noted several strengths of the study, including  
37 the use of a larger cohort drawn from a large number of metropolitan areas and efforts to control  
38 for a variety of individual risk factors in infants (e.g., maternal educational level, maternal  
39 ethnicity, parental marital status, and maternal smoking status). A more recent study by  
40 Woodruff et al. (2006) continues to find associations between PM<sub>2.5</sub> and infant mortality, and  
41 also found the most significant relationships with respiratory-related causes of death.

42  
43 Charge question: Does the Council HES recommend continued reliance on the Woodruff et al.  
44 (1997) study to characterize the association between PM exposure and respiratory inflammation  
45 and infection leading to premature mortality in children under 5 years of age, or recommend that

1 the relationship be characterized by the more recent Woodruff et al. (2006) study, or recommend  
2 some other approach that relies on a third study or some combined consideration of multiple  
3 studies? Are there specific reasons to favor the results of one of these studies or of another  
4 study?

5  
6 HES response: The HES supports EPA's decision to include infant mortality in its analysis.  
7 Although its inclusion has only a small impact on overall benefits, compared with PM effects on  
8 adult mortality, incorporating infant mortality not only is consistent with the Agency's goal of  
9 comprehensiveness but also demonstrates the impacts of PM across the entire human lifespan.

10  
11 An increasing body of literature relates infant mortality and PM exposure. For example, the Ritz  
12 et al., 2006 study of Southern California data further informs the PM-infant mortality  
13 relationships found in the Woodruff et al. studies (1997 and 2006). While there are some  
14 important differences between the available studies within and outside of the United States, the  
15 results consistently show positive associations between PM (both PM<sub>10</sub> and PM<sub>2.5</sub>) and infant  
16 mortality. When PM<sub>10</sub> results are scaled to estimate PM<sub>2.5</sub> impacts, the results yield similar risk  
17 estimates. The number of studies now available may be sufficient to consider pooling results,  
18 rather than relying on a single study, thereby deriving a more robust risk estimate. The strengths  
19 and weaknesses of each study should be assessed to determine whether pooling or using a single  
20 study is the appropriate approach for this analysis.

21  
22 To summarize, several studies, both within and outside of the U.S., have resulted in similar risk  
23 estimates for PM impacts on infant mortality. The committee recommends that EPA do a  
24 reasoned evaluation of relevant studies and synthesize evidence across the studies.

25  
26  
27 Charge question 2d: PM Mortality Effect Threshold

28 Agency-supplied background: Consistent with prior Council and NAS advice, the Project Team  
29 did not attempt to alter the Pope 2002 CRF to reflect an assumed concentration threshold below  
30 which PM concentration changes would yield no change in estimated incidences. In addition to  
31 the lack of compelling evidence for particular effects thresholds, the Project Team is not aware  
32 of any valid procedure for the altering the CRF above an assumed threshold. In other words, the  
33 Project Team presumed that imposition of an (arbitrary) threshold would require respecification  
34 of the CRF to ensure a "with threshold" CRF slope that would accurately account for the total  
35 change in incidence expected based on the epidemiological study from which the CRF was  
36 derived. Prior efforts to apply a threshold simply truncated the incidence change estimated from  
37 a no-threshold CRF, though prior SAB advice indicates this is improper and the Project Team  
38 chose not to apply such an adjustment in the current analysis.

39  
40 Charge question: Does the Council HES support the use of a no-threshold model for generation  
41 of the Primary Estimates of PM mortality incidence reduction? If not, are there methods for  
42 estimating and applying an effects threshold that the Council HES recommends EPA consider,  
43 either for the Primary Estimates or for presentation in the draft uncertainty report?

1 HES response: The HES fully supports EPA's decision to use a non-threshold model to estimate  
2 mortality reductions. This decision is supported by the data, which are quite consistent in  
3 showing effects down to the lowest measured levels. Analyses of cohorts using data from more  
4 recent years, during which time PM concentrations have fallen, continue to report strong  
5 associations with mortality. Therefore, there is no evidence to support a truncation of the CRF.  
6

7  
8 Charge question 2e: Ozone Mortality Concentration-Response Function (CRF)  
9

10 Agency-supplied background: Based in part on prior Council and NAS advice, EPA has included  
11 changes in ozone-related premature mortality as part of the Primary Estimate of benefits in the  
12 draft benefits report. Recognizing the ongoing uncertainty regarding the appropriate study or  
13 studies from which a quantitative CRF should be derived, the Project Team adopted a  
14 placeholder function for the Primary Estimate of changes in ozone mortality which encourages  
15 focus on several key factors: study selection, pooling across studies, and pooling methodology.  
16 Given the particular uncertainties regarding the reasonableness of pooling across the multi-city  
17 NMMAPS 11 studies and the meta-analyses, the Project Team specified a CRF for the Primary  
18 Estimate which reflects inverse variance-weighted pooling of the Bell et al. 2004 and Schwartz  
19 2005 mortality effect estimates, both of which reflect an all-cause mortality endpoint. In  
20 addition, the draft uncertainty report presents alternative results obtained by applying CRFs  
21 derived from each of the three individual multi-city time-series studies and three meta-analyses.  
22 Furthermore, EPA has developed an alternative CRF based on the Jerrett et al. (2009) long-term  
23 ozone mortality study. This approach is described in the technical memorandum included in the  
24 package of review documents.  
25

26 Charge question: Does the Council HES support the use of the ozone mortality CRF derived by  
27 pooling the Bell et al., 2004 and Schwartz 2005 studies for the Primary Estimate and the  
28 presentation of the six alternative estimates in the draft uncertainty report? A particular question  
29 for which the Project Team seeks HES advice is whether application of the respiratory mortality  
30 risk estimate drawn from Jerrett et al., 2009 might be suitable for use in generating the Primary  
31 Estimate, or at least for generation of an Alternative Estimate. If the answer to either, or both, of  
32 these two questions is negative, are there alternative study selection and/or pooling approaches  
33 the Council HES recommends EPA consider for the Primary Estimate in the draft main benefits  
34 report and/or for the Alternative Estimates presented in the draft uncertainty report?  
35

36 HES response: The HES supports the inclusion of ozone mortality in the Section 812 Prospective  
37 Analysis, given the growth of evidence in the time-series literature and the consistency of these  
38 findings with morbidity studies. In terms of the most appropriate ozone CRF, the HES finds that  
39 it is premature to use the cohort mortality evidence from Jerrett et al., 2009 as the basis for the  
40 Primary Estimate, in light of the lack of corroboration from other cohort studies. However, the  
41 HES supports its inclusion as an Alternative Estimate or other sensitivity analysis, as it would be  
42 valuable to convey its implications if, as we expect, the cohort mortality findings are  
43 corroborated elsewhere.  
44

1 For the time-series ozone mortality evidence, the HES supports the consideration of both the  
2 multi-city studies and the literature meta-analyses when generating the Primary Estimate. The  
3 multi-city studies have the advantage of a consistent methodology across cities and the possible  
4 reduction of publication bias, but meta-analytic approaches are the foundation of the CRFs  
5 elsewhere in the prospective analysis. In addition, some investigators have noted that NMMAPS  
6 produces significantly lower CRFs than other epidemiological investigations, which may be in  
7 part attributable to the question investigated and the methodological choices made by various  
8 investigators. As there is not a clear basis for choosing one set of studies over the other, the HES  
9 recommends that the Primary Estimate be derived as an intermediate value between the multi-  
10 city studies and the meta-analyses. The uncertainty distribution can then reflect methodological  
11 uncertainties as well as the uncertainties reported within individual studies.

12  
13 Elaborating on this point, on page 2-9 the report indicates that the mean of the estimates derived  
14 from the three meta-analyses and the mean of the estimates derived from the three NMMAPS-  
15 based studies will be presented. However, the Primary Estimate is instead derived from a pooling  
16 of a 95-city NMMAPS-based study (Bell et al. 2004) and a 14-city case-crossover study not  
17 directly tied to NMMAPS (Schwartz 2005), without weight on the other NMMAPS-based study  
18 (Huang et al., 2005) or the three meta-analyses. It would be better to present a Primary Estimate  
19 reflecting an intermediate value among the multi-city studies and the meta-analyses, with explicit  
20 consideration of the full body of evidence.

21  
22 The uncertainty analyses presented in Exhibit 4-4 raised additional questions regarding the CRFs  
23 chosen and their rationale. For example, the Schwartz (2005) paper reported a 0.23% increase in  
24 mortality per 10 ppb increase in 1-hour maximum ozone concentrations. Using these same  
25 metrics, Bell et al. (2004) reported a value of 0.34%, yet the mortality incidence estimate in  
26 Exhibit 4-4 was lower. This could be explained if the Section 812 Prospective Analysis used the  
27 one-week average ozone findings from Bell et al. (2004), but this goes against the stated  
28 averaging time preferences in the report. Similarly, the three meta-analyses report corresponding  
29 values of 0.34%, 0.39% and 0.41%, but the incidence estimates are substantially greater for these  
30 meta-analyses than for the multi-city studies. More information about the CRFs derived from  
31 each study (and the assumptions regarding averaging times) would help to clarify why the  
32 benefits estimates appear to vary more substantially than the original studies, and in general,  
33 efforts to choose CRFs consistent with this full body of literature would be supported.

34  
35 The HES notes that 'attributable deaths' as estimated using cohort mortality evidence (from  
36 long-term exposure) for PM and time-series mortality evidence (from short-term exposure) for  
37 ozone have potentially very different implications in loss of life expectancy, with mortality from  
38 long-term exposure likely to be much more significant. The HES is concerned about the  
39 implications for the economic valuation of mortality. In line with other guidance, EPA has  
40 adopted a VSL approach (as opposed to a life-year approach) within the Section 812 Prospective  
41 Analysis. The VSL approach treats all deaths as equivalent, irrespective of whether the  
42 associated loss of life expectancy is large or small. While accepting that this is standard practice,  
43 some HES members see it as counter-intuitive. The HES asks that EPA spell out, clearly, the  
44 assumptions underlying its valuation methods, and in particular why the average extent of life  
45 expectancy is considered irrelevant despite differences in method (time series vs. cohort), age, or  
46 other matters, so that readers can better understand the Benefits method and results. In addition

1 to differences by age in life-years lost, the HES accepts that because economic valuation of life-  
2 years would not likely be constant with age, it is not immediately obvious to what extent  
3 differential values should be applied to time-series vs. cohort mortality evidence. This should be  
4 addressed within the valuation uncertainty analysis, which was not provided to the HES for  
5 review.

6  
7  
8 Charge question 2f: Ozone Cessation Lag

9  
10 Agency-supplied background: Based on a perceived lack of empirical data to support  
11 specification of a cessation lag structure for ozone-related mortality effects, the Project Team has  
12 not attempted to apply a cessation lag structure for the Primary Estimate of ozone mortality  
13 reduction benefits in the draft benefits report, and alternatives are not evaluated in the draft  
14 uncertainty report.

15  
16 Charge question: Does the Council HES support the use of a no-lag assumption for the Primary  
17 Estimate of ozone mortality benefits presented in the draft benefits report? If not, are there  
18 methods for estimating and applying a cessation lag structure for ozone mortality that the  
19 Council HES recommends EPA consider, either for the Primary Estimates or for presentation in  
20 the draft uncertainty report?

21  
22 HES response: Given the conclusion of the HES that time-series evidence should be used for the  
23 Primary Estimate, the question of a cessation lag is not relevant, and no cessation lag should be  
24 applied.

25  
26 If Alternative Estimates are derived using cohort mortality evidence, there is no evidence in the  
27 literature to support a different cessation lag between ozone and particulate matter. The HES  
28 therefore recommends using the same cessation lag structure and assumptions as for particulate  
29 matter when utilizing cohort mortality evidence for ozone.

30  
31  
32 Charge question 2g: Ozone mortality effect threshold

33 Agency-supplied background: Based on a perceived lack of empirical data to support application  
34 of a concentration threshold for ozone-related premature mortality effects, the Project Team did  
35 not attempt to apply an effect threshold for the Primary Estimate of ozone mortality reduction  
36 benefits.

37  
38 Charge question: Does the Council HES support the use of a no-threshold model for generation  
39 of the Primary Estimates of ozone mortality incidence reduction? If not, are there methods for  
40 estimating and applying an effects threshold, which the Council HES recommends EPA,  
41 consider, either for the Primary Estimates or for presentation in the draft uncertainty report?

42  
43 HES response: The HES supports the use of a no-threshold model for ozone and mortality. The  
44 current scientific literature does not support a population-based threshold, as studies have found  
45 no supporting evidence for short-term exposure and only suggestive evidence for long-term

1 exposure. For example, time-series analysis of ozone and mortality in 98 U.S. urban studies  
2 examined four model structures for the concentration-response curve: linear; subset; threshold;  
3 and spline models (Bell et al., 2006). All findings support the application of a no-threshold  
4 model, and also support the traditionally-used shape of the concentration-response curve.  
5 Associations between ozone and mortality were present at low concentrations, nearing natural  
6 background levels. If a threshold for short-term ozone exposure and mortality exists, it is likely  
7 below the range of regulatory interest.

8  
9 With respect to increased mortality risk from long-term ozone exposure, there is inconclusive  
10 evidence that a threshold may be present. A study of long-term health effects of ozone in 96 U.S.  
11 metropolitan areas for almost 450,000 persons for the ozone season (April to September)  
12 identified an association between ozone and respiratory-related mortality (Jerrett et al., 2009). A  
13 threshold analysis included a model in which a linear relationship between ozone and  
14 respiratory-related mortality risk is assumed for ozone levels above the specified threshold, and  
15 no association is assumed for levels below the threshold. Model fit was improved under the  
16 threshold model, compared to a no-threshold model (p-value 0.06), providing weak evidence of a  
17 threshold at 56 ppb daily maximum ozone concentration. Given this limited evidence for a  
18 threshold, the HES recommends that analyses based on findings from this study be conducted  
19 both with the no-threshold model and with an assumed threshold model, as an alternative  
20 analysis.

21  
22  
23 Charge question 2h: Baseline Incidence / Prevalence Estimates.

24  
25 Agency-supplied background: Baseline incidence / prevalence are key determinants of the  
26 estimated changes in health effect incidence described in the draft benefits and uncertainty  
27 reports.

28  
29 Charge question: Does the Council HES support the choices made by the Project Team regarding  
30 baseline incidence / prevalence across the various human health endpoints incorporated in the  
31 Primary Estimate of benefits? If not, are there alternative baseline incidence / prevalence data  
32 which the Council HES recommends EPA consider, either for the Primary Estimates or for  
33 presentation in the draft uncertainty report?

34  
35 HES response: The HES generally supports the choices made by the project team regarding  
36 baseline incidence/prevalence across the various human health endpoints. The HES recognizes  
37 that many of these choices are judgments within significant data constraints, and that the  
38 projected estimates have substantial uncertainties. The most important data, in terms of driving  
39 the benefit estimates, are the baseline mortality data, which are of relatively high quality. There  
40 is some concern about the noise and inconsistency in data used to estimate baseline school loss  
41 and work loss days. There may be too much noise to make reasonably reliable quantitative  
42 estimates of air pollution work loss days.

43  
44  
45 Charge question 2i: PM Differential Toxicity Sensitivity Analysis

1 Agency-supplied background: In its review of the Second Prospective Study analytical blueprint,  
2 the Council recognized that the state of the science did not support development and application  
3 of assumptions regarding the potential differential toxicity of PM components suitable for  
4 informing the present analysis. However, the Council did encourage the Project Team to explore  
5 the feasibility of conducting a sensitivity analysis to gauge the potential significance of  
6 differential toxicity. After extensive review of the literature and analysis of options, the Project  
7 Team concluded that currently available data and methodologies remain insufficient to meet the  
8 challenge of developing a reasonably valid and usefully informative sensitivity analysis, even on  
9 a notional basis. Indeed, the Project Team concluded that the potential research utility of such a  
10 sensitivity analysis in the end did not appear to justify the risks from potential misinterpretation  
11 and misapplication of the results of such a sensitivity analysis. The Project Team's evaluation of  
12 the issue of differential toxicity is presented in chapter 5 of the draft uncertainty report.

13  
14 Charge question: Does the Council HES support the Project Team's decision to defer  
15 quantitative sensitivity analysis of potential PM component differential toxicity? If not, are there  
16 data or methods for conducting a quantitative analysis of PM component differential toxicity that  
17 the HES recommends EPA consider, or are there other aspects of differential PM component  
18 toxicity which the HES recommends should be addressed in the draft benefits and/or uncertainty  
19 reports?

20  
21 HES response: The Council had encouraged the EPA Project Team to explore the feasibility of  
22 conducting a sensitivity analysis to gauge the potential significance of differential toxicity. The  
23 Project Team has determined after a review of the literature that the currently available data were  
24 insufficient for developing an informative sensitivity analysis.

25  
26 The Uncertainty Analysis document objectively reviews the evidence for various components,  
27 although research beyond 2007 does not seem to be included. Several recent papers could be  
28 reviewed (e.g., Ostro et al. 2007; Ostro et al., 2008; Smith et al., 2009). The Project Team found  
29 a limited but growing literature addressing the health effects of various PM components,  
30 including (but not limited to) sulfate, nitrate, elemental carbon, organic carbon, and metals. They  
31 conclude that none of the components show consistently greater effects than PM as a whole;  
32 however, the epidemiological evidence base was clearly limited by the high correlations among  
33 many PM components (and between those components and PM as a whole). The Project Team  
34 concluded that "for this evidence base to be applicable to a differential toxicity analysis, it would  
35 need to be able to provide quantitative CRFs for all of the key components, derived in a manner  
36 so that the total reflected the observed effects of PM<sub>2.5</sub> and so that the estimates reflected  
37 possible interactions among components."

38  
39 The HES agrees that the evidence base at this time does not currently support this sort of  
40 assessment. Additionally, the HES finds that differential assessment – even if feasible – would  
41 not lead to substantially different results in an assessment such as the 812 Analysis, which deals  
42 with changes in the pollution mixture as a whole; and so it supports EPA's decision to omit  
43 differential toxicity from the present 812 analysis. However, the HES recognizes that benefits  
44 analysis of specific measures may affect particular constituents of PM only, and that – for other  
45 analyses – the issue of differential toxicity should be considered anew.

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Charge question 2j: Dynamic Population Modeling

Agency-supplied background: Chapter 7 of the draft uncertainty report describes the results of the Project Team’s application of a dynamic population simulation model to the evaluation of changes in pollution-related premature mortality risks. The Project Team continues to consider the potential utility of dynamic population modeling approaches and respectfully requests that the HES review the methodology and results and consider providing advice regarding the potential utility of further development and future application of this approach.

Charge question: Does the Council HES have recommendations regarding the potential value for future analyses of the dynamic population approach described in chapter 7, or any alternative approaches the HES may suggest for addressing the issue of population changes during a study’s reference period?

HES response: The HES appreciates the work EPA has done to illustrate the use of dynamic population modeling for estimating the mortality impacts of long-term exposure to PM2.5. In general, HES supports the use of dynamic population modeling where practicable because it provides the most realistic available modeling of how, over time, changes in population risk lead to changes in the size and age distribution of the population, with consequent implications for estimated mortality impacts, whether expressed as deaths or life years.

We note EPA’s concern that a full dynamic population implementation requires detailed projections for every year up to 2020 (compared with currently for 2000, 2010 and 2020 only), and that this is very resource-intensive if carried out at the small spatial scale of the current core methods. EPA should focus its dynamic population modeling on estimating mortality effects forward in time using one-year changes in exposure, based on the years 2000, 2010 and 2020, i.e. the approach reported by EPA in the Uncertainty Report, when comparing dynamic population modeling with core BenMap methods. As noted by EPA, this is an intermediate strategy, which for a modest increase in effort captures many (though not all) of the gains of dynamic population modeling.

The HES discussed how the dynamic population approach highlights some subtleties in how monetary values are linked with the mortality implications of different pollution scenarios. HES requests EPA to describe, in substantially more detail than at present, the methods it used, and the reasons why these methods rather than others were selected. This includes, but is not limited to, describing (i) the current (‘static population’) BenMap approach to estimating deaths postponed; (ii) how, within this approach, VSL values are linked with changes in risk or deaths, and the assumptions underlying this approach; (iii) why EPA favors valuation based on deaths / VSL rather than life years / VS LY; (iv) whether EPA can link monetary values to the results of dynamic population modeling, and if so how this might be done; and in particular (v) whether the changing year-on-year pattern of deaths, illustrated by dynamic population modeling, can be incorporated into the monetary analysis.

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## APPENDIX A: Specific Comments from Individual Members

-P. 1-6: It appears that the primary benefits result includes CAIR, but this has not yet been promulgated, and would not have influenced the year 2000 benefits in any event. It is later mentioned in the qualitative uncertainty analysis that the issue of assuming CAIR when it is not yet in place is a potentially major uncertainty, but it would be nice to provide even more insight about this question (since CAIR itself has been associated with benefits of \$70B in 1999 dollars and using some other “older” assumptions). Doing new runs assuming no CAIR is impractical, but this should be able to be quantified at first order given the previous RIA of CAIR.

P. 2-7, Exhibit 2-2: It is not clear why “low birth weight” is not quantified; it is a birth outcome which has long lasting impacts on development. Are there too few studies from which reliable estimates can be derived? Some mention of the decision for this outcome should be included in the text or as a footnote to this exhibit.

P. 2-8, Exhibit 2-3, footnote a: In the last sentence “biological similarity” is puzzling. Children between the ages of 5 and 17 continue to develop biologically, including in the nasal-respiratory tract. Is EPA saying that the NRC determined that the differences in respiratory system developmental stages for ages 5-17 were not significant for estimating PM-related health effects? Please clarify or correct this statement; as currently written it is too vague and broad.

P. 2-10: It is not true that the individual time-series studies either used 24-hour average or 1-hour maximum levels for exposures; multiple studies did use 8-hour maximum. For example, Fairley (2003), Klemm (2000), Michelozzi (1998), Saez (2002), Anderson (2001), Bremmer (1999), Roemer (2001) all used 8-hour maximum. These studies (and probably others) are embedded in the meta-analyses.

P. 2-12, School absence section:  
The recommendation from the NRC should be briefly stated so the reader can understand the justification for extending the cited studies to a wider age range. Further, in the last sentence of this section the logic used for deriving the estimate should be expanded.

P. 2-15: It seems strange to argue that there is an extensive body of literature on CHA and RHA and then to choose only 2 studies for the estimates. Why not use, for example, the meta-analyses conducted in Europe in 2006 by the Committee on the Medical Effects of Air Pollutants, which pooled 50+ studies of CHA in a very careful manner?

P. 2-17 to 2-18, Baseline Incidence Rates section:  
The title for this section does not match that of the related exhibit. More importantly, some of the data sources and their limitations are not described in this section. For example, the “School Loss Days” database parameters (e.g., ages covered) and challenges in using them are not included in the text.

P. 2-18, last paragraph: The discussion of the asthma prevalence rates and assumptions needs to be clarified. Does “in future years” refer to post-1999 (e.g., after the ALA data), or from 2009

1 based on 1999, or something else? It is not clear whether the basis for the “current trends” in the  
2 first half of the last sentence is the study cited or whether another study and/or national database  
3 (Ostro et al, CDC, ALA, etc) was/were used for this comment. Further, a trend requires at least  
4 two points in time; what were those points? There is no recognition of whether rates have been  
5 affected by changes in diagnostic, treatment and/or reporting practices that may affect predicted  
6 rates. Last, the 4% chosen may be relevant for the 2020 national population but not for at-risk  
7 subpopulations, such as those noted in Exhibit 2-6. Are demographic changes in these  
8 subpopulations expected to result in overall population prevalence rates that would exceed 4%?  
9 Clarification of these several issues would help the reader understand the bases for and therefore  
10 the validity of the asthma data used and the 2020 prevalence rate assumption.  
11

12 P. 2-20: Exhibit 2.5. Baseline Incidence/Prevalence Rates

- 13 • In the first row (Mortality), the scope and years of data used should be clarified further.  
14 Because “infant mortality” was not specified as a row item, it was not clear to this reviewer  
15 whether infant mortality data were included. Wonder includes infant mortality data for 1995-  
16 2005; were any of these data (or only 1996-1998?) used to derive the risk estimate for the “<  
17 18” rate? If infant mortality data were included, the Mortality row needs to include an “infant  
18 mortality” line for the data source and years of data used. If not, the ages for the “< 18”  
19 column need to be clarified.
- 20 • In the row marked “School Loss Days,” the National Center for Education Statistics (1996)  
21 database is cited. However, this data source is not easily located on the Internet. The citation  
22 for this database is currently missing and needs to be listed in the References section of this  
23 chapter.

24 P. 2-27 and Exhibit 2.14: This reviewer assumes that “School Loss Days” on p. 2-27 became the  
25 “Work Loss Days” in the exhibit. Clarify in the text whether this is the case or not. Further, if  
26 “Work Loss Days” includes several endpoints, then all of the inclusions should be clearly stated.  
27

28 P. 2-29 to 2-31: Applying the eyeball test, it seems strange that mortality incidence is about 3-4  
29 times higher at the mean than the 5<sup>th</sup> percentile, while valuation is about 70 times higher. This  
30 patterning doesn’t exist for morbidity endpoints or for PM mortality, and the text did not mention  
31 (that I noticed) any use of lower valuation for ozone mortality even as a bounding calculation.  
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33

34 Uncertainty Analysis

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36 P. C-8: I’m not sure if I agree with some of the “major/minor” conclusions drawn in Table C-4.  
37 For example, the exclusion of populations under 30 from the mortality health impact assessment  
38 will have a trivial effect on the risk calculations, given the very low baseline mortality rate in  
39 these populations. It seems unlikely that the exclusion of air toxics has a potentially major effect  
40 on the net benefit estimate, as previous studies have shown air toxics cancer risks to be orders of  
41 magnitude less than criteria pollutant risks. The two ozone mortality incidence estimates that  
42 were pooled were from the same study and differed minimally, so this would not be a major  
43 effect.  
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1 P. C-8-11, Table C-4: The rationale for the choice of health effects and their related  
2 uncertainties documented in this table are not apparent. While adult mortality-related  
3 assumptions and uncertainties must be described because they drive the overall estimate, the  
4 table also presents information about an adult morbidity factor. Chronic bronchitis (the  
5 second most important contributor to the overall estimate) is included in the table, but there is  
6 no mention of “nonfatal myocardial infarction,” which is another morbidity outcome and a  
7 close third contributor to the health effects estimate. Additionally, there is no mention of  
8 infant mortality or other childhood health effects.  
9 Whether the purpose of this table was to capture the “key uncertainties” for the top three  
10 contributors to the health estimate or for adult outcomes only, there are inconsistencies within  
11 either scope. The rationale for the choice of health effects and their related uncertainties  
12 documented in this table should be clearly stated in supporting text.