



**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 with additional experts from the Council to conduct a review of 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.

Also in accordance with Section 812, the HES met with the goal 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. Specifically, the OAR asked the HES to consider: 1) the data choices and methodologies used to develop mortality estimates as a function of air pollution concentration for both particulate matter (PM) and ozone; 2) The methods and data used to develop estimates of the lag time between cessation of exposure and reduction in health effects for both PM and ozone; 3) the Agency's assumptions about a threshold concentration for mortality effects for both PM and ozone; 4) the estimates of infant mortality related to PM exposure; 5) Baseline incidence and prevalence of relevant disease conditions in the population; 6) the sensitivity analysis of the analytical results to differences in PM composition; and 7) the approach for dynamic modeling of U.S. population demographic changes.

The HES compliments EPA on a well crafted and implemented analysis. 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 all previous advice in conducting this analysis.

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1 The HES finds the data sources and analytical methodology to be generally sound and well
2 conceived, suggesting that the final results are reasonable, if not a bit conservative. While the HES
3 recognizes that in an effort such as this that uses a number of different data sources and that has
4 many complicated analytical components, a full and complete description becomes difficult, if not
5 impossible. Nonetheless, the HES recommends some items that EPA should modify or clarify in
6 developing its final report. The HES also recommends some minor methodological improvements in
7 response to some of the charge questions. These recommendations, in no way, should detract from
8 the HES overall enthusiasm for the quality of this analysis.
9

10
11 Sincerely,
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13
14
15 Dr. James Hammitt
16 Chair
17 Advisory Council on Clean Air Compliance Analysis

Dr. John Bailar
Chair
Health Effects Subcommittee

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NOTICE

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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
6 Agency. The Council is structured to provide balanced, expert assessment of scientific matters
7 related to problems facing the Agency. This report has not been reviewed for approval by the
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1
2 U.S. Environmental Protection Agency
3 Advisory Council on Clean Air Compliance Analysis
4 Health Effects Subcommittee, Augmented with Members of the Council
5

6 **COUNCIL MEMBERS**

7 **Dr. John Bailar**, Chair of the Health Effects Subcommittee, Scholar in Residence, The National
8 Academies, Washington, DC
9

10 **Dr. Michelle Bell**, Associate Professor, School of Forestry and Environmental Studies, Yale
11 University, New Haven, CT
12

13 **Dr. James K. Hammitt**, Professor, Department of Health Policy and Management, Harvard
14 School of Public Health, Boston, MA
15

16 **Dr. Jonathan Levy**, Associate Professor, Department of Environmental Health, Harvard School
17 of Public Health, Landmark Center, Boston, MA
18

19 **Dr. Arden Pope**, Professor, Department of Economics, Brigham Young University , Provo, UT
20
21

22 **SUBCOMITTEE MEMBERS**

23 **Mr. John Fintan Hurley**, Research Director, Institute of Occupational Medicine (IOM),
24 Edinburgh, United Kingdom, UK
25

26 **Dr. Patrick Kinney**, Professor, Department of Environmental Health Sciences, Mailman School
27 of Public Health , Columbia University, New York, NY
28

29 **Dr. Michael T. Kleinman**, Professor, Department of Medicine, Division of Occupational and
30 Environmental Medicine, University of California, Irvine, Irvine, CA
31

32 **Dr. Morton Lippmann**, Professor, Nelson Institute of Environmental Medicine, New York
33 University School of Medicine, Tuxedo, NY
34

35 **Dr. Bart Ostro**, Chief, Air Pollution Epidemiology Unit, Office of Environmental Health
36 Hazard Assessment, California Environmental Protection Agency, Oakland, CA
37

38 **Dr. Rebecca Parkin**, Professor and Associate Dean, Environmental and Occupational Health,
39 School of Public Health and Health Services, The George Washington University Medical
40 Center, Washington, DC
41

42
43 **SCIENCE ADVISORY BOARD STAFF**

44 **Dr. Marc Rigas**, Designated Federal Officer, 1200 Pennsylvania Avenue, NW, Washington,
45 DC, Phone: 202-343-9978, Fax: 202-233-0643, (rigas.marc@epa.gov)

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U.S. Environmental Protection Agency
Advisory Council on Clean Air Compliance Analysis

CHAIR

Dr. James K. Hammitt, Professor, Center for Risk Analysis, Harvard University, Boston, MA

COUNCIL MEMBERS

Dr. John Bailar, Scholar in Residence, The National Academies, Washington, DC

Dr. Michelle Bell, Associate Professor, School of Forestry and Environmental Studies, Yale University, New Haven, CT

Dr. Sylvia Brandt, Assistant Professor, Department of Resource Economics, University of Massachusetts, Amherst, MA

Dr. Linda Bui, Associate Professor, Department of Economics, Brandeis University, Waltham, MA

Dr. Dallas Burtraw, Senior Fellow, Resources for the Future, Washington, DC

Dr. Ivan J. Fernandez, Professor, Department of Plant, Soil and Environmental Sciences, University of Maine, Orono, ME

Dr. Shelby Gerking, Professor, Department of Economics, University of Central Florida, Orlando, FL

Dr. Wayne Gray, Professor, Department of Economics, Clark University, Worcester, MA

Dr. D. Alan Hansen, Independent Consultant, Fremont, CA

Dr. Nathaniel Keohane, Director of Economic Policy and Analysis, Climate and Air, Environmental Defense Fund, New York, NY

Dr. Jonathan Levy, Associate Professor, Department of Environmental Health, Harvard School of Public Health, Boston, MA

Mr. Richard L. Poirot, Environmental Analyst, Air Pollution Control Division, Department of Environmental Conservation, Vermont Agency of Natural Resources, Waterbury, VT

Dr. Arden Pope, Professor, Department of Economics, Brigham Young University, Provo, UT

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1 **Dr. Armistead (Ted) Russell**, Professor, Department of Civil and Environmental Engineering,
2 Georgia Institute of Technology, Atlanta, GA

3

4 **Mr. Michael Walsh**, Independent Consultant, Arlington, VA

5

6

7 **SCIENCE ADVISORY BOARD STAFF**

8 **Ms. Stephanie Sanzone**, Designated Federal Officer, 1200 Pennsylvania Avenue, NW,
9 Washington, DC, Phone: 202-343-9697, Fax: 202-233-0643, (sanzone.stephanie@epa.gov)

1 **EXECUTIVE SUMMARY**

2
3 The amendment of the Clean Air Act in 1990 included specific language in Section 812 directing
4 the EPA to perform benefit-cost studies of the programs affiliated with the Clean Air Act.
5

6 In response to the Section 812 requirements, on October 15, 1997, EPA issued a retrospective
7 study (1970 – 1990) of benefits and costs of the Clean Air Act. EPA issued a first prospective
8 study (1990 – 2010) on November 15, 1999 and is now completing a second prospective study
9 (1990 – 2020).
10

11 The Health Effects Subcommittee (HES) of the Advisory Council on Clean Air Compliance Analysis
12 (Council) held a public meeting on December 15-16, 2009 to review and provide guidance to the
13 EPA on the draft human health effect estimates in chapters from the second section 812 prospective
14 analysis benefits report (1990 – 2020) as well as the human health components of the draft
15 standalone uncertainty analysis report. In addition to the Chair of the HES, who represents the HES
16 on the Council, Dr. James Hammitt, Chair of the council and several additional members of the
17 Council participated in the discussions.
18

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

37 The HES agrees with EPA’s contention that a majority of health effect benefit from cessation of
38 exposure to PM occurs within the first few years following cessation. The HES therefore supports
39 EPA’s choice of 20-year distributed lag structure, skewed towards benefit gains in the first few years.
40 However, EPA should further examine the decay function and determine if a more rapid decay model
41 is necessary. Further, the HES fully supports EPA’s use of a no-threshold model to estimate the
42 mortality reductions associated with reduced PM exposure.
43

44 EPA’s estimates an association between PM exposure and respiratory inflammation and
45 infection leading to premature mortality in children under 5 years of age, relying on a cohort
46 study (Woodruff et al., 1997). The HES generally supports this approach with some caveats. The

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1 age range of interest for respiratory-related mortality should be 1 month of age (since infections
2 earlier than this are not from air pollution) to about 12 months of age. There is limited data
3 available to make strong estimates of these effects. EPA should better describe the data
4 challenges and uncertainties in these estimates.

5
6 For estimates of the reductions in mortality resulting from reducing ozone concentrations, the HES
7 finds the

8
9 With respect to ozone, the HES supports the EPA decision to not apply a cessation lag for ozone and
10 also supports the assumption of no threshold for ozone-related mortality effects, as data is not
11 conclusive to support such a threshold.

12
13 In their analysis, EPA incorporates data on the baseline incidence and prevalence of the various
14 health endpoints that are the basis of the benefits. The HES generally supports the choices made
15 by the EPA. The most important data, in terms of driving the benefit estimates, are the baseline
16 mortality data, which are of relatively high quality. There is some concern about the noise and
17 inconsistency in data used to estimate baseline school loss and work loss days. There may be too
18 much noise to reasonably make reliable quantitative estimates of air pollution work loss days.

19
20 The Council had previously recommended that EPA perform an analysis of the sensitivity of their
21 benefit results to differing PM composition (both in terms of size and chemical composition). EPA
22 determined that there is insufficient data to perform such an analysis. While this may be true for
23 chemical composition, some recent data may allow an analysis of the effects of different particle size
24 distributions.

25
26 EPA developed a dynamic population simulation model to explore how changes in population
27 age structure change pollution-related premature mortality risks. EPA asked HES to comment on
28 the methodology and results and consider providing advice regarding the potential utility of
29 further development and future application of this approach. Overall, the HES commends EPA
30 for developing this to demonstrate how dynamic population modeling can be used in estimating
31 the mortality impacts of long-term exposure to PM_{2.5}. Clearly, if practical, the approach would
32 be a preferred one. However, the HES recognizes that these methods are complicated and
33 computationally intensive, necessitating some tradeoffs between resolution of the results and run-
34 time for the models. The HES recommends that the EPA describe in more detail, the methods
35 used and the reasons why EPA chose these particular methods over others.

36
37 Overall, the HES finds the analysis thorough and the results of the analysis reasonable...

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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 that 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 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.

GENERAL CHARGE AND SUMMARY

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

- 1
2 a. Does the Council HES support the data choices made by the 812 Project Team for the
3 development of the human health-related chapters and appendices of the draft benefits and
4 uncertainty reports? If not, are there alternative data sets the Council HES recommends should
5 be applied instead?
6
7 b. Does the Council HES support the methodological choices made for analyzing those data and
8 developing the human health effect estimates for the relevant scenarios, and for characterizing
9 their uncertainty? If not, are there alternative methodologies the Council HES recommends
10 should be applied instead?
11
12 c. What advice does the HES have for the Council regarding the validity and utility of the human
13 health effect analyses incorporated in the draft benefits report and the uncertainty analyses
14 incorporated in the draft uncertainty report? If the validity and/or utility of the reports and their
15 underlying analyses could be improved, what specific improvements does the Council HES
16 recommend that the 812 Project Team consider, either for the present analysis or as part of a
17 longer term research and development program?
18

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

27 In particular, the HES supports the presentation of the PM mortality effect estimates and the data
28 choices using the Pope et al., 2002 and Laden et al., 2006 studies. The HES notes that using the
29 Pope studies for the primary estimate may result in fairly conservative results. The HES
30 supports EPA's position, that it's aim is to give best estimates, not conservative ones. The HES
31 proposes an alternative simple combination of the Pope and Laden studies. This alternative is
32 discussed in the HES response to charge question 2a on the PM mortality concentration response
33 function. With respect to PM, the HES also generally supports the Agency's proposed 20-year
34 distributed cessation lag model, though it should be made clear that most of the benefits of
35 cessation appear to occur in the first few years after cessation of exposure. The HES also
36 commends the efforts to quantify infant mortality related to PM, even though, as the EPA
37 acknowledges, these do not add a large amount to the overall benefits in the 812 Analysis, and
38 the estimates are based on only a small number of studies. The HES also supports the Agency's
39 choice of a non-threshold model for PM-related effects.
40

41 The generation of a concentration response function to estimate mortality effects of ozone is
42 more difficult. The HES supports EPA's data choices for the Primary Estimates. Because time
43 series studies were used for the Primary Estimate, a cessation lag for effects is not relevant, and
44 the HES supports the Agency's use of a no-lag assumption for the primary mortality effect
45 estimates for ozone. Finally, the HES supports EPA's use of a no-threshold model for ozone

1 mortality effects, based on the time-series results. The HES supports the inclusion, as an
2 Alternative Estimate of benefits, a CRF from long-term exposure based on Jerrett et al. (2009).

3
4 There should be a short but thoughtful discussion of the purposes of this report and how it may
5 be used beyond the initial determination of the overall impact of the Clean Air Act and its
6 amendments. There should also be some discussion of the likely background of expected readers
7 (Congressional staffers? State health officials? Other?), and the report should be written to be
8 comprehensible to them. This includes spelling out the meaning of, and assumptions underlying
9 concepts such as Value of Statistical Life (VSL), which appear easy to understand but which
10 have quite precise and non-intuitive technical meanings. Overall the report needs technical
11 editing, and two sections (Chapter 6, PM mortality cessation lag and Chapter 7, Dynamic
12 population modeling) were difficult to follow, even for this committee of experts; these sections
13 may need a complete re-write to achieve better clarity for non-experts.

14
15 The HES makes some general observations and recommendations for improvement of the
16 presentation in the draft documents. Health effects are pretty consistently modeled in ways that
17 imply multiplicative effects. The report should discuss this and say why other models (e.g.,
18 additive) were not used. The report should also explicitly say why it is limited to PM and ozone,
19 when EPA regulates a much larger number of pollutants.

20
21 There is no discussion of the differences between values at central monitors and what people
22 actually inhale. The HES recognizes that broad data on inhaled levels of pollutants do not now
23 exist, but some comment is in order. (Elementary considerations of epidemiology suggest that
24 better measures of what is inhaled would probably lead to bigger estimates of effects.)

25
26 It would be helpful to have an integrated assessment of overall uncertainty. Are the estimates
27 here within 10% of the true value? 50%? 200%? Does it matter? What is missing is, at bottom,
28 a sense of what level of accuracy is needed in this report and how close the report may come to
29 that.

30
31 The report reveals a large number of research needs. It would help readers to add some
32 discussion of the most important gaps, especially if EPA is to repeat this exercise at some future
33 time.

34
35 The HES did not find specific references to the appendices in the main uncertainty document.
36 Although a general pointer to the appendices is on p. 5 (e.g., the appendix document “features a
37 comprehensive qualitative evaluation of key uncertainties”), this characterization does not orient
38 the reader to the technical boundaries and related rationales for the content in the appendices.

39 Like the EPA draft, the appendices are not comprehensive and the scope for
40 “comprehensiveness” is not stated. There is no supporting text in the appendix document. It is
41 not clear how the authors determined “key uncertainties”. For example, were the authors
42 documenting the uncertainties related to adults only, mortality only, the top three health effects
43 that contribute to the cost estimate, or something else? Rather than brief footnoted statements, it
44 would be helpful to have introductory text describing the scope of the uncertainties listed, the
45 significance classification definitions (e.g., “potentially major” and “probably minor”), and
46 rationales for these choices.

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

14
15 Finally, the HES suggests that a section be added to the report that compiles in a brief form the
16 primary and secondary data sources and the model sources used. This section should include
17 links to detailed descriptions of the data and models used.
18

19
20 **SPECIFIC CHARGE**

21
22 Charge question 2a: PM Mortality Concentration-Response Function (CRF).

23
24 Agency-supplied background: The current draft benefits report reflects adoption of the Pope et
25 al. 2002 study as the basis for the Primary Estimates of the difference in incidences of PM-
26 related premature mortality. Also within the main benefits report, an Alternative Estimate is
27 presented prominently which is based on the Laden et al. 2006 study. Furthermore, the Project
28 Team is currently assessing the potential significance of the recent Krewski et al. (2009)
29 publication since it appears to strengthen the evidence for PM-related Ischemic heart disease and
30 lung cancer mortality and could provide the basis for a revised Primary Estimate or an additional
31 Alternative Estimate. Uncertainty in the Primary Estimate is further described in the draft
32 uncertainty report through graphical presentation of results obtained by applying each of the 12
33 expert elicitation study functions to the differences in PM exposure estimated for the with-
34 CAAA90 and without-CAAA90 core scenarios. In addition, the Project Team has recently been
35 considering an approach developed by Industrial Economics which uses a Copula function to
36 generate results representing the 12 expert functions. This approach is summarized in a draft
37 briefing which the Project Team proposes to present to the HES on December 15 for its
38 consideration.

39
40 Charge question: Does the Council HES support these study selections and the organization and
41 presentation of PM mortality estimates in the draft benefits and uncertainty reports? In addition,
42 a particular question for which the Project Team seeks HES advice is whether the application of
43 mortality risk coefficients drawn from the Krewski et al. (2009) study should be considered for
44 use in generating the Primary Estimate, or at least as the foundation for an Alternative Estimate.
45 If the answer to either or both of these two questions is negative, are there alternative study
46 choices and/or methods for generating, organizing, and presenting results which the Council

1 HES recommends EPA consider?
2

3 HES response: The HES emphasizes the continued importance of the ACS and Six Cities cohort
4 studies for underpinning our understanding of the mortality effects of PM_{2.5} in the U.S.
5 Findings from both cohorts have been robust to extensive analyses and independent re-analyses.
6 The HES believes that the Pope et al. (2002) and Laden et al. (2006) analyses of the ACS and
7 Six Cities cohorts, respectively, currently represent the most useful findings from these studies.
8 The Krewski et al. (2009) findings, while informative, have not yet undergone the same degree
9 of peer review as have the aforementioned studies. Thus, the HES recommends that EPA not use
10 the Krewski et al. (2009) findings for generating the Primary Estimate at this time.
11

12 The HES also reviewed the findings from EPA's Expert Elicitation (EE) study. The central
13 effect estimates from each of the 12 experts cover a range that encompasses the Pope et al.
14 (2002) effect estimate at the low end, and the Laden et al. (2006) effect estimate at the high end.
15 This is illustrated in the figure below (from an EPA Technical Memorandum dated November
16 15, 2009 and presented to the HES) reproduced below, which shows the monetized PM-related
17 mortality benefits associated with meeting a hypothetical 50 ppb NO₂ standard in 2020 for the
18 studies of Pope et al. and Laden et al. as well as the twelve experts consulted in the EE study.
19 Further, a quantitative analysis by EPA consultants demonstrates that the Pope et al. (2002)
20 effect estimate falls at about the 25th percentile of the EE distribution; the Laden et al. (2006)
21 effect estimate falls at about the 75th percentile of the EE distribution. HES reviewed the
22 proposed Copula function approach to combine the 12 response functions from the EE. While
23 supporting EPA's investigation of alternative methods for combining evidence using advanced
24 quantitative methods, the HES feels that adoption of the Copula function approach as a
25 prominent feature of the current prospective analysis might hinder interpretability and
26 transparency of the findings for a general audience, given the very technical nature of the
27 method.
28

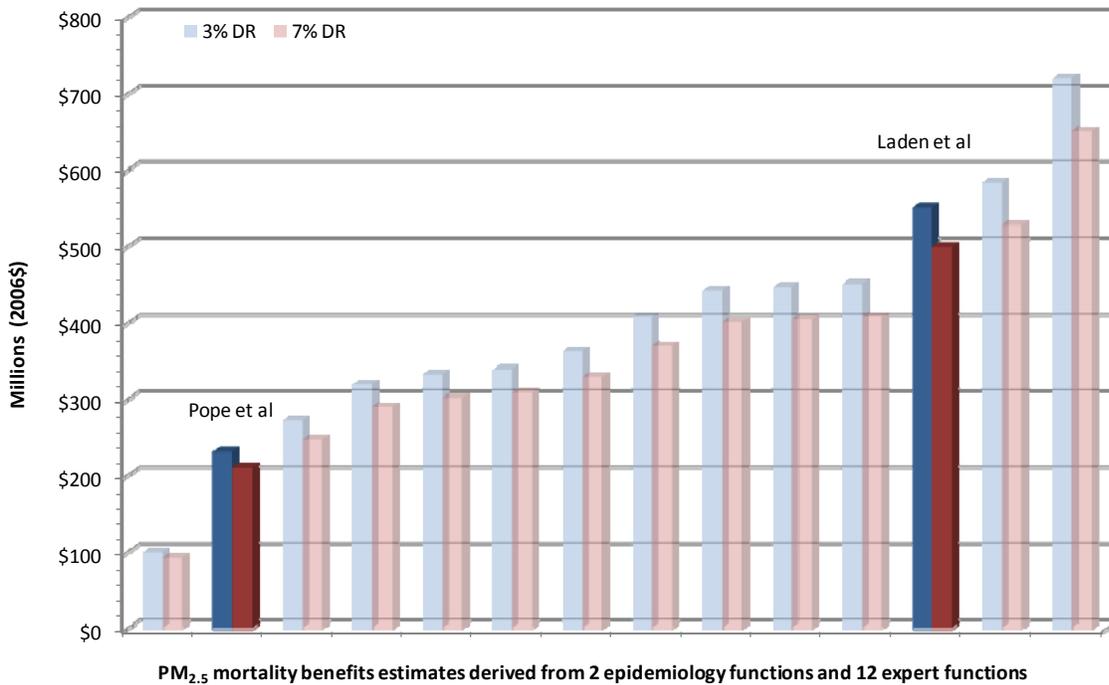
29 HES considered the EE results from 2006 remain relevant, i.e. that new evidence since 2006
30 informs, but does not fundamentally contradict, the data gathered then.
31

32 Based on these findings, there is a general view among HES members that the Pope et al. (2002)
33 results are likely to underestimate the national mortality effect of PM_{2.5}. Given EPA's stated
34 objective of generating unbiased benefits estimates in the 812 prospective analysis, HES
35 recommends that EPA adopt a new approach for developing its primary mortality benefits
36 estimates for the 812 analysis, as described in the following paragraph:
37

38 Define a distribution of possible coefficients (perhaps a truncated normal distribution
39 with zero probability below a value of zero, or gamma distribution), with 25th percentile
40 equal to a 0.6 percent change in mortality per 1 µg/m³ change in annual average PM_{2.5}
41 (the Pope et al., 2002 central estimate), with 75th percentile equal to 1.5 percent change in
42 mortality per 1 µg/m³ change in annual average PM_{2.5} (the Laden et al., 2006 central
43 estimate), and with mean equal to approximately the mean of these two values. Such a
44 distribution would be adopted for the primary estimates.
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PM-related monetized benefits of attaining an NO₂ standard of 50ppb nationwide in 2020



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Source: Technical Memorandum, Neal Fann, EPA/OAR/OAQPS to Jim DeMocker, EPA/OAR/OPAR Re: Estimating PM2.5 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 represents a 3% discount rate; red bars represent a 7% discount rate

10 Charge question 2b: Cessation lag

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

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28

Charge question: Does the Council HES support the use of the 20-year distributed lag structure described above for generation of the Primary Estimates of the monetary value of PM mortality incidence reduction and the specific alternative lag functions presented in the draft uncertainty

1 report? If not, are there alternative study choices and/or methods for organizing and presenting
2 results that the Council HES recommends EPA consider?
3

4 HES response: EPA has done an admirable job responding to the suggestions of earlier reviews
5 by the Council and NAS. However, EPA should cite and include information from the recent
6 analyses of the Nurses' Health Study (Pruett et al., 2009) and the Harvard Six Cities Study
7 (Schwartz et al., 2008; Laden et al., 2006). These studies suggest that most of the health effects
8 of exposure (and benefits from reduction) occur within a few years. However, the EPA analysis
9 of alternative assumptions about the lag using a given cohort study (i.e., exhibit 6-5 and 6-8)
10 indicates that the 20-year distributed lag default assumption generates a result that is close to the
11 mean of a range of reasonable assumptions. Therefore, in the face of uncertainty, this lag
12 structure strikes this committee as a reasonable assumption. A question remains as to whether a
13 similar result would hold under various alternative model assumptions. If not, than a lag
14 structure that suggests more immediate effects, such as a 5-year distributed lag, may be
15 warranted, especially if a relatively high discount rate like 7% is used.
16

17 There is an additional question about the ingenious analysis that EPA conducted to determine the
18 value of the decay function k . We note that the value of k is sensitive to the original cohort study
19 risk estimate. This may suggest that the methodology needs additional development or that
20 different pairs of studies can be used to generate the estimate of k . HES suggests that if the decay
21 function approach is used, EPA should ensure that its choice of parameter k is consistent with its
22 choice of risk coefficient, in terms of the cohort studies used to generate both.
23

24 Charge question 2c: PM Infant Mortality
25

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

36 Charge question: Does the Council HES recommend continued reliance on the Woodruff et al.
37 (1997) study to characterize the association between PM exposure and respiratory inflammation
38 and infection leading to premature mortality in children under 5 years of age, or recommend that
39 the relationship be characterized by the more recent Woodruff et al. (2006) study, or recommend
40 some other approach that relies on a third study or some combined consideration of multiple
41 studies? Are there specific reasons to favor the results of one of these studies or of another
42 study?
43

44 HES response: The committee applauds EPA for including infant mortality in its analysis.
45 Although its inclusion has only a small impact on overall benefits, compared with PM effects on

1 adult mortality, incorporating infant mortality not only is consistent with the Agency's goal of
2 comprehensiveness but also demonstrates the impacts of PM across the entire human lifespan,
3 not just in old or middle-aged persons.
4

5 An increasing body of literature relates infant mortality and PM exposure. For example, the Ritz
6 et al., 2006 study of Southern California data further informs the PM-infant mortality
7 relationships found in the Woodruff et al studies (1997 and 2006). While there are some
8 important differences between the available studies within and outside of the United States, the
9 results consistently show positive associations between PM (both PM₁₀ and PM_{2.5}) and infant
10 mortality. When PM₁₀ results are scaled to estimate PM_{2.5} impacts, the results yield similar risk
11 estimates. The number of studies now available may be sufficient to consider pooling results,
12 rather than relying on a single study, thereby deriving a more robust risk estimate. The strengths
13 and weaknesses of each study should be assessed to determine whether pooling or using a single
14 study is the appropriate approach for this analysis.
15

16 Important issues to consider include, for example, the size of the PM studied, the geographic
17 scale and its relevance to this analysis, the classification of infant deaths, the time periods (e.g.,
18 developmental stage) at the time of death, the ability to control potential confounders, and the
19 quality and relevance of the sources of data. Most studies have examined PM₁₀ rather than PM_{2.5}
20 because of the limitations of available monitoring data. While relying on the latter only would
21 be simpler and more relevant here, there is no national study that focuses on PM_{2.5} exposures.
22 Most studies have relied on estimating PM exposures based on monitoring stations within a
23 specified distance of the residence at the time of birth. Available studies have examined
24 different timeframes for exposures (same day as death and various lags prior to death) and for
25 deaths (e.g., neonatal (up to 27 days of life) and post neonatal (after 27 days to 1 year) periods).
26 Some have compared normal vs. low birth weight mortality rates, recognizing that regardless of
27 ambient exposures low birth weight has significant impacts on infant mortality. It is not clear
28 whether or how much the effect of low birth weight may be related to maternal exposure to air
29 pollutants; thus some judgment will be required in deciding whether and how to adjust PM
30 effects for low birth weight. Other important contributors to infant mortality such as prenatal
31 care, maternal smoking, maternal education, maternal ethnicity, parental marital status, etc) have
32 been controlled in many but not all studies. Furthermore, the assumptions and/or extrapolations
33 made to account for limitations in the exposure, mortality and confounder databases must be
34 evaluated for their potential impacts on the risk estimates. That said, it is striking that so many
35 studies both within and outside of the U.S. have resulted in similar risk estimates for PM impacts
36 on infant mortality.

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3 Charge question 2d: PM Mortality Effect Threshold

4 Agency-supplied background: Consistent with prior Council and NAS advice, the Project Team
5 did not attempt to alter the Pope 2002 CRF to reflect an assumed concentration threshold below
6 which PM concentration changes would yield no change in estimated incidences. In addition to
7 the lack of compelling evidence for any particular effects threshold, the Project Team is not
8 aware of any valid procedure for the altering the CRF above an assumed threshold. In other
9 words, the Project Team presumed that imposition of an (arbitrary) threshold would require
10 respecification of the CRF to ensure a “with threshold” CRF slope that would accurately account
11 for the total change in incidence expected based on the epidemiological study from which the
12 CRF was derived. Prior efforts to apply a threshold simply truncated the incidence change
13 estimated from a no-threshold CRF, though prior SAB advice indicates this is improper and the
14 Project Team chose not to apply such an adjustment in the current analysis.

15
16 Charge question: Does the Council HES support the use of a no-threshold model for generation
17 of the Primary Estimates of PM mortality incidence reduction? If not, are there methods for
18 estimating and applying an effects threshold that the Council HES recommends EPA consider,
19 either for the Primary Estimates or for presentation in the draft uncertainty report?

20
21 HES response: The Council HES fully supports EPA’s decision to use a non-threshold model to
22 estimate mortality reductions. This decision is supported by both the data, which are quite
23 consistent in showing effects down to the lowest measurable levels, and theory, which requires
24 that a population threshold be the average of the individual thresholds of all persons in the
25 population at risk, some of whom may have very low or absent personal thresholds because of
26 inherent variability or because any personal protective element may have been “used up” by
27 prior exposures to other toxic agents. Analyses of cohorts using data from more recent years
28 during which time PM concentrations have fallen, continue to report strong associations with
29 mortality. Therefore, there is no evidence to support a truncation of the CRF.

30

31 Charge question 2e: Ozone Mortality CRF

32

33 Agency-supplied background: Based in part on prior Council and NAS advice, EPA has included
34 changes in ozone-related premature mortality as part of the Primary Estimate of benefits in the
35 draft benefits report. Recognizing the ongoing uncertainty regarding the appropriate study or
36 studies from which a quantitative CRF should be derived, the Project Team adopted a
37 placeholder function for the Primary Estimate of changes in ozone mortality which encourages
38 focus on several key factors: study selection, pooling across studies, and pooling methodology.
39 Given the particular uncertainties regarding the reasonableness of pooling across the multi-city
40 NMMAPS 11 studies and the meta-analyses, the Project Team specified a CRF for the Primary
41 Estimate which reflects inverse variance-weighted pooling of the Bell et al. 2004 and Schwartz
42 2005 mortality effect estimates, both of which reflect an all-cause mortality endpoint. In
43 addition, the draft uncertainty report presents alternative results obtained by applying CRFs
44 derived from each of the three individual multi-city time-series studies and three meta-analyses.

1 Furthermore, EPA has developed an alternative CRF based on the Jerrett et al. (2009) long-term
2 ozone mortality study. This approach is described in the technical memorandum included in the
3 package of review documents.
4

5 Charge question: Does the Council HES support the use of the ozone mortality CRF derived by
6 pooling the Bell et al., 2004 and Schwartz 2005 studies for the Primary Estimate and the
7 presentation of the six alternative estimates in the draft uncertainty report? A particular question
8 for which the Project Team seeks HES advice is whether application of the respiratory mortality
9 risk estimate drawn from Jerrett et al., 2009 might be suitable for use in generating the Primary
10 Estimate, or at least for generation of an Alternative Estimate. If the answer to either, or both, of
11 these two questions is negative, are there alternative study selection and/or pooling approaches
12 the Council HES recommends EPA consider for the Primary Estimate in the draft main benefits
13 report and/or for the Alternative Estimates presented in the draft uncertainty report?
14

15 HES response: The Council HES supports the inclusion of ozone mortality in the Section 812
16 Prospective Analysis, given the growth of evidence in the time-series literature and the
17 consistency of these findings with morbidity studies. In terms of the most appropriate ozone
18 CRF, the HES finds that it is premature to use the cohort mortality evidence from Jerrett et al.,
19 2009 as the basis for the Primary Estimate, in light of the lack of corroboration from other cohort
20 studies. However, the HES supports its inclusion as an Alternative Estimate or other sensitivity
21 analysis, as it would be valuable to convey its implications if the cohort mortality findings were
22 corroborated elsewhere.
23

24 For the time-series ozone mortality evidence, the HES supports the consideration of both the
25 multi-city studies and the literature meta-analyses within the prospective analysis. The multi-city
26 studies have the advantage of a consistent methodology across cities and the possible reduction
27 of publication bias, but meta-analytic approaches are the foundation of the CRFs elsewhere in
28 the prospective analysis. In addition, some investigators have noted that NMMAPS produces
29 significantly lower CRFs than other epidemiological investigations, which may be in part
30 attributable to the hypotheses tested and the methodological choices made by various
31 investigators, and these methodological uncertainties should be captured within the Prospective
32 Analysis.
33

34 On page 2-9 the report indicates that the mean of the estimates derived from the three meta-
35 analyses and the mean of the estimates derived from the three NMMAPS-based studies will be
36 presented. However, the Primary Estimate is instead derived from a pooling of a 95-city
37 NMMAPS-based study (Bell et al. 2004) and a 14-city case-crossover study not directly tied to
38 NMMAPS (Schwartz 2005), without weight on the other NMMAPS-based study (Huang et al.,
39 2005) or the three meta-analyses. It would be preferable to see a primary estimate reflecting an
40 intermediate value among the multi-city studies and the meta-analyses, with explicit
41 consideration of the full body of evidence.
42

43 The uncertainty analyses presented in Exhibit 4-4 raised additional questions regarding the
44 precise CRFs chosen and their rationale. For example, the Schwartz (2005) paper reported a
45 0.23% increase in mortality per 10 ppb increase in 1-hour maximum ozone concentrations. Using
46 these identical metrics, Bell et al. (2004) reported a value of 0.34%, yet the mortality incidence

1 estimate in Exhibit 4-4 was lower. This could be explained if the Section 812 Prospective
2 Analysis used the one-week average ozone findings from Bell et al. (2004), but this goes against
3 the stated averaging time preferences in the report. Similarly, the three meta-analyses report
4 corresponding values of 0.34%, 0.39% and 0.41%, but the incidence estimates are substantially
5 greater for these meta-analyses than for the multi-city studies. More transparency regarding the
6 CRFs derived from each study (and the assumptions regarding averaging times) would help to
7 clarify why the benefits estimates appear to vary more substantially than the original studies, and
8 in general, efforts to choose CRFs consistent with this full body of literature would be supported.
9

10 The HES notes that ‘attributable deaths’ as estimated using cohort mortality evidence (from
11 long-term exposure) for PM and time-series mortality evidence (from short-term exposure) for
12 ozone have potentially very different implications in loss of life expectancy, with mortality from
13 long-term exposure likely to be much more significant. The HES raises questions about the
14 implications for the economic valuation of mortality. In line with other guidance, EPA has
15 adopted a VSL approach (as opposed to a life-year approach) within the Section 812 Prospective
16 Analysis. The VSL approach treats all deaths as equivalent, irrespective of whether the
17 associated loss of life expectancy is large or small. While accepting that this is standard practice,
18 some HES members see it as counter-intuitive. The HES asks that EPA spell out, clearly, the
19 assumptions underlying its valuation methods, and in particular why the extent of life expectancy
20 is considered irrelevant, so that readers can better understand the Benefits method and results.
21 The HES accepts that because economic valuation of life-years would not likely be constant with
22 age, it is not immediately obvious to what extent differential values should be applied to time-
23 series vs. cohort mortality evidence. This should be addressed within the valuation uncertainty
24 analysis, which was not provided to the HES for review.
25
26

27 Charge question 2f: Ozone Cessation Lag

28
29 Agency-supplied background: Based on a perceived lack of empirical data to support
30 specification of a cessation lag structure for ozone-related mortality effects, the Project Team has
31 not attempted to apply a cessation lag structure for the Primary Estimate of ozone mortality
32 reduction benefits in the draft benefits report, nor are alternatives evaluated in the draft
33 uncertainty report.
34

35 Charge question: Does the Council HES support the use of a no-lag assumption for the Primary
36 Estimate of ozone mortality benefits presented in the draft benefits report? If not, are there
37 methods for estimating and applying a cessation lag structure for ozone mortality that the
38 Council HES recommends EPA consider, either for the Primary Estimates or for presentation in
39 the draft uncertainty report?
40

41 HES response: Given the conclusion of the HES that time-series evidence should be used for the
42 Primary Estimate, the question of a cessation lag is not relevant, and no cessation lag should be
43 applied.
44

45 If Alternative Estimates are derived using cohort mortality evidence, there is no direct indication
46 from the epidemiological literature regarding a cessation lag that would be differential between

1 ozone and particulate matter. The HES therefore recommends use of the same cessation lag
2 assumptions as for particulate matter when utilizing cohort mortality evidence for ozone.

3
4

5 Charge question 2g: Ozone mortality effect threshold

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

10

11 Charge question: Does the Council HES support the use of a no-threshold model for generation
12 of the Primary Estimates of ozone mortality incidence reduction? If not, are there methods for
13 estimating and applying an effects threshold, which the Council HES recommends EPA,
14 consider, either for the Primary Estimates or for presentation in the draft uncertainty report?
15

15

16 HES response: The Council HES supports the use of a no-threshold model for ozone and
17 mortality. The current scientific literature does not support a population-based threshold, as
18 studies have found no supporting evidence for short-term exposure and only suggestive evidence
19 for long-term exposure. For example, time-series analysis of ozone and mortality in 98 U.S.
20 urban studies examined four model structures for the concentration-response curve: linear;
21 subset; threshold; and spline models (Bell et al., 2006). All findings support the application of a
22 no-threshold model, and also support the traditionally-used shape of the concentration-response
23 curve. Associations between ozone and mortality were present at low concentrations, nearing
24 natural background levels. If a threshold for short-term ozone exposure and mortality exists, it is
25 likely below the range of regulatory interest.

26

27 With respect to increased mortality risk from long-term ozone exposure, there is inconclusive
28 evidence that a threshold may be present. A study of long-term ozone in 96 U.S. metropolitan
29 areas for almost 450,000 persons for the ozone season (April to September) identified an
30 association between ozone and respiratory-related mortality (Jerrett et al., 2009). A threshold
31 analysis included a model in which a linear relationship between ozone and respiratory-related
32 mortality risk is assumed for ozone levels above the specified threshold, and no association is
33 assumed for levels below the threshold. Model fit was improved under the threshold model,
34 compared to a no-threshold model (p-value 0.06), providing weak evidence of a threshold at 56
35 ppb daily maximum ozone concentration. Given this limited evidence for a threshold, the HES
36 recommends that analyses based on findings from this study be conducted both with the no-
37 threshold model and with an assumed threshold model, as an alternative analysis.

38

39

40 Charge question 2h: Baseline Incidence / Prevalence Estimates.

41

42 Agency-supplied background: Baseline incidence / prevalence are key determinants of the
43 estimated changes in health effect incidence described in the draft benefits and uncertainty
44 reports.

45

1 Charge question: Does the Council HES support the choices made by the Project Team regarding
2 baseline incidence / prevalence across the various human health endpoints incorporated in the
3 Primary Estimate of benefits? If not, are there alternative baseline incidence / prevalence data
4 which the Council HES recommends EPA consider, either for the Primary Estimates or for
5 presentation in the draft uncertainty report?
6

7 HES response: The Council HES generally supports the choices made by the project team
8 regarding baseline incidence/prevalence across the various human health endpoints. HES
9 recognizes that many of these choices are judgments within significant data constraints, and that
10 the projected estimates have substantial uncertainties. The most important data, in terms of
11 driving the benefit estimates, are the baseline mortality data, which are of relatively high quality.
12 There is some concern about the noise and inconsistency in data used to estimate baseline school
13 loss and work loss days. There may be too much noise to reasonably make reliable quantitative
14 estimates of air pollution work loss days.
15
16

17 Charge question 2i: PM Differential Toxicity Sensitivity Analysis
18

19 Agency-supplied background: In its review of the Second Prospective Study analytical blueprint,
20 the Council recognized that the state of the science did not support development and application
21 of assumptions regarding the potential differential toxicity of PM components suitable for
22 informing the present analysis. However, the Council did encourage the Project Team to explore
23 the feasibility of conducting a sensitivity analysis to gauge the potential significance of
24 differential toxicity. After extensive review of the literature and analysis of options, the Project
25 Team concluded that currently available data and methodologies remain insufficient to meet the
26 challenge of developing a reasonably valid and usefully informative sensitivity analysis, even on
27 a notional basis. Indeed, the Project Team concluded that the potential research utility of such a
28 sensitivity analysis in the end did not appear to justify the risks from potential misinterpretation
29 and misapplication of the results of such a sensitivity analysis. The Project Team's evaluation of
30 the issue of differential toxicity is presented in chapter 5 of the draft uncertainty report.
31

32 Charge question: Does the Council HES support the Project Team's decision to defer
33 quantitative sensitivity analysis of potential PM component differential toxicity? If not, are there
34 data or methods for conducting a quantitative analysis of PM component differential toxicity that
35 the HES recommends EPA consider, or are there other aspects of differential PM component
36 toxicity which the HES recommends should be addressed in the draft benefits and/or uncertainty
37 reports?
38

39 HES response: The Council had encouraged the Project Team to explore the feasibility of
40 conducting a sensitivity analysis to gauge the potential significance of differential toxicity. The
41 Project Team has determined after a review of the literature that the currently available data were
42 insufficient for developing an informative sensitivity analysis.
43

44 However, there are some available regional data on the components of PM. In the PM ISP,
45 variability in PM_{2.5} components across the U.S. was examined by focusing on fifteen
46 metropolitan areas chosen based on their geographic distribution and coverage in recent health

1 effects studies. The urban areas selected were Atlanta, Birmingham, Boston, Chicago, Denver,
2 Detroit, Houston, Los Angeles, New York, Philadelphia, Phoenix, Pittsburgh, Riverside, Seattle,
3 and St. Louis. On an annual average basis, sulfate was the dominant PM_{2.5} component in the
4 eastern cities, ranging from 42% of PM_{2.5} mass in Chicago to 56% in Pittsburgh. Organic
5 carbon mass (OCM) was the next largest component. In the western cities, OCM was the largest
6 constituent of PM_{2.5} on an annual basis, ranging from 34% in Los Angeles to 58% in Seattle.
7 Sulfate, nitrate and crustal material were all important components in the western cities analyzed.
8 Sulfate ranged from 18% in Denver to 32% in Los Angeles. Nitrate was particularly large in
9 Riverside (22%), Los Angeles (19%) and Denver (15%); crustal material constituted a
10 substantial fraction of PM_{2.5} year-round in Phoenix (28%) and Denver (16%) and during the
11 summer in Houston (26%), even though the annual average was much lower (11%).
12

13 Differentiation on the basis of size fraction was addressed with respect to PM₁₀ vs. PM_{2.5} but the
14 Project Team decided that there was insufficient information to discuss PM_{10-2.5} separately.
15 There are some new studies that could be considered (see e.g.....) and it would be useful to
16 evaluate whether such studies might be the basis of a limited sensitivity analysis. The emerging
17 database on nanoparticle components of PM was not addressed but might be similarly
18 considered. One important consideration might be whether the current plan for measuring coarse
19 particles and nanoparticles concentrations will eventually provide sufficient data to allow for a
20 more formal sensitivity analysis. This may be more important for some health outcomes than for
21 others.
22

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

36 The HES agrees that the evidence base at this time does not currently support this sort of
37 assessment. Additionally, HES thinks that differential assessment – even if feasible – would not
38 lead to substantially different results in an assessment such as the 812 Analysis, which deals with
39 changes in the pollution mixture as a whole; and so it supports the EPA’s approach, of no
40 differential toxicity, in the present 812 analysis. However, HES recognizes that benefits analysis
41 of specific measures may affect particular constituents of PM only, and that – for other analyses
42 – the issue of differential toxicity should be considered anew.
43
44

45 Charge question 2j: Dynamic Population Modeling
46

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

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

12
13 HES response: HES appreciates the work EPA has done to illustrate the use of dynamic
14 population modeling for estimating the mortality impacts of long-term exposure to PM2.5. In
15 general, HES supports the use of dynamic population modeling where practicable because it
16 provides the most realistic available modeling of how, over time, changes in population risk lead
17 to changes in the size and age distribution of the population, with consequent implications for
18 estimated mortality impacts, whether expressed as deaths or life years.

19
20 We note EPA’s concern that a full dynamic population implementation requires detailed
21 projections for every year up to 2020 (compared with currently for 2000, 2010 and 2020 only),
22 and that this is very resource-intensive if carried out at the small spatial scale of the current core
23 methods. We recommend therefore that EPA bases its dynamic population modeling on
24 estimating mortality effects forward in time, of 1-year differences between scenarios, based on
25 the years 2000, 2010 and 2020, i.e. the approach reported by EPA in the Uncertainty Report,
26 when comparing dynamic population modeling with core BenMap methods. As noted by EPA,
27 this is an intermediate strategy, which for a modest increase in effort captures many (though not
28 all) of the gains of dynamic population modeling.

29
30 The HES discussed how the dynamic population approach highlights some subtleties in how
31 monetary values are linked with the mortality implications of different pollution scenarios. HES
32 requests EPA to describe, in substantially more detail than at present, the methods it used, and
33 the reasons why these methods rather than others were selected. This includes, but is not limited
34 to, describing (i) the current (‘static population’) BenMap approach to estimating deaths
35 postponed; (ii) how, within this approach, VSL values are linked with changes in risk or deaths,
36 and the assumptions underlying this approach; (iii) why EPA favors valuation based on deaths /
37 VSL rather than life years / VS LY; (iv) whether EPA can link monetary values to the results of
38 dynamic population modeling, and if so how this might be done; and in particular (v) whether
39 the changing year-on-year pattern of deaths, illustrated by dynamic population modeling, can be
40 incorporated into the monetary analysis.

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REFERENCES

Bell, ML, McDermott A, Zeger SL, Samet JM, and Dominici F 2004. Ozone and short-term mortality in 95 US urban communities, 1987-2000. *Journal of the American Medical Association* 292(19): p. 2372-8

Bell ML, Peng RD, Dominici F 2006. The exposure-response curve for ozone and risk of mortality and the adequacy of current ozone regulations. *Environmental Health Perspectives* 114:532-536.

Huang Y, Dominici F, and Bell ML 2005. Bayesian Hierarchical Distributed Lag Models for Summer Ozone Exposure and Cardio-Respiratory Mortality. *Envirometrics* 16: 547-562.

Jerrett M, Burnett RT, Pope CA 3rd, Ito K, Thurston G, Krewski D, Shi Y, Calle E, Thun M. 2009. Long-term ozone and mortality. *New England Journal of Medicine* 360:1085-1095.

Laden, F, Schwartz J, Speizer FE, and Dockery DW 2006. Reduction in Fine Particulate Air Pollution and Mortality. *American Journal of Respiratory and Critical Care Medicine* 173: 667-672.

Ostro BD, Feng WY, Broadwin R, Green S, and Lipsett M 2007. The effects of components of fine particulate air pollution on mortality in California: results from CALFINE. *Environmental Health Perspectives* 115(1): 13-19.

Ostro BD, Feng WY, Broadwin R, Malig BJ, Green RS, Lipsett MJ 2008. The impact of components of fine particulate matter on cardiovascular mortality in susceptible subpopulations. *Occupational and Environmental Medicine* 65: 750-756.

Pope, CA III, Burnett RT, Thun MT, Calle EE, Krewski DD, Ito K, and Thurston GD 2002. "Lung Cancer, Cardiopulmonary Mortality, and Long-term Exposure to Fine Particulate Air Pollution." *Journal of the American Medical Association* 287:1132-1141

Ritz B, Wilhelm M, Zhao Y 2006. Air Pollution and Infant Death in Southern California, 1989-2000. *Pediatrics* 118:493-502.

Smith KR, Jerrett M, Anderson HR, Brunett RT, Stone V, Derwent R, Atkinson RW, Cohen A, Shonkoff SB, Krewski D, Pope CA 3rd, Thun MJ, and Thurston G 2009. Public health benefits of strategies to reduce greenhouse-gas emissions: health implications of short-lived greenhouse pollutants. *The Lancet* 374 (9707): 2091-2103.

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APPENDIX A: Specific Comments

-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 5th 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.
32
33

34 Uncertainty Analysis

35
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.
44

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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.
13

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1 APPENDIX B: BIOSKETCHES
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